Neural and Muscular Factors Influence Maximal Power Generation

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We accept this thesis as conforming
to the required standard

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Supervisor: Dr. H.A. Wenger

Abstract

The purpose of this thesis was to investigate the role of selected neuromuscular factors thought to affect the generation of maximal power outputs in a complex multi-joint movement, using multiple muscles, contracting across multiple joints (cycle ergometry). The reliability of measurement for the neuromuscular variables was initially determined. There was a range of reliabilities, and this must be considered in the interpretation of both the cross-sectional and longitudinal studies. A cross-sectional study suggested that neural factors were not important in maximal power generation, but rather the amount of muscle, especially Type II muscle, seemed to differentiate those that could produce high power outputs and those that could not. Since there was no difference in the magnitude of relationship between either single or multi-joint strength, and multi-joint power, it was also suggested that the simulation of a power movement pattern (neural specificity) in a strength movement, would not influence power acquisition. A longitudinal study supported this since there was no difference in the rate of power acquisition between single and multi-joint strength training. Further, sprint training using an identical movement to that used in testing maximal power output, was not more effective than the strength training modalities in increasing power output, and the adaptations between these three training modes were similar. Likewise, sequencing of neurally specific sprint training after strength training does not cause greater power acquisition than sprint training alone. The muscle hypertrophy and strength or power improvements caused by training in these modes does not necessarily cause intrinsic improvements in muscle transferable to other movements using different modes of contraction (isokinetic strength). Thus some type of neural training effect seems to be evident. It does not involve increasing the activation of the muscle mass involved in a movement, but may involve plasticity of the motoneurones themselves (increased nerve conduction velocity) or a motor learning effect such that the co-ordination and synchronization of muscle and motor unit activation occurs more readily after training. In the long-term, this may be overridden by muscle adaptation since in the cross-sectional study no neural differences were noted.

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OVERVIEW

The purpose of this thesis was to investigate the role of selected neuromuscular factors thought to affect the generation of maximal power outputs in a complex multi-joint movement, using multiple muscles contracting across multiple joints (cycle ergometry). Three separate studies were utilized to perform this research.

The reliability of measurement was determined for those neuromuscular variables where this was not well established, or where protocols were sufficiently different from other previous investigators. After establishing reliability, a cross-sectional research design was utilized to determine the extent of neuromuscular differences between power, endurance and non-athletic groups, and to determine whether significant relationships existed between key neuromuscular variables and maximal power generation. Since a cross-sectional research design does not allow "cause and effect" to be determined, a longitudinal 14 week training study was also utilized to determine whether neuromuscular variables could be manipulated through single or multi-joint strength training, sprint training, or sequenced strength-sprint training in order to improve multi-joint power generation.

Twenty-three subjects were studied in order to appraise the reliability of measurement for selected neuromuscular variables. As assessed using intraclass reliability coefficients (R) peak torque measured on the Cybex was highly reliable for leg extension at angular velocities of 0-3.14 rad·s^{-1} (R=0.83-0.94), but showed lower reliability at 4.19 rad·s^{-1} (R=0.64). Plantar flexion peak torque was reliable for isometric and 1.05 rad·s^{-1} contractions (R= 0.72 and 0.76 respectively) but sharply decreased at angular velocities of 2.10-4.19 rad·s^{-1} (R=0.55-0.58) and leg press peak torque was reliable for isometric (R=0.72) and isokinetic peak torques at low to high velocities (0.76-0.91). Thus there is some difference in the reliability of single vs multi-joint strength movements, since as angular velocity increased in single-joint movements reliability decreased, yet isometric leg press showed low reliability. Peak rate of torque development (RTD) and the percentage of peak torque that this value occurred at were not reliable for any movement (R=0.02-0.28) nor was mean RTD between 30 and 60% of peak torque for leg press (R=0.46), yet mean RTD was fairly reliable for both knee extension and plantar flexion (R=0.61, 0.63 respectively). Mean integrated electromyography (IEMG) showed low but still acceptable reliability for isometric leg press (R=0.66) and much higher reliability at 1.05 rad·s^{-1} (R=0.90). Mean IEMG was also reliable for both isometric and 1.05 rad·s^{-1} plantar flexion (R=0.86, 0.75) and leg extension (R=0.85, 0.84). As well, nerve conduction velocity (NCV) was highly reliable (R=0.89). It seems then that the majority of these neuromuscular variables may be measured reliably; however, there is a range of reliabilities, and these must be considered in the interpretation of both the cross-sectional and longitudinal studies that investigate these variables.

In order to clarify the roles of neuromuscular factors in multi-joint maximal power production (15 s cycle ergometer power test) those variables evaluated as reliable were studied across power, endurance and non-athlete groups (n=10 each group). In addition, power was tested on a cycle ergometer and muscle biopsies were utilized to provide fiber type and cross-sectional area information. The power group had higher absolute cycle ergometer powers than both endurance (26%) and control (15%) groups, but these differences disappeared when power was expressed relative to body mass or leg volume. Power athletes were also stronger than both endurance (51, 52%) and control (33, 35%) subjects for both leg extension and plantar flexion respectively, at all velocities tested. In leg press they were stronger then endurance (32%) and control subjects (36%) for only the isometric and 1.05 rad·s^{-1} contraction. The power group also had much higher RTD values than the endurance and control groups respectively, in both leg extension (83, 56%) and plantar flexion (40, 66%), however, NCV was not different between groups. Histochemical analyses of vastus lateralis biopsy samples in a sub-sample (n=24) revealed no differences in the percentage of Type II muscle fibers. While Type I and II fiber cross-sectional areas between groups were similar, power athletes had a15% larger Type II/I fiber area ratio than controls. Strength, RTD and power were related to muscle and muscle fiber size variables, but not fiber distribution or NCV. The cross-sectional area of type II muscle fibers seemed to be especially important since this was the only variable related to power when adjusted for body size. Thus, muscle size variables and not fiber type or neural variables seem especially important in maximal power production. Whether these traits are inherited or responive to long-term physical training is open to question. A longitudinal training study was therefore performed.

The primary purpose of the longitudinal study was to determine whether or not neural and muscular adaptation, as well as power acquisition, differed between single-joint strength training, multi-joint strength training and sprint training. A secondary purpose was to determine the effect of sequencing 8 weeks of strength training (single or multi-joint strength) prior to sprint training (6 weeks) versus sprint training alone (14 weeks) on multi-joint power acquisition (cycle ergometer). 32 male subjects, age 20-28, were randomly assigned to either control (C), sprint-sprint (SS), multi-joint strength-sprint (MJS) or single-joint strength-sprint groups (SJS), (n=8 each group). Subjects were tested utilizing the same tests as in the cross-sectional research, prior to beginning training, mid-training at the end of 8 weeks of either single or multi-joint strength training or sprint training, and post training after another 6 weeks of sprint training. In addition, SJS and MJS, were tested weekly, and C tested pre, mid and post training for 10 repetition maximum (RM) strength on the Universal weight training equipment. A sub-sample of subjects also recieved biopsies (m. vastus lateralis) pre, mid and post training and their muscle was histochemically analyzed. At mid-test both SJS (43.6%) and MJS (41.1%) were stronger than pre-training (mean 10 RM strength) on the training equipment, but this strength was not transferable to isometric or isokinetic Cybex strength or RTD, and likewise SS showed no Cybex improvements. All training groups did however increase multi-joint power output by 8 weeks (eg. 5s mean power output increases, SS = 7%, SJS = 4%, MJS = 4%, C = -4%) and showed similar Type I and II fiber hypertrophy (SS=8.4%, SJS=13.5%, MJS=11.7%) however IEMG did not change. With a subsequent 6 week period of sprint training power continued to increase, but not differentially between groups (increase from pre-test: SS = 11%, SJS = 6%, MJS = 7%, C = -4%), there was no further muscle hypertrophy, no IEMG changes but NCV had increased significantly by a small amount in all training groups (SS=5%,SJS=3%, MJS=3%,

C=1%). These data suggest little difference in adaptation to single and multi-joint strength training, and also indicate that muscle hypertrophy and strength or power improvements caused by training in these modes do not necessarily cause intrinsic strength improvements in muscle transferable to other movements using different modes of contraction. Furthermore, sequenced strength-speed training provided no additional power gain than sprint training alone.

The cross-sectional research suggested that neural factors were not important in maximal power generation, but rather the amount of muscle, especially Type II muscle, seemed to differentiate those who could produce high power outputs and those who could not. Since there was no difference in the magnitude of relationship between either single or multi-joint strength, and multi-joint power, it was also suggested that the simulation of a power movement pattern (neural specificity) in a strength movement, would not impact power acquisition. The longitudinal study supported this since there were no differences in the rates of power acquisition between single and multi-joint strength training. Further, sprint training, using an identical movement to that used in testing maximal power output, was not more effective than the strength training modalities at increasing power output, and the adaptations between these three training modes were similar. The sequencing of neurally specific sprint training after strength training does not cause greater power acquisition than sprint training alone. The muscle hypertrophy and strength or power improvements caused by training in these modes does not necessarily cause intrinsic improvements in muscle transferable to other movements using different modes of contraction (isokinetic strength), thus some type of neural training effect seems to be evident. It does not involve increasing the activation of the muscle mass used in a movement, but may involve plasticity of the motoneurons themselves (increased NCV) or a motor learning effect such that the co-ordination and synchronization of muscle and motor unit activation occur more readily after training. In the long-term this may be overridden by muscle adaptation since in the cross-sectional study no neural differences were noted.

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Dedication

This thesis is dedicated to the memory of my father, Douglas Grant Sleivert, a great man, who always encouraged me to further my education.

Chapter 1

The reliability of measuring neuromuscular variables related to force generation.

Abstract

In order to determine the reliability of common neuromuscular measures utilized in exercise science, 20 males and 3 females, mean (SD) age 24.7 (3.6) years, body mass 75.8 (9.6) kg, height 184.1 (6.3) cm and sum of 8 skinfolds 80.0 (32.9) mm, visited the laboratory on 3 occasions. The first visit was an orientation session. In the remaining two visits which were separated by 48 hours, subjects underwent identical physiological testing including determination of tibial nerve conduction velocity (NCV), isometric and isokinetic strength (1.05-4.19 rad·s^{-1}) and maximal and mean rates of isometric torque development (RTD) on the Cybex isokinetic dynamometer, for leg press, leg extension, and plantar flexion. In addition average IEMG for isometric and 1.05 rad·s^{-1} contractions in these movements was measured. As assessed using intraclass reliability coefficients (R), peak torque measured on the Cybex was highly reliable for leg extension at angular velocities of 0-3.14 rad·s^{-1} (R=0.83-0.94), but showed lower reliability at 4.19 rad·s^{-1} (R=0.64). Plantar flexion was reliable for isometric and 1.05 rad·s^{-1} contractions (R= 0.72 and 0.76 respectively) but sharply decreased at angular velocities of 2.10-4.19 rad·s^{-1} (R=0.55-0.58). Leg press peak torque was reliable for isometric (R=0.72) and isokinetic peak torques at low to high velocities (0.76-0.91). Thus, there is some difference in the reliability of single vs multi-joint strength movements, since as angular velocity increased in single-joint movements reliability decreased, yet isometric leg press showed low reliability. Peak RTD and the percentage of peak torque at which this value occurred were not reliable for any movement (R=0.02-0.28) nor was mean RTD between 30 and 60% of peak torque for leg press (R=0.46), yet mean RTD showed fair reliability for both knee extension and plantar flexion (R=0.61, 0.63 respectively). Mean IEMG showed fair reliability for isometric leg press (R=0.66) and much higher reliability at 1.05 rad·s^{-1} (R=0.90). Mean IEMG was also reliable for both isometric and 1.05 rad·s^{-1} plantar flexion (R=0.86, 0.75) and leg extension (R=0.85, 0.84). As well, NCV was highly reliable (R=0.89). It seems then, that a range of reliabilities can be expected when measuring common neuromuscular variables, and therefore must be determined in the course of the experiment.

Introduction

The measurement of in vivo muscular force and power and those neuromuscular variables that may influence them are widespread in both longitudinal (Sale et al. 1982; Ewing Jr. et al. 1990; Hakkinen et al. 1992) and cross-sectional research (MacDougall et al. 1982; Hakkinen & Keskinen, 1989; Taylor et al. 1991), yet reliability of these measurements is frequently unreported (Sale et al. 1982; Hakkinen & Keskinen, 1989; Ewing Jr. et al. 1990). Given that the validity, of a measurement is at maximum equal to the square root of reliability (Baumgartner & Jackson, 1991, p156), it is imperative that the measurement technique and test protocols are reliable; that is, they give results that are consistent between testing occasions (Maguire & Hazlett, 1969). It was therefore the purpose of this study to report reliability statistics for the measurement of the following neuromuscular variables:

1. Maximal Cybex isometric and isokinetic force (1.05 to 4.19 rad $\cdot$ s^{-1}) for the single joint movements of leg extension and plantar flexion, and multi-joint leg press (combined hip extension, leg extension and plantar flexion).

2. Relative rate of isometric torque development for leg extension, plantar flexion, and leg press.

3. Integrated EMG for isometric and isokinetic (1.05 rad $\cdot$ s^{-1}) leg extension, plantar flexion and leg press.

4. Tibial nerve conduction velocity.

Methods

After University of Victoria Human Subjects Committee approval, 20 male and 3 female subjects, mean (SD) age 24.7 (3.6) years, body mass 75.8 (9.6) kg, height 184.1 (6.3) and sum of 8 skinfolds 80.0 (32.9) mm, signed informed consent and agreed to participate in the study. Each subject visited the lab on 3 occasions, separated by 48 hours. The first visit was an orientation session where the testing procedures were fully explained, and the physical characteristics of each subject measured. In each of the remaining two laboratory sessions subjects underwent identical physiological testing. Each subject was asked to refrain from vigorous exercise during the 24-hour period before testing sessions and not to eat in the 2 hours immediately preceding testing.

Experimental Procedures

Nerve Conduction Velocity

Upon arrival at the laboratory, tibial nerve conduction velocity was measured. Subjects lay prone on a padded table with the lower limb supported so that there was 120 degrees flexion of the leg and 90 degrees flexion at the ankle (Vecchierini-Blineau & Guiheneuc, 1979). Skin temperature was measured at two sites using skin thermistors taped to the lateral retro-malleolar groove and medial popliteal fossa (Halar et al. 1983). The thermistors were interfaced with a telethermometer (Yellowsprings). Maximal tibial nerve conduction velocity was obtained on the left leg using the traditional double stimulation technique (Smorto & Basmajian, 1979) and corrected for temperature effects (Halar et al. 1983)using the mean of the 2 leg temperatures. Briefly, square pulses of 0.1 ms duration and sufficient intensity to evoke a supramaximal compound muscle action potential were applied to the tibial nerve 1 cm laterally to the mid-line of the popliteal fossa, and at the medial retro-malleolar groove. The motor response was detected by two surface electrodes on the abductor hallucis muscle (Chu-Andrew, 1986; Dorfman & Bosley, 1979). All electrode positions were marked on the skin with indelible ink so that electrode placement was consistent between testing days. The difference in the latency of the motor response between the proximal and distal stimulation sites, together with the distances between the two sites of stimulation, was used to calculate nerve conduction velocity (Chu-Andrews, 1986). An integrated neurostimulator/amplifier/storage oscilloscope (Cadwell 5200) was used for all nerve stimulation and latency response measurements. Nerve distance was measured using metal calipers. The closest two values of three trials were averaged and taken to represent nerve conduction velocity for each session (Kamen et al. 1984).

Strength Measurements

Force exerted by the left leg was measured on an isokinetic dynamometer (Cybex) interfaced with a microcomputer using ATCODAS signal processing software (DATAQ). Maximal voluntary contractions at 0, 1.05, 2.10, 3.14 and 4.19 $rad \cdot s^{-1}$ were measured for leg press, leg extension and plantar flexion in order to construct a force velocity curve for each movement. For all Cybex tests, straps were used to immobilize the upper body and the command "ready-set-go" was used for each contraction. Subjects were instructed to perform each movement as fast and as hard as possible upon hearing "go". For isometric contractions subjects were required to hold maximal force for a period of 3s. Three repetitions were performed at each speed, with peak torque from the strongest repetition, taken to represent velocity specific strength. Half the subjects completed the testing in the order of leg press-plantar flexion-leg extension, while the other half tested in the opposite order.

Leg Press: Peak torque exerted in the leg press exceeds the maximal torque capacity of the Cybex, therefore it was modified with a gear and chain system as previously described by Vandervoort et al. (1984). Subjects were in a seated position and for isometric tests, strength was measured with the hip and knee positioned at 100°. For concentric contractions subjects started with the knee at 90° (Vandervoort et al. 1984).

Leg extension: Leg extension was measured in the seated position with a hip angle of 90° and the knee set at 100° for isometric contractions.

Plantar Flexion: Subjects were secured to a Cybex UBXT in the supine position. All contraction velocities were performed with the knee set at 100° and for the isometric contractions the ankle was also set at this angle.

Rate of Torque Development

For each movement the rate of torque development (RTD) was calculated from the isometric force-time curve. Force data was sampled at 2000 Hz and the first derivative of each force curve, smoothed by a factor of 7 was taken to provide a measure of RTD in $Nm \cdot s^{-1}$. The smoothing factor of 7 resulted in slopes being calculated from 7 points over a duration of 3.5 ms for each slope. Mean and maximal RTD between 30 and 60% of peak torque and the percentage of peak torque at maximal RTD were calculated.

Electromyography (EMG)

The motor point areas of the vastus lateralis (VL), rectus femoris (RF) and the medial (MG) and lateral (LG) heads of the gastrocnemius muscles were determined using an electrical stimulator. After reducing the skin impedance with sandpaper and rubbing alcohol, bipolar silver/silver chloride surface electrodes (3M) were placed over the motor point along the muscles longitudinal axes 20 mm apart rim to rim. Electrodes position was marked on the skin with indelible ink and subjects were asked to maintain these marks between testing sessions to ensure the same electrode positioning for each session (Hakkinen et al. 1991).

For each movement, EMG was collected for the isometric contractions as well as the 1.05 $rad \cdot s^{-1}$ concentric contraction. The myoelectric signal was sampled at 2000 Hz, amplified, and low pass (20Hz, 3rd order response) and high pass (1.5KHz, 2nd order response) filtered. The signal was subsequently rectified, integrated and averaged with ATCODAS signal processing software (DATAQ) for each muscle during the maximal force phase of the isometric contraction (1 s) and over the duration of the 1.05 $\text{rad} \cdot \text{s}^{-1}$ concentric contraction. For both isometric and isokinetic contractions the average IEMG values for each of the muscles monitored was summed and then averaged in order to provide a single EMG value for each movement (Hakkinen et al. 1992). Thus mean IEMG activity for each movement was calculated as follows:

Mean Leg extension IEMG = (VL-IEMG + RF-IEMG)/2
Mean Plantar flexion IEMG = (MG-IEMG + LG-IEMG)/2
Mean Leg Press IEMG = (VL-IEMG + RF-IEMG + MG-IEMG + LG-IEMG)/4

These average IEMG values represent quantitative measures of the amount of electrical activity produced by the muscle fibers of activated motor units during each maximal contraction (Sale,1991).

Statistics

The mean and standard deviation are used to describe the data. Reliability for each variable was evaluated by calculating intraclass coefficients (equation 1) from the corresponding repeated measures one-way analysis of variance (Maguire & Hazlett, 1969).

Equation 1:

$$\text{Intraclass R} = \frac{MS_{\text{between}} - MS_{\text{within}}}{MS_{\text{between}} + MS_{\text{within}}}$$

It has been suggested that an intraclass R of greater than 0.80 is acceptable for clinical work (Burdett & Van Swearingen, 1987; Currier, 1984). Given the importance of reliable measures in research this standard of comparison will be used as the criteria for "good" reliability in this study. Measurement of certain physiological phenomena may not show this level of reliability, but still provide the researcher with some degree of information, thus intraclass R values of 0.60-0.80 will be classified as "fair" reliability. Measurements with intraclass R values below 0.60 will be classified as unreliable.

The standard error of measurement (SEM) was calculated as a further measure of reliability (equation 2). This statistic reflects the limits with which an individuals test score should fall 68 times out of 100, or the degree one may expect a test score to vary due to measurement error (Baumgartner & Jackson, 1991; p 141).

Equation 2: $\mathrm{SEM} = \mathrm{SD}(1\text{-R})^{0.5}$

Results

Reliability statistics (Table 1) for leg extension, plantar flexion and leg press at contraction velocities ranging from 0 to 4.19 rad·s^{-1} showed that for the two single joint movements reliability decreased as the speed of contraction increased, however in the multijoint leg press movement no discernible trend was evident. Reliability of mean RTD (Table 2) between 30 and 60 percent of peak torque for the two single joint movements was similar to that found for high velocity strength, but for multijoint leg press it was lower than strength reliability. Peak RTD (Table 2) and the percentage of peak torque at which peak RTD occurred (Table 2) was not reliable for any movement. The reliability of mean IEMG (Table 3) for both isometric and concentric (1.05 rad·s^{-1}) leg extension, plantar flexion and leg press was high for all conditions except isometric leg press. Excellent reliability was found for the measurement of tibial motor nerve conduction velocity (Table 4).

Table 1: Peak torque: trial means (SD), test-retest intraclass reliability coefficients (R) and standard error of measurements (SEM) for isokinetic single- and multi-joint lower extremity movements (n=23).

Movement	Velocity ($rad \cdot s^{-1}$)	Trial 1 (Nm) Mean(SD)	Trial 2 (Nm) Mean(SD)	R	SEM (Nm)
Leg Extension	0	276 (65)	289 (73)	0.94	17
	1.05	204 (38)	200 (42)	0.93	11
	2.10	161 (30)	156 (35)	0.91	10
	3.14	126 (25)	119 (25)	0.83	10
	4.19	99 (21)	90 (17)	0.64	12
Plantar Flexion	0	91 (27)	98 (29)	0.72	15
	1.05	79 (17)	80 (19)	0.76	9
	2.10	50 (10)	46 (12)	0.58	7
	3.14	31 (8)	27 (9)	0.58	6
	4.19	21 (7)	18 (6)	0.55	4
Leg Press	0	551 (169)	532 (169)	0.72	89
	1.05	419 (91)	428 (87)	0.89	30
	2.10	205 (42)	199 (42)	0.76	20
	3.14	152 (35)	149 (34)	0.91	11
	4.19	116 (31)	116 (31)	0.88	11

Table 2: Rate of torque development between 30 and 60 percent of peak torque (RTD): trial means (SD), test-retest intraclass reliability coefficients (R) and standard error of measurements (SEM) in single- and multi-joint lower extremity isometric contractions (n=23).

Movement	**RTD Variable**	**Trial 1 Mean (SD)**	**Trial 2 Mean (SD)**	**R**	**SEM**
Leg Extension	Mean RTD ($Nm \cdot s^{-1}$)	733 (273)	638 (158)	0.61	216
	Peak RTD ($Nm \cdot s^{-1}$)	1982 (1300)	1999 (1344)	0.08	1268
	% Peak Torque @ Peak RTD	44 (8)	45 (8)	0.08	8
Plantar Flexion	Mean RTD ($Nm \cdot s^{-1}$)	246 (85)	261 (92)	0.63	53.9
	Peak RTD ($Nm \cdot s^{-1}$)	1006 (401)	735 (195)	0.13	278
	% Peak Torque @ Peak RTD	43 (8)	42 (9)	0.02	8
Leg Press	Mean RTD ($Nm \cdot s^{-1}$)	1304 (568)	1212 (408)	0.46	359
	Peak RTD ($Nm \cdot s^{-1}$)	4188 (1656)	3714 (1033)	0.28	1141
	% Peak Torque @ Peak RTD	43 (9)	45 (9)	0.11	8

Table 3: Integrated electromyography (IEMG): trial means (SD), test-retest intraclass reliability coefficients (R) and standard error of measurements (SEM) in single- and multi-joint lower extremity movements (n=23).

Movement	Velocity rad·s^{-1}	Trial 1(uv) Mean (SD)	Trial 2 (uv) Mean (SD)	R	SEM (uv)
Leg Extension	0	591 (231)	651 (300)	0.85	103
	1.05	524 (228)	491 (188)	0.84	83
Plantar Flexion	0	290 (101)	298 (111)	0.86	40
	1.05	293 (083)	299 (82)	0.75	41
Leg Press	0	276 (76)	287 (66)	0.66	41
	1.05	308 (109)	317 (88)	0.90	31

Table 4: Tibial motor nerve conduction velocity: trial means (SD), test-retest intraclass reliability coefficient (R) and standard error of measurement (SEM) (n=23).

Trial 1 (m·s^{-1}) Mean (SD)	Trial 2 (m·s^{-1}) Mean (SD)	R	SEM
52.5 (3.5)	52.4 (3.7)	0.89	1.2

Discussion

Strength measurement of the knee extensors on the Cybex showed acceptable reliability at contraction velocities ranging from 0 to 3.14 rad·s^{-1} and there was a trend of decreasing reliability as the speed of contraction increased. This was most evident at 3.14 and 4.19 rad·s^{-1} where there were sharp decreases in the magnitude of the intraclass reliability coefficients (R) in comparison to the slower contraction velocities. Peak torque at 4.19 rad·s^{-1} showed only fair reliability. Tredinnick and Duncan (1988) have similarly reported a large decrease in reliability on the Cybex as velocity of leg extension was increased from 2.10 to 3.14 rad·s^{-1}. At Cybex velocities of 3.14 and 4.19 rad·s^{-1} the true speed of the lever arm deviates more (0.1-5.2%) than slower speeds (0.1-1.9%), which could account for the decreased reliability at the higher speeds of contraction (Bemben et al. 1988). Alternatively, in untrained subjects, the novel task of recruiting motor units for a high velocity contraction may limit the reproducibility of strength measurement in this situation. Many other studies have not reported decreasing reliability in knee extensor peak torque as the velocity of contraction increases. Intraclass R values in these studies ranged between 0.78 and 0.98 for contraction velocities ranging from isometric to 4.19 rad·s^{-1}. Larger sample sizes (Burdett & VanSwearingen, 1987; Thigpen et al. 1990), the use of gravity corrected peak torques (Burdett & VanSwearingen, 1987), different populations (Thigpen et al. 1990) and differences in joint angles and set-up procedures (Molczyck et al. 1991) could account for this.

The measurement of plantar flexion was not as reliable as leg extension, and reliability similarly decreased as the velocity of contraction increased. The reliability of peak torque measures at velocities greater than 2.10 rad·s^{-1} was unacceptable. Little data exists on the reliability of Cybex plantar flexion however Wennerberg (1991) reported R coefficients of 0.79 and 0.67 at velocities of 0.53 and 2.10 rad·s^{-1} respectively, which agrees with the data of the present study. The ankle has 3 articulations making ankle biomechanics complex, since the ankle may move through a number of planes and axes of rotations (Oberg et al. 1987). As well, the musculature of the ankle is small and passes over multiple joints, thus consistent joint positioning, and stabilization of the ankle, knee and hips is critical when testing the plantar flexors (Karnofel et al. 1989). This may cause greater variability in tests scores than those observed with a less complex 1-joint structure such as the knee. Anecdotally, subjects also reported that the motor pattern of isolated plantar flexion was difficult to perform and required great concentration, especially at higher velocities. Subjects were constantly reminded that only the plantar flexors were to be utilized, since in the flexed-leg position there were tendencies to extend the leg during plantar flexion. Despite precautions aimed at minimizing the contribution of leg extensor muscles to force generation at the foot plate (immobilization with straps), it is possible that in some subjects this occurred, which reinforces the importance of isolating the muscle group to be tested. This is especially difficult in flexed leg plantar flexion which may account for the low reliability scores, and require that subjects are given extra familiarization time for this task.

Higher R coefficients have also been reported for plantar flexion. Clarkson et al. (1980) measured isometric plantar flexion strength in power and endurance athletes and reported R values of 0.90 and 0.94 respectively. An athletic population experienced in strength training would be more familiar with movements such as isolated plantar flexion; therefore, better reliability is not surprising. Karnofel et al. (1989) reported R values similar to those of Clarkson (1980) at 1.05 and 2.10 $rad \cdot s^{-1}$. These investigators reported mean peak torque values calculated from the last 5 of 6 repetitions at each velocity. This may have served to reduce the variability observed between testing sessions and increase reliability of measuring plantar flexion. They also used a knee angle of 45° versus the 100° used in the present study. They suggested that this knee angle placed the plantar flexors at a length most representative of normal functional activity. A more functional joint angle could increase the reliability of producing plantar flexion force.

Unlike plantar flexion and leg extension, no pattern of decreasing reliability with increasing velocity of contraction was evident for the multi-joint leg press movement, and R ranged from 0.72 to 0.91. The multi-joint movement pattern is probably more familiar to subjects than the isolated single-joint movements, which may contribute to more consistent reliability across all contraction velocities than in the single-joint movements. The isometric contractions showed the lowest reliability score, and was characterised by very high torque values. Varying influences of neural inhibitory mechanisms such as the Golgi tendon organs (Caiozzo et al. 1981) may play a role at high torque levels in decreasing the reliability of the strength measurement. The reliability of measuring multi-joint isokinetic leg-press force has previously been reported using the method-error statistic (Vandevoort et al. 1984). These researchers showed method-error to equal 12.4% for peak torque measurements made on separate days, but did not report this statistic for different velocities of contraction. When method-error is calculated for unilateral leg press (16.2%) in the present study it is slightly higher than Vandervoort et al. (1984) reported for the isometric condition but of the same magnitude or lower for higher velocity contractions: 1.05 rad·s^{-1}(6.6%); 2.10 rad·s^{-1}(9.5%); 3.14 rad·s^{-1} (9.0%); 4.18 rad·s^{-1} (12.4%). These method-error values are in agreement with values reported by Sale (1991). He reported data of Vandervoort et al. (1980) that found method-error for unilateral leg extension torque between days equalled 8.2 and 13.4% for contraction velocities of 0.26 and 6.6 rad·s^{-1}, respectively. Thus it appears that these data support those of the present study and indicate that the measurement of multi-joint unilateral leg press peak torque is reliable.

Absolute rate of force or torque development is calculated by measuring the time taken to reach a given or maximum force or torque level, while relative measures of force and torque development utilize the time taken to increase force between given percentages of maximum, eg 10% to 30%, 60% and 90% of maximum. Both average and maximal rates of force (N•s^{-1}) or torque (Nm•s^{-1}) development can also be calculated between these values. Sale (1991) suggested that a limitation of using absolute measures is the difficulty in identifying the precise point where force takes off from the baseline or reaches peak. Relative measures avoid these problems. Although both of these methods have been frequently reported in the literature (Duchateau & Hainaut, 1984; Hakkinen & Keskinen, 1989; Hakkinen et al.

1992), only a limited amount of reliability data has been reported (Viitasalo & Komi,1978; Viitasalo et al. 1980), making it difficult to interpret the validity of the RTD research. The reliability of measuring relative rates of torque development was therefore investigated.

Between day reliability for mean RTD between 30 and 60% of peak torque was not high for any movement. Fair reliability is reported for leg extension and plantar flexion but it was substantially lower and unacceptable for leg-press. The lower values for leg press may reflect the chain and gear modifications to the Cybex. Chain tension may have varied between trials and the rate of torque development would also vary as a function of chain tension. This would not be a problem in the single-joint movements where the lever arm was attached directly to the input shaft of the Cybex. Viitasalo and Komi (1978) measured bilateral leg extension and reported between trial Pearson r values of 0.80 and 0.38 for absolute rate of force development at force levels below and above 90% of peak isometric force respectively. Between day Pearson r values were substantially lower and ranged from 0.66 to 0.76 at force levels below 90% of peak isometric force. These researchers concluded that absolute rate of force development showed "satisfactory" reliability below 90% of peak isometric force. Their conclusion is problematic however, since Pearson r is not sensitive to changes in means and standard deviations between trials and therefore does not give a true picture of reliability (Maguire & Hazlett, 1969). Viitasalo et al. (1980) measured average relative rates of force development in unilateral leg extension. Within day reliability was calculated between the average of the first and third (trial 1) and second and fourth (trial 2) repetitions, but no between day reliability was calculated. Mean relative rate of force development values had between trial Pearson r values of 0.76 to 0.87. Between day reliability would be expected to be substantially lower and given the problems with using the Pearson r statistic to indicate reliability, the data of the present study seems more reasonable.

Viitasalo et al. (1980) also calculated maximal rate of force development from the highest slope coefficient of a tangent to the force-time curve and reported a same-day between trial Pearson r of 0.84. The percentage of peak torque at which this value occurred was less reliable (Pearson r=0.59). The present study examined between day measurements for these variables and showed little reliability for any movement. The lack of reliability for these measures may be influenced by two factors. Firstly, the task of producing force as quickly as possible is very unfamiliar to untrained subjects. It may require more extensive familiarization and practice before reliable results can be produced. Secondly, instantaneous rates of torque development were being measured every 3.5 msec. This short sampling time results in large variability of instantaneous RTD values. Different smoothing protocols or sampling frequencies could reduce the variability and increase reliability of this measurement. Differences in analysis procedures between studies are probably the main reason for varying reliability reports. Environmental factors and individual variation will also decrease the between day reproducibility of maximal rate of torque development and the percentage of peak torque at which this occurs.

During voluntary contractions, the electrical activity produced by muscles may be recorded and quantified using surface electromyography (EMG). Quantification of the EMG signal is generally accomplished by integration of a full-wave rectified signal to give an absolute value of the EMG called the integrated EMG (IEMG) (Winter,1990; p204). Increases or decreases in IEMG as a result of exercise training or detraining are thought to reflect the interaction of factors that both facilitate and inhibit various levels of the nervous system (Moritani & deVries, 1979). Collectively, changes in these factors are referred to as neural adaptation (Sale et al. 1982) and the IEMG technique is commonly utilized in conjunction with maximal voluntary isometric contractions to monitor neural adaptation (Moritani & deVries, 1979; Hakkinen et al. 1992). Although factors such as electrode placement, skin preparation, temperature and electrical conductivity status of muscle tissue (Yang & Winter, 1983; Hering et al. 1988; Winter, 1990; p197) may influence the EMG signal, the reliability of IEMG has not been frequently reported.

Of those studies reporting IEMG reliability the majority report high Pearson Product Moment correlations ($r > 0.98$) (Moritani & deVries, 1979; Viitasalo et al. 1980; Hering et al. 1988). Two of these investigators (Moritani & deVries, 1979;

Hering et al. 1988) pooled a large number of contractions at various percentages of a maximum voluntary contraction (MVC) which effectively increases the range of data and artificially increases the magnitude of the Pearson r (Clarkson et al. 1980). As previously mentioned another problem with using the Pearson r statistic to estimate reliability is insensitivity to changes in means and standard deviations (Maguire & Hazlett, 1969). The intraclass reliability coefficient avoids the problems inherent with the Pearson r for estimating reliability. Only one study utilized intraclass reliability coefficients for estimating EMG reliability and these investigators reported intraclass R values ranging from 0.52 for one trial on one day to R=0.76 for one trial on each of three days (Yang & Winter, 1983). These reliabilities were for maximal voluntary isometric contractions. No research has reported reliability statistics for EMG during dynamic contractions.

In this study, mean IEMG proved to be reliable for both isometric and concentric (1.05 $rad \cdot s^{-1}$) single joint movements. The reliability of multi-joint leg press IEMG was also acceptable for the concentric contraction but lower for the isometric condition. This lower reliability in isometric leg press paralleled the lower reliability of isometric torque production previously observed in leg press. Since EMG activity is quantitatively linked to isometric muscle tension development (Winter, 1990; p207) this is not surprising. As previously mentioned neural inhibitory mechanisms such as the Golgi tendon organs (Caiozzo et al. 1981) may play a role at high torque levels in decreasing the reliability of the strength measurement. This may also be true for the EMG signal and require that subjects are given extensive familiarization and practice in movements where high forces are generated in order to minimize inhibitory influences on force generation. In agreement with this, Yang & Winter (1983) found lower intraclass R values at 100% of a triceps maximal voluntary contraction (MVC) than at submaximal levels. Contrary to this, Hering et al. (1988) reported that IEMG during isometric contraction of the triceps brachii had higher between day coefficients of variation (CV) at 10 to 20 % of MVC (CV=21.3-28%) than at 100% MVC (CV=15.8%). These were simple single joint movements and the reason for these discrepancies are not obvious. It may be hypothesized that a learning phenomenon may be more important in high force movements where multiple muscles are working across multiple joints since these movements are inherently more complex. Yang & Winter (1983) suggest that in movements where a number of synergists are involved and a maximal effort is required, more variability would be observed than in simpler movements, due to varying contributions and inconsistent synchronization of the synergists between trials and between days. Force level and associated EMG activity could change simply as a result of different muscles being recruited in different patterns or to different extents. Varying degrees of agonist co-contraction could also influence the net force and EMG signal detected during a MVC.

Nerve conduction velocity (NCV) has been measured both in cross-sectional studies using different athletic groups (Kamen et al. 1984; Upton & Radford, 1976; Singh & Maini, 1980) and in longitudinal strength training studies (Sale et al. 1982). Numerous environmental and technical factors can influence the accuracy and reliability of measuring NCV, however only Kamen et al. (1984) have reported reliability statistics (Pearson r = 0.70 to 0.84). Considering the limitation of the Pearson r statistic and the variety of factors that can influence NCV more reliability information was required.

The test-retest reliability of tibial NCV was measured and found to be higher than that reported by Kamen et al. (1984) even though measurement procedures were similar. Since NCV is known to increase with increasing temperature (Kimura, 1983; Halar et al. 1983; Todnem et al. 1989) all NCV values were temperature corrected according to Halar et al. (1983). Kamen et al. (1984) did not correct for temperature, but rather tried to control nerve temperature by keeping ambient room temperature constant. Large variability was noted in limb temperature of the same subjects between days, even though room temperature was constant. It is likely then that the higher test-retest reliability of NCV in this study is a function of correcting NCV for temperature influences. Other environmental factors that can impact NCV were also controlled. It is known that extracellular fluid changes are of some consequence to NCV. For example, NCV changes have been noted in patients with renal failure undergoing haemodialysis (Waxman, 1980). Considering the possibility that dehydration or changes in electrolyte concentration could affect NCV the subjects in the present study were instructed to avoid exhaustive exercise in the two hours before NCV testing and NCV was measured prior to any exercise testing. Subjects were also tested at the same time of day for each session in order to avoid possible diurnal effects on NCV (Wyrick, 1970; Ferrario, 1980).

Technical factors can also affect accurate and consistent NCV measurement. One of the main errors in calculating NCV is inaccurate measurement of the interstimulus distance. Generally a tape measure is used for this however in the present study metal calipers were used for better accuracy. The position of the stimulating electrodes between testing days was also standardized by marking the skin with indelible ink, since it is important that the nerve is stimulated from the same position each session. Subjects were tested on each occasion by the same technician. This ensured consistent subject preparation, anthropometric techniques, standard stimulator angle and pressure on the skin and reliable localization of the M-wave on the oscilloscope. By following these procedures the measurement of tibial NCV is highly reliable between testing sessions.

The results of this study therefore suggest that peak torque may be measured reliably for both leg extension and leg press at lever arm velocities of 0-4.19 $rad \cdot s^{-1}$. Plantar flexion peak torque is also reliable at 0 and 1.05 $rad \cdot s^{-1}$, but caution must be exercised in interpreting higher velocity strength measurements for this movement since reliability was low. Mean RTD showed fair reliability for leg extension and plantar flexion, but not leg press, while peak RTD and the position in the torque time curve where this occurred was not reliable for any movement. IEMG showed fair to good reliability for both single and multi-joint movements and tibial nerve conduction velocity was reliable. Therefore of the variables considered, peak torque measurements for each movement at all velocities, mean RTD for leg extension and plantar flexion, IEMG for all movements and NCV will be included for further use in studies 2 and 3.

In attempting to answer important scientific questions an investigator may have a sound hypothesis, excellent experimental control, adequate statistical power, and appropriate statistical models but still obtain indecisive or inaccurate results due to poor reliability of the methods used to collect data. Reliable measures are a prerequisite to performing valid research. The present study illustrates that for neuromuscular variables commonly examined in the exercise science literature a range of reliabilities can be expected and therefore must be determined in the course of the experiment.

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Chapter 2

Neuromuscular differences between power and endurance athletes and sedentary controls.

Abstract

In order to determine whether neuromuscular differences existed between power, endurance and non-athletes, and to clarify the roles of neuromuscular factors in maximal power production, subjects visited the laboratory on 3 occasions, the first of which was an orientation and anthropometric session. Physical characteristics of the power (varsity volleyball, mean (SE) height 188.0 (2.2) cm, mass 81.1(2.9) kg, sum of 8 skinfolds 57.3(5.6) mm, leg volume 5562(178) cm^3), endurance (varsity distance runners, mean (SE) height 178.6(1.7) cm, mass 66.5(1.9) kg, sum of 8 skinfolds 51.9(3.6) mm, leg volume 4256(196) cm^3), and controls (mean (SE) height 180.5(1.4) cm, mass 72.3(1.5) kg, sum of 8 skinfolds 71.8(6.3) mm, leg volume 4759(191) cm^3) were measured (n=10 each group). In the remaining 2 laboratory sessions subjects underwent identical physiological testing, including determination of tibial nerve conduction velocity (NCV), isometric and isokinetic strength (1.05-4.19 rad·s^{-1}) for leg press, leg extension and plantar flexion and mean rate of isometric torque development (RTD) on the Cybex isokinetic dynamometer for leg extension and plantar flexion. As well subjects performed a 15s cycle ergometer power test (load = 95g/kg of body mass). For each test the highest value of the two sessions was retained for data analysis. The power group had higher absolute cycle ergometer power than both endurance (26%) and control (15%) groups, but these differences disappeared when power was expressed relative to body mass or leg volume. Power athletes were also stronger than both endurance (51, 52%) and control (33, 35%) subjects for both leg extension and plantar flexion respectively, at all velocities tested. In leg press they were stronger than endurance (32%) and control subjects (36%) for only the isometric and 1.05 rad·s^{-1} contraction . The power group also had much higher RTD's than the endurance group and controls, respectively in both leg extension (83, 56%) and plantar flexion (40, 66%) however NCV was not different between groups. Histochemical analyses of vastus lateralis biopsy samples in a sub-sample (n=24) revealed no differences in the percentage of Type II muscle fibers, or Type I and II fiber cross-sectional area between groups, yet power athletes had a larger Type II/I fiber area ratio than controls (15%). Both strength, RTD and power were related to muscle and muscle fiber size variables, but not fiber distribution or NCV. The size of type II muscle fibers seemed to be especially important since this was the only variable related to power when adjusted for body size. Thus, muscle size variables and not fiber type or neural variables seem important in maximal power production.

Introduction

The rapid generation of muscular force to provide acceleration, deceleration, maintain velocity or change direction of the body mass is critical in many sports. This ability to contract the muscles both forcefully (strength) and quickly (speed) is referred to as muscular power (Edgerton,1986). Athletes participating in power sports have been observed to differ from untrained individuals in both their muscular and neural properties (Sale, 1991) and these differences are thought to arise from both genetic and environmental factors (repeated exposure to power-type activities) (Perusse et al. 1987).

In general it has been shown that power athletes demonstrate higher muscular power than endurance athletes (Bouchard et al. 1991), however the extent to which neural and/or muscular factors explain these power differences have not been determined. For example, wide ranges in muscle fiber type profiles have been reported for both power and endurance athletes (Gollnick et al. 1972; Gregor et al. 1981; Viitasalo & Komi, 1978) and a high percentage of type II fibers does not always correlate with high power outputs (Patton et al. 1990; Mackova et al. 1985). Muscle cross-sectional area is generally larger and strength higher in power versus endurance athletes (Johansson et al. 1987; Hakkinen & Keskinen, 1989), however these two variables do not always correlate with each other (Gregor et al. 1981; Sale et al. 1992) or with maximal power (Bell et al. 1989; Patton et al. 1990). Similarly, power athletes have been reported to have significantly larger type II/I fiber area ratios than control or endurance subjects (Tesch et al. 1989) yet larger type I than type II fibers for both sprint and endurance athletes have also been reported (Gregor et al. 1981).

The unclear relationship existing between muscular variables and maximal power production is paralleled when neural variables are examined, although there is a paucity of data in this area. Motor nerve conduction velocity (NCV) has been reported to be faster in strength than endurance athletes (Kamen et al. 1984) but confounding data observing slower NCV in sprinters versus untrained controls also exists (Upton & Radford, 1975). Reflex latencies have been shown to differ between power athletes and distance runners (Kamen et al. 1981) and antagonist co-contraction is greater in sprint versus endurance athletes (Osternig et al. 1986). The relationship of these neural variables to maximal power production has not been reported.

Considering the equivocal nature of the relationship between neuromuscular variables and power production the purpose of this study was twofold. First, in order to determine whether neuromuscular differences existed between power, endurance and control groups the following variables were measured and compared between groups:

- multi-joint leg power (cycle ergometer)
- thigh volume
- Type I and II muscle fiber cross-sectional area (XSA)
- percentage of Type II fibers
- single-joint isokinetic strength (leg extension, plantar flexion)
- multi-joint isokinetic strength (leg press)
- rate of isometric torque development (leg extension, plantar flexion)
- posterior tibial nerve conduction velocity (NCV)

Second, in order to determine the contribution of both neural and muscular factors to maximal power expression, the relationship between these neuromuscular variables was described.

Methods

Following University of Victoria Human Subjects Committee approval, 10 male subjects were recruited from each of three different populations. Since volleyball is an intermittent sport characterized by explosive jumps, short powerful movements and long rest intervals, varsity volleyball players were selected to comprise a power group. Varsity middle distance runners with histories of high volumes of endurance training were used as endurance subjects and untrained university students were used as controls. After signing informed consent and agreeing to participate in the study, each subject visited the lab on 3 occasions, separated by 48 hours. The first visit was an orientation session where the testing procedures were fully explained, and body height, mass, sum of 8 skinfolds (Ross & Marfell-Jones, 1991), and left thigh volume (Katch et al. 1974) were measured anthropometrically for each subject. In each of the remaining two laboratory sessions, subjects underwent identical physiological testing. Each subject was asked to refrain from vigorous exercise during the 24-hour period before testing sessions and not to eat in the 2 hours immediately preceding testing.

Experimental Procedures

Nerve Conduction Velocity

Upon arrival at the laboratory, tibial nerve conduction velocity was measured. Subjects lay prone on a padded table with the lower limb supported so that there was 120 degrees flexion at the knee and 90 degrees flexion at the ankle (Vecchierini-Blineau & Guiheneuc, 1979). Skin temperature was measured at two sites using skin thermistors taped to the lateral retro-malleolar groove and medial popliteal fossa (Halar et al. 1983). The thermistors were interfaced with a telethermometer (Yellowsprings). Maximal tibial nerve conduction velocity was obtained on the left leg using the traditional double stimulation technique (Smorto & Basmajian, 1979) and corrected for temperature effects (Halar et al. 1983) using the mean of the two leg temperatures. Briefly, square pulses of 0.1 ms duration and sufficient intensity to evoke a supramaximal compound muscle action potential were applied to the tibial nerve 1 cm lateral to the mid-line of the popliteal fossa, and at the medial retro-malleolar groove. The motor response was detected by two surface electrodes on the abductor hallucis muscle (Chu-Andrew, 1986; Dorfman & Bosley, 1979). All electrode positions were marked on the skin with indelible ink so that electrode placement was consistent between testing days. The difference in the latency of the motor response between the proximal and distal stimulation sites, together with the distances between the two sites of stimulation, was used to calculate nerve conduction velocity (Chu-Andrews, 1986). An integrated neurostimulator/amplifier/storage oscilloscope (Cadwell 5200) was used for all nerve stimulation and latency response measurements. Nerve distance was measured using metal calipers. The closest two values of three trials were averaged and taken to represent nerve conduction velocity for each session (Kamenet al. 1984).

Strength Measurements

Force exerted by the left leg was measured on an isokinetic dynamometer (Cybex) interfaced with a microcomputer using ATCODAS signal processing software (DATAQ). Maximal voluntary contractions at 0, 1.05, 2.10, 3.14 and 4.19 rad$\cdot$s^{-1} were measured for leg press, leg extension and plantar flexion in order to construct a force-velocity curve for each movement. For all Cybex tests, straps were used to immobilize the upper body and the command "ready-set-go" was used for each contraction. Subjects were instructed to perform each movement as fast and as hard as possible upon hearing "go" and for isometric contractions were required to hold maximal force for a period of 3s. Three repetitions were performed at each speed, with peak torque from the strongest repetition, taken to represent velocity specific strength. Half the subjects completed the testing in the order of leg press-plantar flexion-leg extension, while the other half tested in the opposite order.

Leg Press: Peak torque exerted in the leg press exceeds the maximal torque capacity of the Cybex, therefore it was modified with a gear and chain system as previously described by Vandervoort et al. (1984). Subjects were in a seated position and for isometric tests, strength was measured with the hip and knee at 100°. For concentric contractions subjects started with the knee at 90° (Vandervoort et al. 1984).

Leg extension: Leg extension was measured in the seated position with the hip at 90° and the knee set at 100° for isometric contractions.

Plantar Flexion: Subjects were secured to a Cybex UBXT in the supine position. All contraction velocities were performed with the knee set at 100° and for the isometric contractions the ankle was also set at this angle.

Rate of Torque Development

For both leg extension and plantar flexion the rate of torque development (RTD) was calculated from the isometric force-time curve. Force data was sampled at 2000 Hz and the first derivative of each force curve, smoothed by a factor of 7 was taken to provide a measure of RTD expressed as $Nm \cdot s^{-1}$. The smoothing factor of 7 resulted in slopes being calculated from 7 points over a duration of 3.5 ms. Mean RTD between 30 and 60% of peak torque was calculated.

Multi-joint Power

Fifteen minutes post strength testing, subjects performed a 15s power test on a cycle ergometer (Monark) equipped with toe clips and racing bars. A photoelectric cell registered every 45 degrees of flywheel revolution and was interfaced with an electronic timing device in order to accurately count flywheel revolutions during the test. Subjects were given a two minute warm-up at 50 Watts after which time they were told to increase their revolutions to a maximum as fast as possible. Simultaneously, the flywheel resistance was set at 95 g/kg of body weight, rounded to the nearest quarter Kp. Once the resistance was set a verbal signal was given and the 15s test began. Peak 1s power, and mean 5, 10 and 15s power values were calculated in Watts (W) and also expressed relative to body mass (W/kg) and thigh volume (W/cm^3 x 10^3).

Muscle Biopsy

Eight subjects from the control, six from the endurance and 10 from the power groups reported to the lab on a fourth testing day for the muscle biopsy procedure. A muscle sample was taken from the vastus lateralis of the left leg using the percutaneous needle-biopsy technique (Bergstrom, 1962) with the addition of suction in order to facilitate the acquisition of an adequate muscle sample (Evans et al. 1982). The biopsy procedure was administered by a physician experienced in this technique. The depth of the biopsy sample was standardized so that the error associated with generalizing a single biopsy to the entire muscle was minimized (Lexell et al. 1985; Elder et al. 1982).

Immediately following the biopsy, the muscle sample was mounted on a cork with the fibers oriented in a vertical position. The muscle samples were then rapidly frozen in isopentane cooled to -160 °C with liquid nitrogen and stored at -70 °C until histochemical analysis (Martin et al. 1988).

Histochemistry

The properties of single fibers in the biopsy sample were determined from tissue sections cut (10 um thick) using a cryostat maintained at a temperature of -20 °C. Fibers were histochemically stained for myofibril ATPase activity using an alkaline preincubation (pH 10.0) as described by Brooke & Kaiser (1970). Visual inspection of the staining intensity allowed subsequent classification of the muscle fibers as Type I (light) or Type II (dark). The fiber-type composition of the biopsy sample was then calculated and corresponding cross-sectional areas determined for each fiber type by video-scanning each biopsy preparation into a microcomputer using image analysis software (Optimas). The areas of all Type I and II fibers present in one field of view were calculated by tracing each fiber with a mouse that had been previously calibrated to a stage micrometer.

Statistics

For each of the strength, power, and histochemical variables One-Way Analysis of Variance followed by Neumann-Keuls post-hoc comparisons were utilized to test for differences between groups. A similar model was used for NCV, however height and age were used as covariates since these factors account for inter-individual differences in NCV (Chu-Andrews, 1986; Dorfman & Bosley, 1979). The magnitude of relationship between the multi-joint power and other neuromuscular variables was assessed using Pearson Product Moment Correlation. For all statistics, significance was set at $p<0.05$.

Results

The endurance athletes were older than the control and power groups and carried less fat than the controls, while the power athletes were taller, heavier and had larger thigh volumes than both the endurance and control groups (Table 1). Both Type I and II fiber cross-sectional areas were not significantly different between groups, however there was a trend in both fiber types for the endurance athletes to have the smallest fiber size, and for larger type II fibers in the power athletes relative to the other groups, as well as to their own Type I fibers, indicated by the larger II to I area ratio in power athletes. (Table 2). The percentage of Type II fibers present in the biopsy sample was not significantly different between groups (Table 2).

The power group was characterized by higher absolute multi-joint powers from 1 to 15s than the endurance and control groups (Figure 1). This power difference disappeared when power was expressed relative to body mass (Figure 2) or leg volume (Figure 3). The power group was also stronger than the endurance and control groups at all velocities for the two single joint strength movements of leg extension (Figure 4) and plantar flexion (Figure 5), however in the multi-joint leg-press movement they were stronger only for the isometric contraction and the slow velocity contraction at 1.05 rad/s (Figure 6). The power athletes had higher rates of isometric torque development than the endurance and control groups for both leg extension and plantar flexion (Figure 7). Tibial motor nerve conduction velocity was not significantly different between groups (Figure 8).

Absolute multi-joint power was significantly related to body mass, leg volume and mean Type I and II area but not the percentage of Type II fibers (Table 3). When expressed relative to body mass, power was related only to vastus lateralis Type II cross-sectional area, and when expressed relative to leg volume it was not significantly related to any variable (Table 3). Leg extension and plantar flexion strength at all velocities, as well as RTD for these movements, were related to absolute 5s power as was isometric and high velocity (4.18 rad/s) multi-joint leg press strength (Table 4). No strength variables were related to power when it was expressed relative to body mass or leg volume (Table 4). Leg extension and plantar flexion strength at all velocities and RTD for these movements as well as isometric leg press strength were significantly correlated to body mass and leg volume (Table 5). Type II fiber cross-sectional area in the vastus lateralis muscle was related to knee extensor strength and RTD and both type I and II fiber cross-sectional areas were related to leg press isometric strength (Table 5). NCV and percentage of Type II fibers were not significantly related to any strength variables. The single joint strength values at all velocities were significantly intercorrelated, however only isometric leg press strength was correlated with the single-joint strength measures (Table 6).

Table 1: Mean (SE) physical characteristics of control, endurance and power athletes, (N=10 for each group).

Group	Age (years)	Height (cm)	Mass (kg)	Sum of 8 (mm)	Leg Volume (cm^3)
Control	22.9 (0.9)	180.5 (1.4)	72.3 (1.5)	71.8 (6.3)	4759 (191)
Endurance	26.0* (1.4)	178.6 (1.7)	66.5 (1.9)	51.9† (3.6)	4256 (196)
Power	21.4 (0.3)	188.0* (2.2)	81.1* (2.9)	57.3 (5.6)	5562* (178)

Note: * represents significantly different from other groups, † significantly different from controls ($p<0.05$).

Table 2: Mean (SE) characteristics of the vastus lateralis muscle for control subjects and endurance and power athletes.

Group	Type I XS Area x $10^2(um^2)$	Type II XS Area x $10^2(um^2)$	II/I Area Ratio	% Type II	Fibers Analyzed (number)
Control **(n=8)**	45.7 (2.6)	50.4 (3.3)	1.11 (0.06)	49.9 (4.0)	56 (6)
Endurance **(n=6)**	43.0 (4.6)	48.5 (5.1)	1.15 (0.10)	39.1 (4.9)	58 (7)
Power **(n=10)**	45.6 (2.6)	58.9 (5.2)	1.28† (0.05)	45.8 (3.1)	56 (4)

Note: † represents significantly different from controls.

Figure 1: Mean (SE) absolute power scores from a 15s cycle ergometer test for control subjects, and endurance and power athletes, n=10 each group, * significantly different from other groups ($p<0.05$).

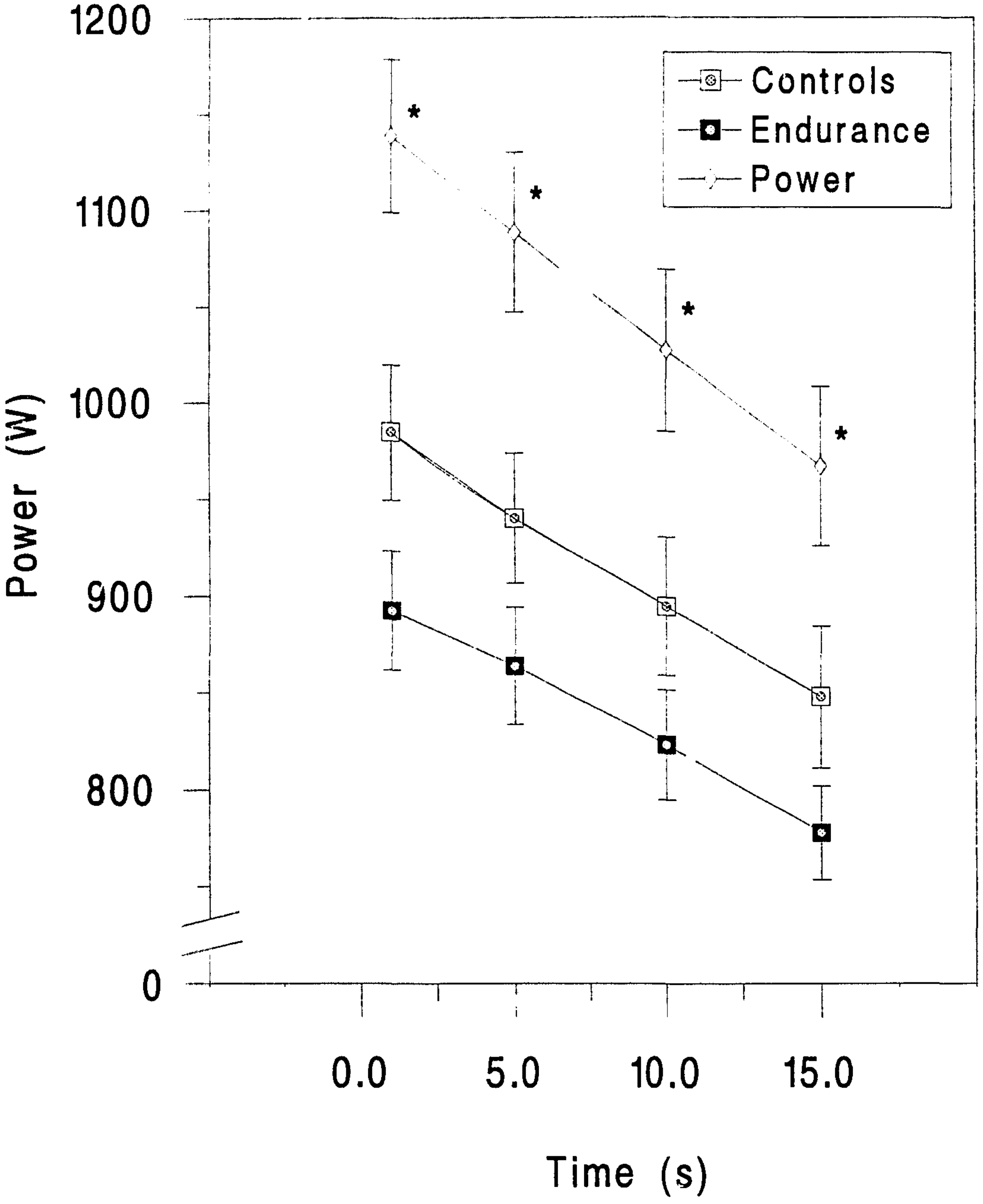
1200
1100
1000
900
800
0
Power (W)
0.0
5.0
10.0
15.0
Time (s)
Controls
Endurance
Power
*
*
*
*

Figure 2: Mean (SE) power/body mass scores from a 15s cycle ergometer test for control subjects, and endurance and power athletes, n=10 each group.

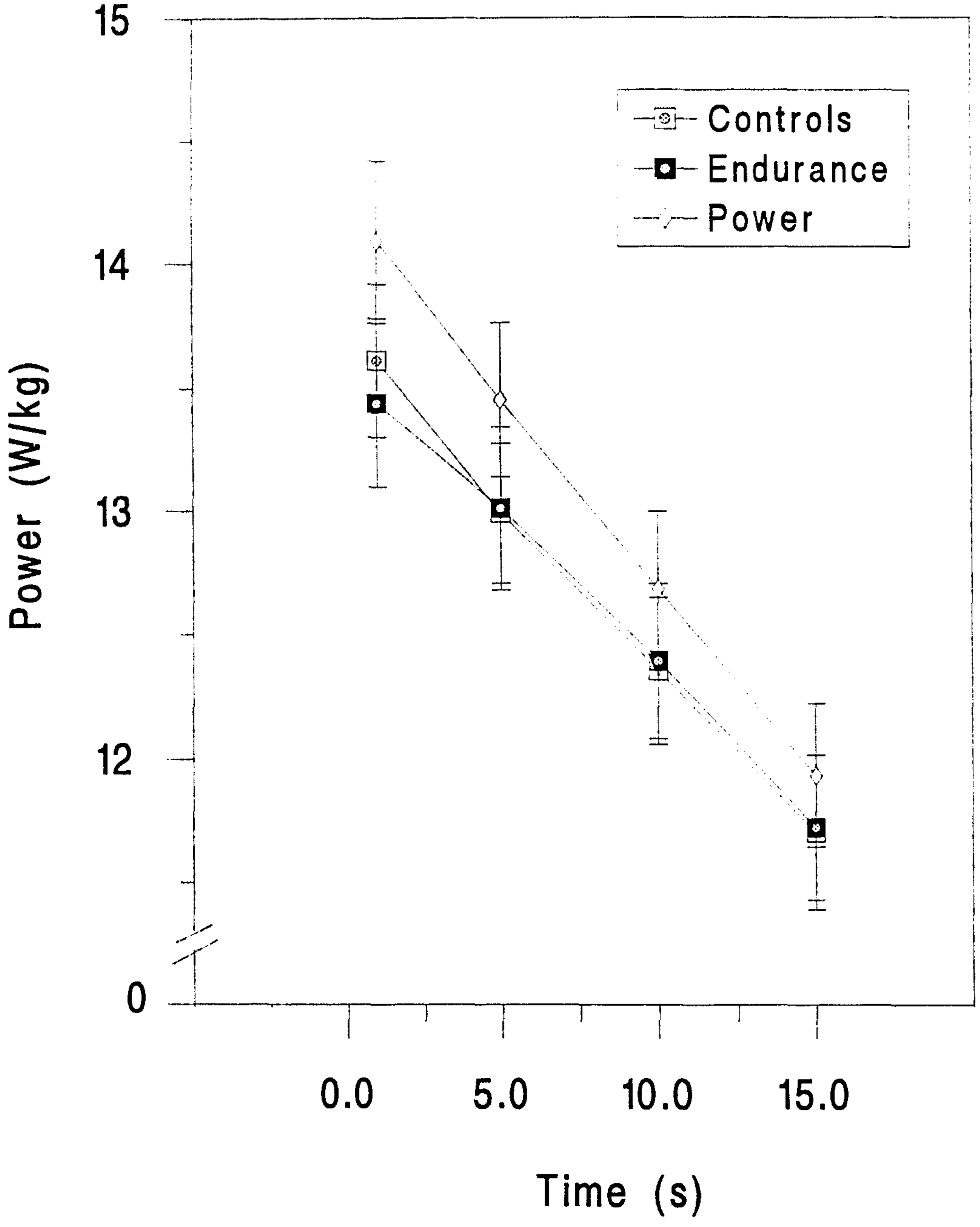
15
14
13
12
0
Power (W/kg)
Controls
Endurance
Power
0.0
5.0
10.0
15.0
Time (s)

Figure 3: Mean (SE) power/thigh volume scores from a 15s cycle ergometer test for control subjects, and endurance and power athletes, n=10 each group.

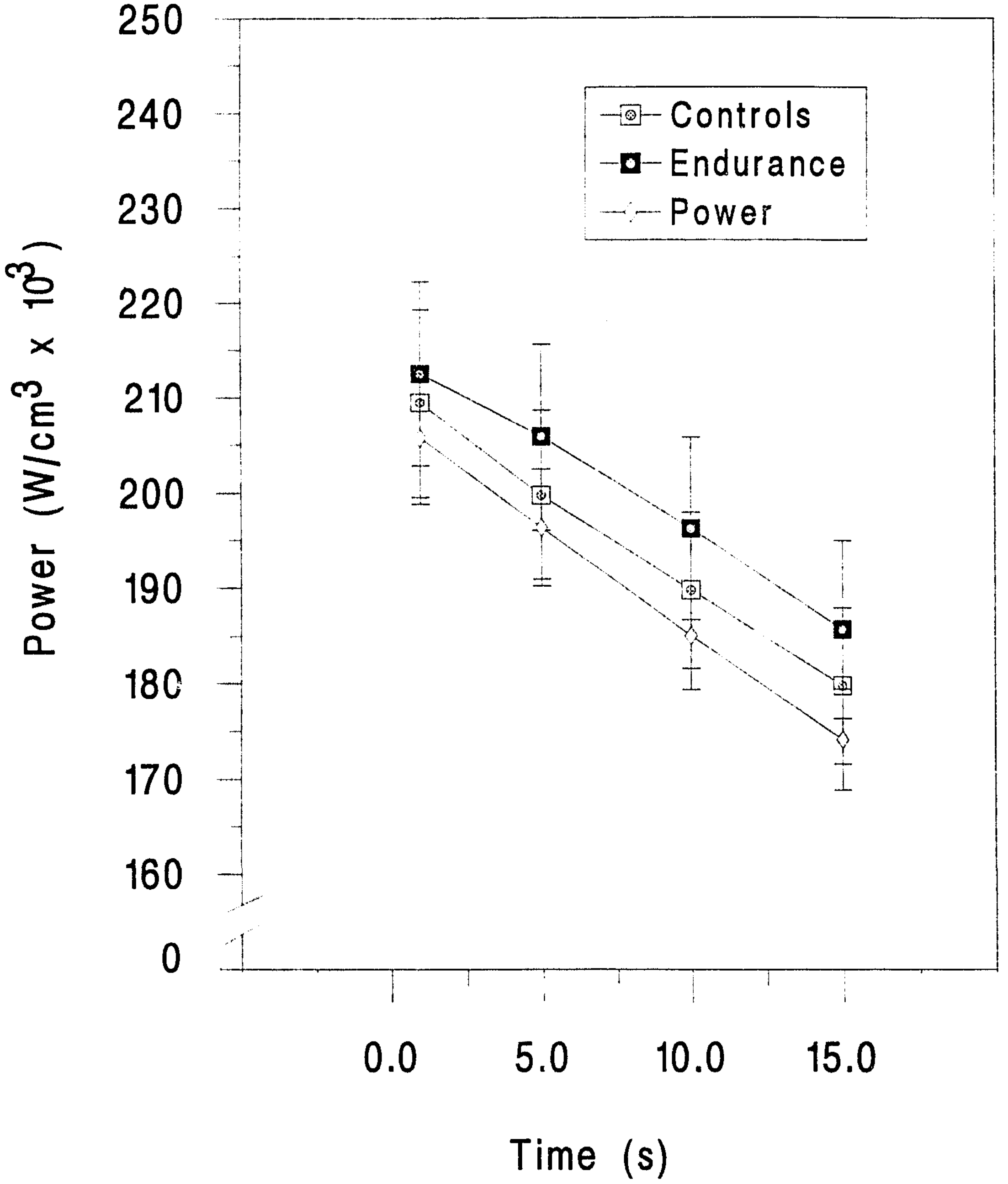
250
240
230
220
210
200
190
180
170
160
0
Power (W/cm^3 x 10^3)
Controls
Endurance
Power
0.0
5.0
10.0
15.0
Time (s)

Figure 4: Mean (SE) leg extension force-velocity curves for control subjects, and endurance and power athletes, n=10 each group, * significantly different from other groups ($p < 0.05$).

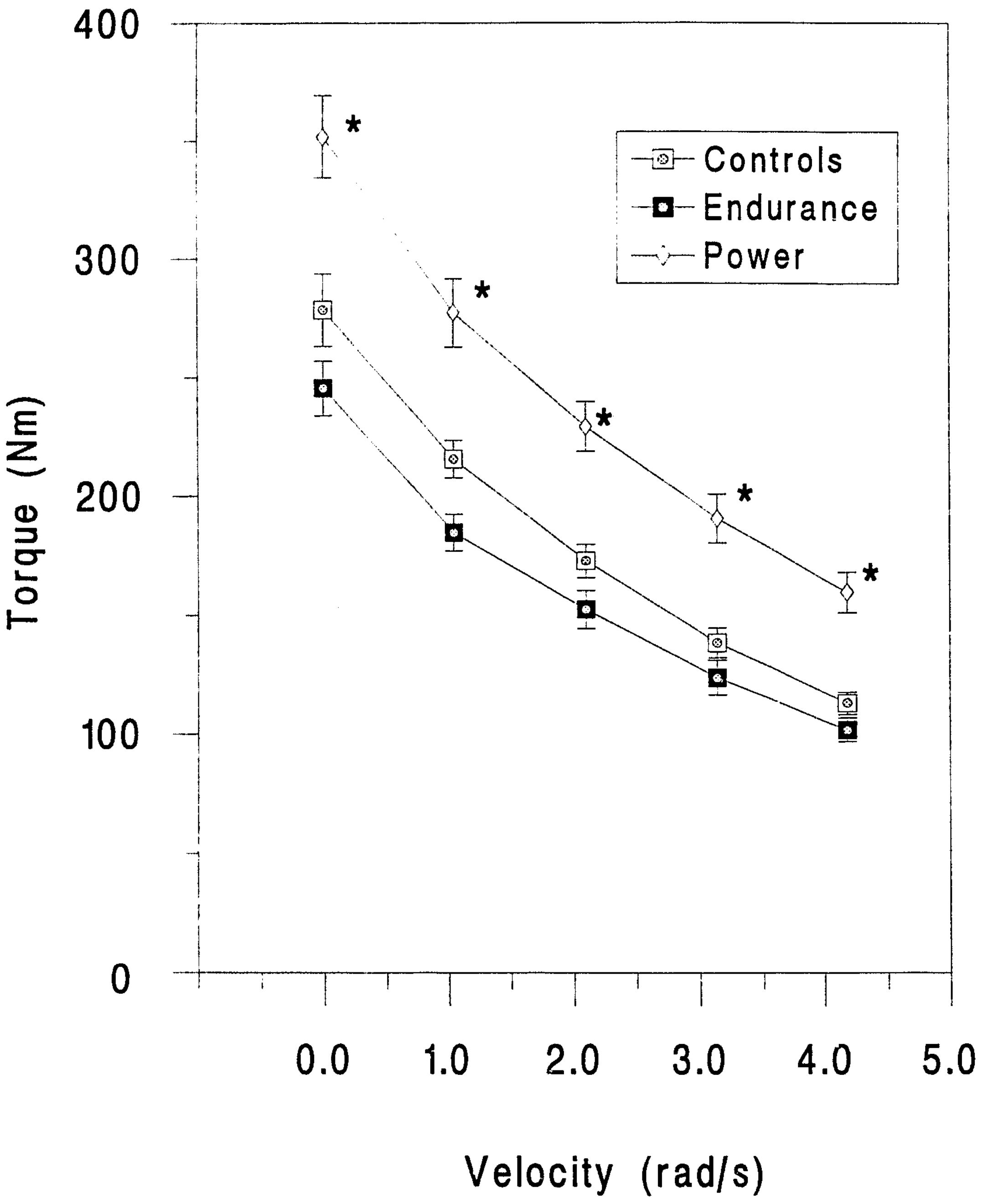
400
300
200
100
0
Torque (Nm)
0.0
1.0
2.0
3.0
4.0
5.0
Velocity (rad/s)
Controls
Endurance
Power
*
*
*
*
*

Figure 5: Mean (SE) plantar flexion force-velocity curves for control subjects, and endurance and power athletes, n=10 each group, * significantly different from other groups, ($p < 0.05$).

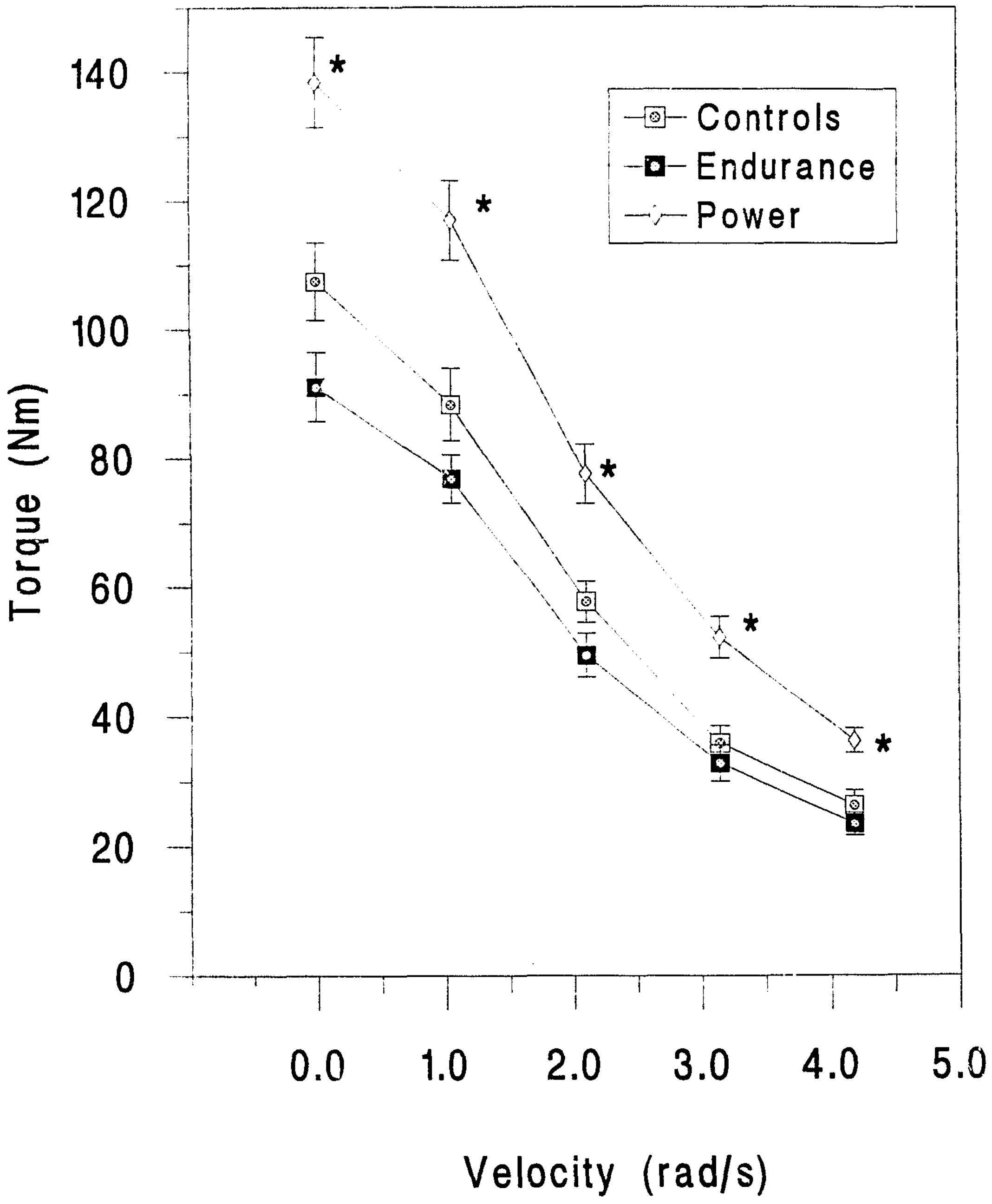
140
120
100
80
60
40
20
0
Torque (Nm)
0.0
1.0
2.0
3.0
4.0
5.0
Velocity (rad/s)
Controls
Endurance
Power
*
*
*
*
*

Figure 6: Mean (SE) leg press force-velocity curves for control subjects, and endurance and power athletes, n=10 each group, * significantly different from other groups ($p<.05$).

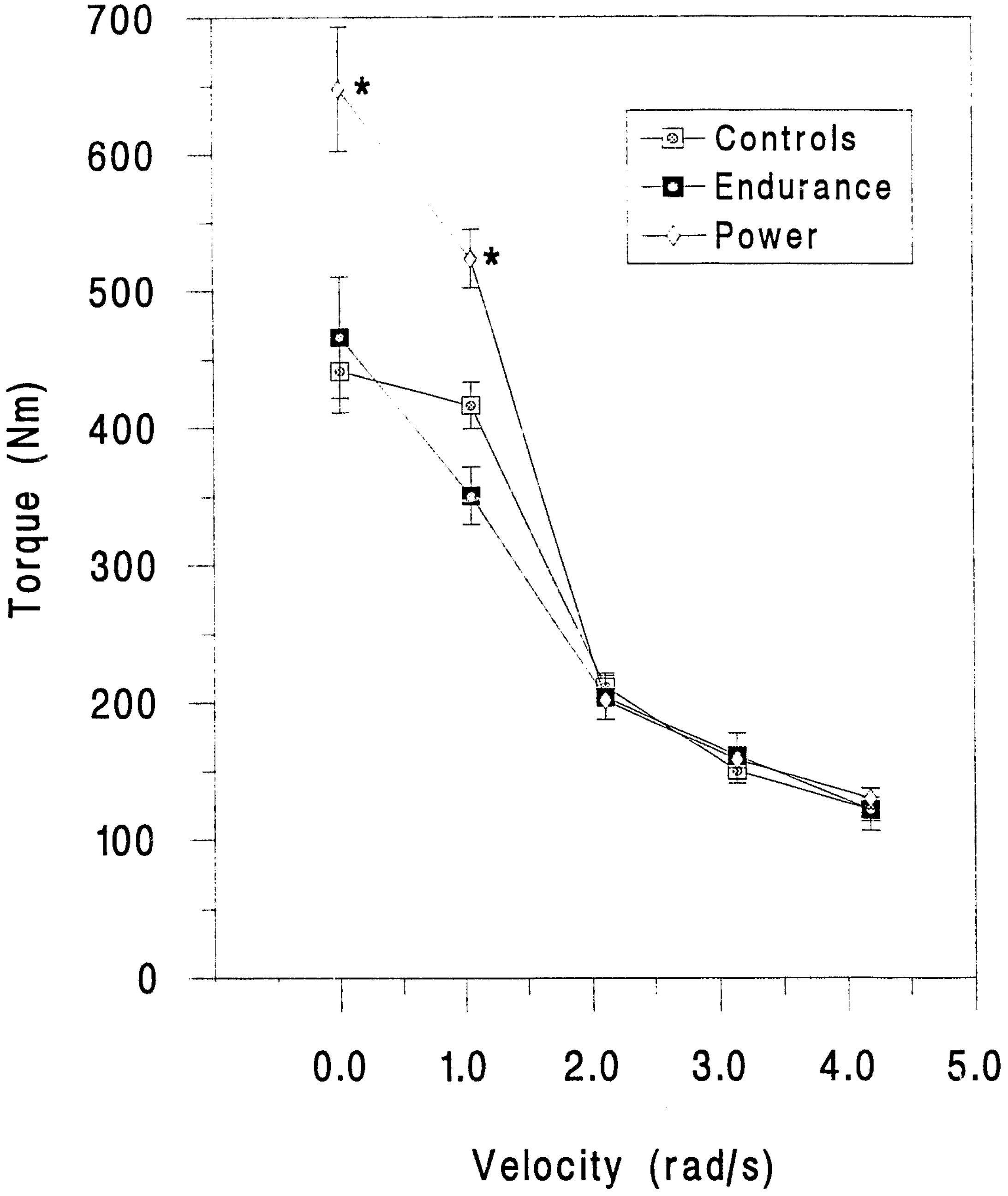
700
600
500
400
300
200
100
0
Torque (Nm)
0.0
1.0
2.0
3.0
4.0
5.0
Velocity (rad/s)
Controls
Endurance
Power
*
*

Figure 7: Mean (SE) rate of isometric torque development (RTD) between 30 and 60 percent of peak isometric torque for leg extension and plantar flexion in control subjects, and endurance and power athletes, n=10 each group, * significantly different from other groups ($p<0.05$).

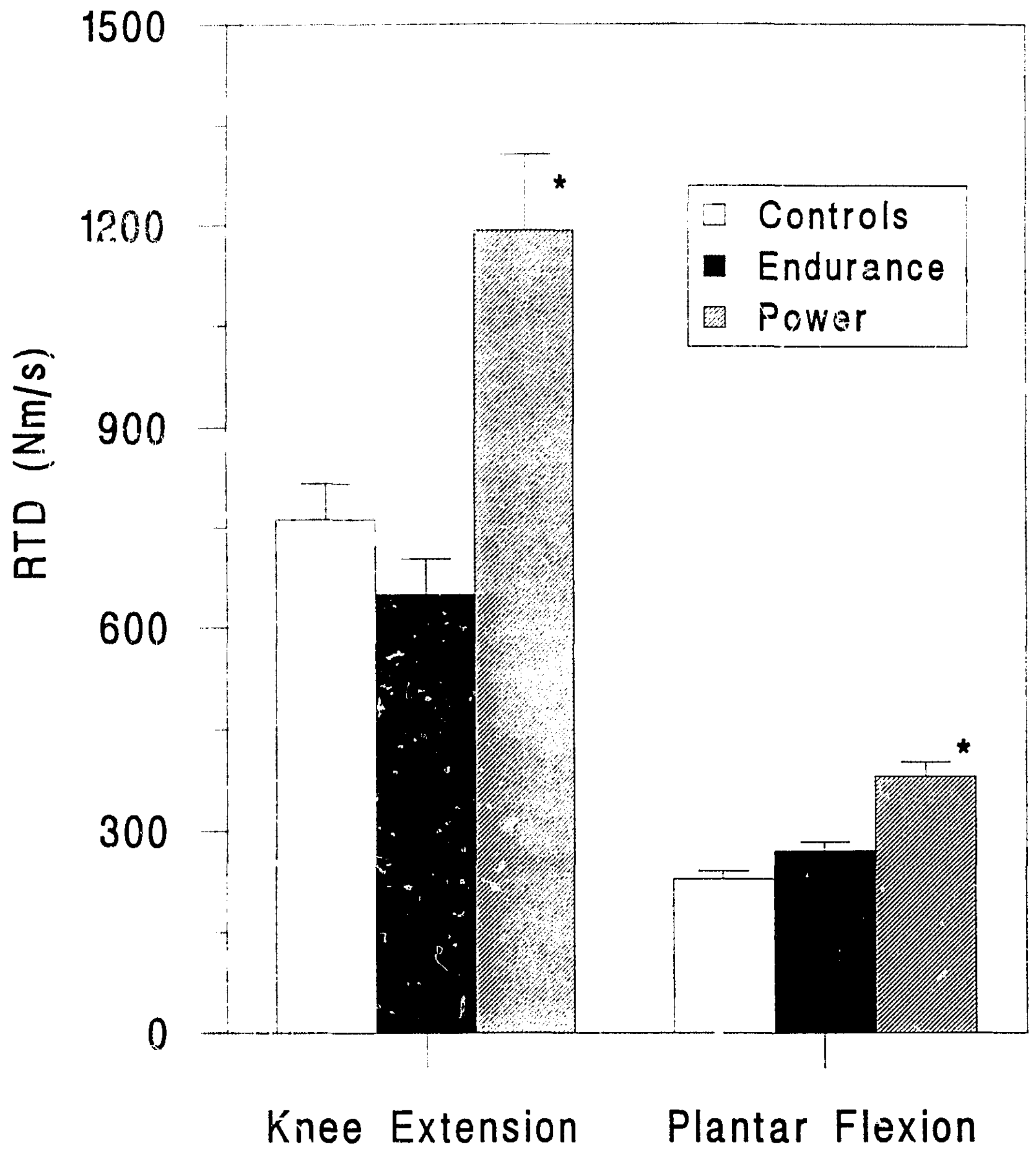
RTD (Nm/s)
1500
1200
900
600
300
0
Controls
Endurance
Power
*
*
Knee Extension
Plantar Flexion

Figure 8: Mean (SE) tibial motor nerve conduction velocity (NCV) in control subjects, and endurance and power athletes, n=10 each group.

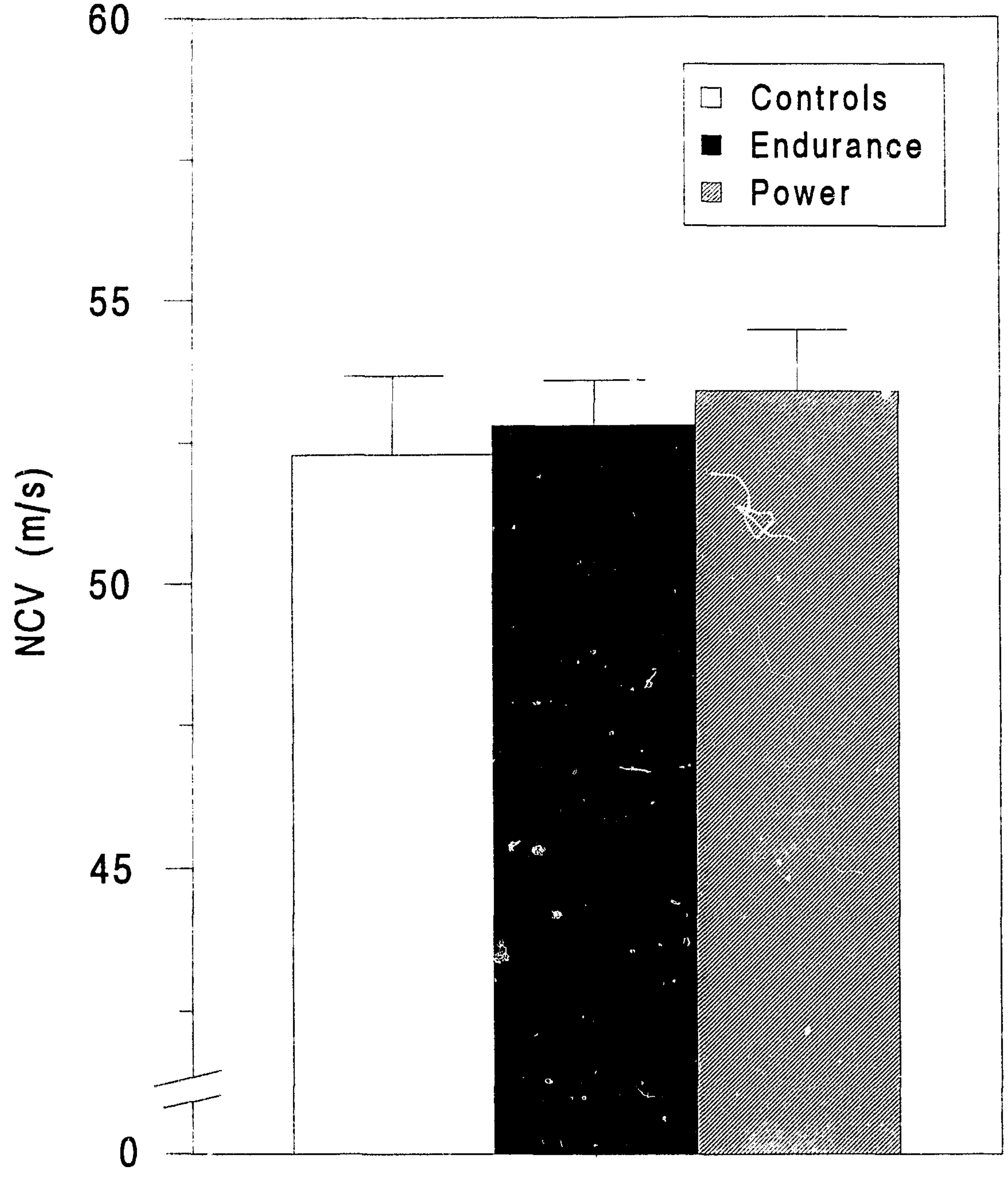
60
55
50
45
0
NCV (m/s)
☐ Controls
■ Endurance
▨ Power

Table 3: Intercorrelations of neuromuscular variables and multi-joint power ($n=30$, $n=24$★).

Neuromuscular Variables	Absolute 5s Power (W)	5s Power/Body Mass (W/kg)	5s Power/Leg Volume (W/cm^3)
Mass	0.86*	0.08	-0.11
Leg Volume	0.73*	0.07	-0.54*
% Type II★	0.09	0.01	-0.06
Mean Type I Area★	0.49*	0.34	0.08
Mean Type II Area★	0.66*	0.56*	0.26
NCV	0.04	0.03	0.10

Note: * ($p<0.05$).

Table 4: Intercorrelations of strength and multi-joint power measures (n=30).

Strength Variables	Absolute 5s Power (W)	5s Power/Body Mass (W/kg)	5s Power/Leg Volume (W/cm^3)
Leg Ext. 0	0.66*	0.26	-0.02
Leg Ext. 2.10	0.80*	0.32	0.05
Leg Ext. 4.19	0.83*	0.37	0.05
Leg Ext. RTD	0.60*	0.24	0.05
Plantar 0	0.63*	0.19	0.04
Plantar 2.10	0.70*	0.29	0.04
Plantar 4.19	0.60*	0.32	-0.02
Plantar RTD	0.61*	0.39	0.13
Leg Press 0	0.57*	0.39	0.15
Leg Press 2.10	0.20	0.24	0.01
Leg Press 4.19	0.41*	0.24	-0.06

Note: * ($p < 0.05$).

Table 5: Intercorrelations of strength and neuromuscular variables (n=30, n=24★).

Strength Variable	Mass	Leg Volume	% Type II★	Type I Area★	Type II Area★	NCV
Leg Ext. 0	0.66*	0.60*	0.18	0.20	0.43*	0.29
Leg Ext. 2.10	0.78*	0.73*	0.14	0.23	0.48*	0.21
Leg Ext. 4.19	0.77*	0.71*	0.12	0.31	0.54*	0.18
Leg Ext. RTD	0.59*	0.49*	0.03	0.09	0.46*	0.10
Plantar 0	0.67*	0.53*	0.26	0.01	0.18	0.14
Plantar 2.10	0.68*	0.58*	0.18	0.16	0.28	0.06
Plantar 4.19	0.54*	0.54*	0.17	0.21	0.28	-0.01
Plantar RTD	0.50*	0.42*	0.15	0.15	0.30	0.27
Legpress 0	0.47*	0.39*	0.08	0.43*	0.65*	0.19
Legpress 2.10	0.10	0.18	-0.16	-0.16	-0.05	0.21
Legpress 4.19	0.35	0.37	-0.06	-0.04	-0.02	0.17

Note: * $(p<0.05)$

Table 6: Intercorrelations of strength variables (n=30).

		Leg Extension				Plantar Flexion				LegPress		
		0	**2.10**	**4.19**	**RTD**	**0**	**2.10**	**4.19**	**RTD**	**0**	**2.10**	**4.19**
Leg Extension	**0**											
	2.10	0.88*										
	4.19	0.82*	0.97*									
	RTD	0.77*	0.84*	0.81*								
Plantar Flexion	**0**	0.68*	0.65*	0.63*	0.43*							
	2.10	0.65*	0.74*	0.77*	0.53*	0.83*						
	4.19	0.53*	0.65*	0.68*	0.43*	0.78*	0.91*					
	RTD	0.58*	0.65*	0.73*	0.53*	0.70*	0.69*	0.70*				
Leg Press	**0**	0.65*	0.72*	0.74*	0.59*	0.43*	0.49*	0.57*	0.58*			
	2.10	0.23	0.19	0.10	0.10	0.05	0.05	-0.01	-0.09	-0.15		
	4.19	0.34	0.38	0.32	0.15	0.18	0.28	0.21	0.06	0.08	0.82*	

Note: * $(p<0.05)$

Discussion

The main finding of this study is that the higher absolute leg power values observed in power athletes versus endurance athletes and controls disappear when expressed relative to body mass or thigh volume. This implies that power differences between these groups are due to differences in muscular variables and not neural factors. Contrary to this finding, other studies have reported that power athletes maintain a power advantage over endurance and control subjects even when power is expressed relative to body mass (Smith & Stokes, 1985; Serrese et al. 1989; Bouchard et al. 1991). Although not statistically significant, the present data also show this trend, yet when power is corrected for thigh volume, the trend disappears and endurance athletes show slightly higher power (NS). Expressing leg power relative to body mass may be important in those activities where the body mass must be carried or propelled but it can also be misleading when examining physiological mechanisms contributing to power expression. It is possible that in those athletes with high leg-power/body-mass ratios the legs can contribute disproportionably to body mass, giving the impression that qualitative muscular differences or neural factors are influencing power generation, when in fact it is the larger and stronger leg muscles responsible for extra power production.

The lack of difference between groups when power was expressed relative to body mass or thigh volume is partially explained by the significant relationships of body mass, thigh volume and Type I and II fiber area to absolute 5s power output. Type II fiber area seems to be especially important since it is still significantly related to power after correction for body mass differences. This is consistent with the known properties of Type II motor units which include higher tension outputs and shorter time to peak tension (Edgerton, 1976). Others have also reported significant relationships between body mass (Goslin & Graham, 1985) or thigh volume (McCartney et al. 1983) and maximal power production on a cycle ergometer. Larger versus smaller muscles are able to produce greater amounts of force (Goldspink, 1991; MacDougall, 1991), thereby allowing individuals to better overcome an external load, such as that provided by constant load cycle ergometry, and generate more power.

The large thigh volumes in the power athletes when compared to the controls and endurance groups could be due to genetic or environmental (training) influences. Theoretically, muscle size is determined by both the number and size of muscle fibers, and the amount of interstitial connective tissue (MacDougall, 1991). The amount of subcutaneous fat on the thigh does not impact the thigh volume values in this study since it is factored out in the volume calculation, however no estimation of interstitial connective tissue was made. Nevertheless, connective tissue differences would not be expected between groups since previous research has shown, that in individuals of different training backgrounds the amount of connective tissue present in a limb is extremely consistent and proportional to muscle mass (MacDougall et al. 1984).

The number of muscle fibers in the vastus lateralis was not calculated in the present study. By dividing muscle fiber cross-sectional area into muscle cross-sectional area others have estimated muscle fiber numbers in different groups. Johansson et al. (1987) calculated equal numbers of muscle fibers in the vastus lateralis muscles of sprinters and marathon runners, and MacDougall (1984) found that elite bodybuilders had similar numbers of fibers to untrained controls. Other studies using single fiber electromyography techniques have estimated bodybuilders to have greater numbers of muscle fibers than controls (Tesch & Larsson, 1982; Larsson & Tesch, 1986) and Sjostrom et al. (1991) reported higher numbers of muscle fibers in the dominant versus non-dominant hand of young healthy adults, suggesting a long-term hyperplasia response. Therefore, it is possible that muscle fiber number may vary between individuals, due to genetic or environmental factors, and therefore impact muscle size. Since in the present study muscle fiber size was not significantly different between groups but power athletes had larger fat free thigh volumes than the endurance and control groups, it is tempting to speculate that the power athletes may have had a greater number of muscle fibers. Although muscle fiber area was not statistically different between groups, mean fiber area in the power athletes was 29% higher than the endurance and 17% higher than control groups. This is of a similar magnitude to thigh volume differences between groups since power athletes were 31% higher than endurance and 17% higher than controls groups. Both Type I (r = 0.40) and Type II (r = 0.46) fiber area were also significantly correlated to thigh volume. The thigh volume calculation not only accounts for cross-sectional area of the thigh but also the length of the thigh, and height was significantly correlated to thigh volume (r = 0.56). If one assumes that taller individuals also have longer muscles it would appear that thigh volume differences between groups are probably due not only to muscle fiber cross-sectional area differences but also muscle length differences. A greater number of sarcomeres in parallel (muscle cross-sectional area) improves the force generation characteristics of a muscle while an increased number of sarcomeres in series (muscle length) enhances the peak velocity of muscle shortening (Goldspink, 1991). Since high power outputs require both fast and strong contractions it can be hypothesized that the combination of greater muscle fiber cross-sectional area and muscle length combine to account for the higher power outputs in the power versus endurance and control groups.

The fiber type profile may also impact muscle size since Type II muscle fibers are usually larger than Type I fibers (Edgerton, 1976). This is not supported in the present study however since the percentage of Type II fibers was not different between groups. The IIa and IIb fiber types were not differentiated and it is possible that differences in Type II subtypes could account for some muscle size differences.

The Type II/I fiber area ratio was higher in the power group than in the controls. Whether this is a genetic characteristic of the power athletes or a result of long-term power training is equivocal. Longitudinal training studies have reported preferential Type II hypertrophy (Thorstensson, 1976; MacDougall et al. 1979) and cross sectional studies have also reported power athletes to have larger Type II/I fiber area ratios than endurance athletes (Tesch et al. 1989;Mackova et al. 1985; Schantz et al. 1983; Tesch & Karlsson, 1985). It has been suggested that preferential hypertrophy of Type II fibers may reflect a greater relative involvement of these high threshold units with respect to daily activity in power or strength activities (MacDougall, 1991).

Some researchers have reported maximal cycle ergometer power outputs from the Wingate test to be positively related to Type II fiber composition of the vastus lateralis (Bar-Or et al. 1980; Froese & Houston; 1987). This agrees with a number of studies that have observed fiber type compositions in athletes consistent with the demands of their sport (Gollnick et al. 1972; Clarkson, 1982; Tesch & Karlsson, 1985). These same studies and others (Saltin et al. 1977; Viitasalo & Komi, 1978; Gregor et al. 1981) have also reported a wide range of fiber types within groups of both power and endurance type athletes. The data of the present study also found a wide range within groups of fiber type profiles and the percentage of Type II fibers was not different between groups. Further, no relationship was observed between Type II fiber composition and maximal power on the cycle ergometer. However, the significant correlation between Type II fiber area and absolute, as well as body mass corrected, power indicate that power athletes with lower percentages of Type II muscle fibers may still be able to generate high power outputs if their Type II muscle fibers are sufficiently large. They may be born with this characteristic or specific power training may cause preferential Type II hypertrophy (MacDougall et al. 1979; Gollnick et al. 1972). This is consistent with the findings of Patton et al. (1990) who observed no relationship between Type II composition and cycle ergometer power and concluded that power production was more strongly related to body size. McCartney et al. (1983) also concluded that muscle size was more important than muscle fiber type in the production of maximal power during isokinetic cycle ergometry. The literature is therefore equivocal, and suggests that both muscle fiber size and composition can influence power production. The modality of testing power output (constant velocity cycle ergometry versus constant load cycle ergometry), limitations of estimating muscle fiber composition from needle biopsy samples (Elder et al. 1982; Lexell et al. 1985), or differences in subject characteristics (training status, heterogeneity of sample) may ultimately determine which characteristic is most influential in a given situation. A combination of a high percentage of Type II muscle fibers and large Type II muscle fibers might prepare the athlete for maximal power production.

Consistent with the power and muscular differences exhibited between power, endurance and control groups, the power athletes also showed higher levels of leg extension and plantar flexion strength at all velocities as well as higher isometric and low velocity (1.05 rad·s^{-1}) leg press strength. These strength measures were intercorrelated, except for the high velocity leg press strength values which may indicate that performance of this high velocity multi-joint movement is not influenced by strength per se, but by some other factor. Higher velocity leg press strength (2.10-4.19 rad·s^{-1}) was also not different between groups. The velocity for leg press was set in order to control the speed of the lever arm. Angular velocities of the hip, knee and ankle joints were not measured but they would be different than that of the lever arm. Since the leg is pushing linearly against the lever arm the angular velocities of the ankle, knee and hip joints would have to be faster than that set for the lever arm in order to engage the Cybex servo-motor. At these fast angular velocities, torque and power may be too low to differentiate between groups or alternatively some individuals may have had difficulty in achieving the very fast movements.

Many other studies have noted similar strength differences to those seen in this study between power/strength and endurance athletes (Gregor et al. 1981; Clarkson et al. 1980; Taylor et al. 1991; Johansson et al. 1987; Schantz et al. 1983) and between groups split on the basis of fiber type profiles (Coyle et al. 1979; Suter et al. 1993). A common finding in these studies has been increasing torque and power differences between groups as the velocity of contraction increases due to the power group maintaining higher torque generation. The ability to maintain a high percentage of isometric strength at high velocities has been related by some researchers to a higher percentage of Type II motor units (Thorstennson et al. 1976; Coyle et al. 1979; Ryushi & Fukanuga, 1986; Suter et al. 1993). Others have noted no relationship with Type II fiber composition (Gregor et al. 1981; Clarkson et al. 1982; Schantz et al. 1983; Froese & Houston, 1985) and it has been suggested that heterogeneity in training background may account for some of these results since velocity specific strength adaptation is known to occur (Caiozzo et al. 1981; Coyle et al. 1981). Others have shown that a high percentage of Type II fibers do not enable an individual to generate more torque at high velocities but allow earlier generation of peak torque within a movement (Schantz et al. 1983). This may cause peak torque to be generated at a more optimal joint angle and allow individuals with high percentages of Type II fibers to utilize a mechanical advantage. Froese & Houston (1985) reported that, as velocity of contraction increased, peak torque was produced later in the movement, however there was no correlation of this angle with the percentage of Type II muscle fibers. In the present study the torque differences between groups did not increase with increasing velocity of contraction. Schantz et al. (1983) observed similar results when comparing physical education students and bodybuilders, although the latter cannot be considered power athletes. In agreement with the findings of others (Gregor et al. 1981; Clarkson et al. 1982; Schantz et al. 1983; Froese & Houston, 1985), no significant correlations between Type II fiber composition and torque at any velocity was observed in the present study. When this result is combined with the significant correlations between the strength variables and body mass, thigh volume and Type II fiber area the results suggest that muscle size, and especially Type II fiber size is the most important determinant of torque generation at any velocity.

Very little work has examined the relationship between isokinetic strength variables measured on a dynamometer and power measured on ergometers that require multiple muscles working across multiple joints for power production. Smith (1987) reported significant relationships between isokinetic strength measures (hip flexion, knee flexion, knee extension) and power output in a 30s cycle ergometer test. Significant correlations have also been reported between both slow and fast velocity leg extension torque, and maximal power generated in rowing; however, in this same study no improvement of anaerobic rowing performance occurred after 5 weeks of either slow or fast isokinetic strength training showing that isokinetic training may not necessarily impact multi-joint power production (Bell et al. 1989). Another study has shown improvements in 30s mean power on the cycle ergometer after 5 weeks of isokinetic strength training in swimmers, possibly due to a neural adaptation since, unlike the rowers in the study of Bell et al. (1989) the swimmers were unfamiliar with both the strength and cycle tasks (Petersen et al.1984). This previous work has utilized muscle function tests of single joint movements on the dynamometer that utilize similar muscles recruited on the multi-joint ergometer but do not mimic multi-joint movement patterns (ie. proximal to distal muscle sequencing). With this in mind, isokinetic strength in the present study was tested in two single-joint movements (leg extension and plantar flexion), and one multi-joint movement utilizing proximal to distal sequencing (leg-press) thought to be more specific to cycle ergometry. As in the study of Bell et al. (1989), the strength variables were strongly correlated with absolute 5s power output, but there was no specificity of movement or neural effect since strength in all movements correlated equally well to cycle ergometer power production. Body mass and thigh volume were related to isokinetic strength generation in all movements, and Type II fiber area in the vastus lateralis was related to those movements where the vastus lateralis is considered a prime mover (leg extension, leg press strength), re-enforcing the importance of muscle size and strength to the generation of high force. It appears then that the primary mechanism of transferring strength to power production lies in muscle size and force generation potential, independent of a neural effect.

Besides strength, the rate of developing force may be equally important in those activities where force or power must be generated very quickly. For example Viitasalo et al. (1981), reported that vertical jump height was not related to peak force generation but was related to the rate of force development in both plantar flexion and leg extension. In the present study, higher relative rates of torque development (RTD) between 30 and 60 percent of peak isometric torque in both plantar flexion, and leg extension were observed for the power athletes when compared to both controls and endurance athletes. Several other researchers have reported similar findings (Hakkinen & Keskinen. 1989; Viitasalo & Komi. 1978; Viitasalo et al. 1981). RTD was also significantly correlated with 5s maximal power on the cycle ergometer, although of no greater magnitude than correlations of power with peak torque measurements. Even though RTD in the present study was measured relative to isometric peak torque, it was still significantly related to peak torque at every velocity in both leg extension and plantar flexion, therefore some common factors may influence both RTD and strength. This is supported by the significant relationships observed between RTD and body mass, leg volume and Type II cross-sectional area, variables that were also related to strength. This was not the case in the study by Viitasalo et al. (1981) and has not been reported in other studies.

Given the mechanical properties of Type II muscle fibers, their proportion in a muscle might also be expected to influence the RTD yet there was no significant relationship between Type II fiber composition and RTD in this study. Others have noted weak (r=0.44-0.50) but significant correlations between Type II fiber composition and RTD (Viitasalo et al. 1981; Viitasalo & Komi, 1978) and also suggested that preferential Type II hypertrophy could positively affect the RTD (Hakkinen & Komi, 1986). That Type II fiber area was related to RTD in the present study, gives some support to the latter suggestion however differences in the populations studied and their training backgrounds make the influence of fiber type percentage on RTD unclear.

The importance of a neural influence on RTD has also been suggested (Hakkinen & Komi, 1986). These researchers suggested that higher firing frequencies or altered recruitment patterns could influence RTD although they could not demonstrate a significant change in the EMG/time curve to parallel improvements in RTD with training. In the present study, training background differences must be considered as one possible reason for group differences in RTD, since volleyball players are routinely exposed to explosive type jump work. This type of training has previously been shown to elicit considerable increases in muscle activation levels (Hakkinen & Komi, 1985; Schmidtbleicher et al. 1988) and qualitative changes in recruitment patterns (Schmidtbleicher et al. 1988). Further, by examining monozygotic and dizygotic twins, the genetic influence on RTD has been estimated to be quite low (Viitasalo & Komi, 1978) therefore training status should play a significant role in determining RTD.

It is known that both electrophysiological and metabolic properties of motoneurons are consistent with the properties of the types of muscle fibers they innervate (Gardiner, 1991) and there is some evidence that the motoneuron will adapt to altered functional demand (Edgerton et al. 1980; Roy et al. 1983). Therefore, conduction velocity of the tibial nerve (NCV) was measured in order to determine whether differences existed between groups that could account for force or power differences. No significant differences were observed between groups and NCV was not related to any other variables. Kamen et al. (1984) also found no differences in NCV when 10 different groups of athletes were collapsed into power and endurance type groups. Only in very specific and extreme comparisons (weight lifters vs marathon runners) was NCV different. Upton & Radford, (1975) reported that elite track sprinters have slower NCV than untrained controls, and similarly Kamen et al. (1984) found jumpers and male sprinters to have NCV values slower than the mean of all other subjects tested. Why NCV would be lower in power versus endurance type athletes is not clear. However, Elam (1985) tested football players and reported no significant relationship between NCV and strength, but a significant inverse relationship between NCV and vertical jump power ($r = -0.50$). He speculated that a greater number of terminal branches from the descending alpha motoneuron would recruit auxiliary motor units and enhance muscle force production, but reduce conduction velocity through dispersion of the action potential. The heritability of NCV was investigated by Komi et al. (1973) and estimated to be low, therefore environmental factors such as training may play a substantial role in determining NCV and indeed higher NCV values have been reported in humans as a result of strength training after immobilization (Sale et al. 1982). Cross-sectional studies with greater statistical power, and longitudinal training studies are required in order to clarify the exact role of NCV in power production. In addition, the traditional double stimulation technique for the measurement of NCV only monitors the velocity of the fastest nerve fibers in a nerve (Chu-Andrews, 1986). This technique may not be sensitive enough, and new methods such as the Collision Technique (Arasaki et al. 1991) which measures the distribution of fiber conduction velocities within a nerve may be better able to determine whether true NCV differences exist between power and endurance athletes, and whether this variable is important to power production.

In summary the results of this cross-sectional study show that power athletes demonstrate higher absolute power outputs on a cycle ergometer than endurance and control subjects, but when corrected for body mass or leg volume these power differences disappear. It is suggested that differences in muscle size, and especially Type II muscle fiber area, but not fiber composition, largely account for the observed differences in absolute power through improving strength. Since these variables also correlate with strength, and since it does not matter whether or not the strength movement simulates the power movement (ie. single vs multi-joint, proximal/distal sequencing of muscle recruitment) no neural effect is suggested. This implies that the act of performing a strength movement versus a power movement is sufficiently different, and that simulating the pattern of movement (ie. proximal to distal sequencing) will not influence power acquisition through a neural training effect. It may therefore be more effective to concentrate on enlarging the size of muscles that will be utilized in the power movement by isolating them in single-joint movements and then learning to appropriately recruit those muscles with specific power training. This remains to be confirmed in a longitudinal training study.

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Chapter 3

Sprint Training and single vs multi-joint strength training: their influence on multi-joint power acquisition

ABSTRACT

The primary purpose of this study was to determine whether neural and muscular adaptation, as well as power acquisition, differed between single-joint strength training, multi-joint strength training and sprint training. A secondary purpose was to determine the effect of sequencing 8 weeks of strength training (single or multi-joint strength) prior to sprint training (6 weeks) versus sprint training alone (14 weeks) on multi-joint power acquisition (cycle ergometer). 32 male subjects, age 20-28, were randomly assigned to either control (C), sprint-sprint (SS), multi-joint strength-sprint (MJS) or single-joint strength-sprint groups (SJS), n=8 each group. The physical characteristics of the subjects did not change over the 14 week study in any group (mean (SD) height 178.2 (6.5) cm, body mass 76.8 (9.5) kg, sum of 8 skinfolds 91.0 (38.2) mm, thigh volume 5370 (894) cm^3). Subjects were tested prior to beginning training, mid-training at the end of 8 weeks of either single or multi-joint strength training or sprint training, and post training after another 6 weeks of sprint training. At each time they underwent physiological testing including determination of tibial nerve conduction velocity (NCV), isometric and isokinetic strength (1.05-4.19 $rad \cdot s^{-1}$) for leg press, leg extension and plantar flexion, integrated electromyography (IEMG) for these movements (0,1.05 $rad \cdot s^{-1}$), mean rate of isometric torque development (RTD) for leg extension and plantar flexion, and a 15s cycle ergometer power test (load = 95g/kg of body mass). In addition, SJS and MJS, were tested weekly, and C tested pre, mid and post training for 10 RM strength on the Universal weight training equipment. A sub-sample of subjects also received biopsies (m. vastus lateralis) pre, mid and post training and their muscle was histochemically analyzed. At mid-test both SJS (43.6%) and MJS (41.1%) were stronger than pre-training (mean 10 repetition maximum strength) on the training equipment, but this strength was not transferable to isometric or isokinetic Cybex strength or RTD, and likewise SS showed no improvements. All training groups did however increase multi-joint power output by 8 weeks (eg. 5s mean power output increases, SS = 7%, SJS = 4%,MJS = 4%, C = -4%) and showed similar Type I and II fiber hypertrophy (SS=8.4%, SJS=13.5%, MJS=11.7%) however IEMG did not change. With a subsequent 6 week period of sprint training power continued to increase, but not differentially between groups (increase from pre-test: SS = 11%, SJS = 6%, MJS = 7%, C = -4%), there was no further muscle hypertrophy, no IEMG changes but NCV had increased significantly in all training groups by a small amount (SS=5%,SJS=3%, MJS=3%, C=1%). These data suggest little difference in adaptation to single and multi-joint strength training, and also indicate that muscle hypertrophy and strength or power improvements caused by training in these modes do not necessarily cause intrinsic strength improvements in muscle transferable to other movements using different modes of contraction. Further, sequenced strength-sprint training provided no additional power gain than sprint training alone.

INTRODUCTION

Force production in a complex movement requires the contraction of several muscles or muscle groups across multiple joints. The magnitude of force produced is a function of the mass of agonistic and synergistic muscles (Thorstensson et al. 1975; Komi, 1986), the extent to which these muscles can be activated by the nervous system (eg. increased number and frequency of motor units firing) (Moritani & DeVries, 1979; Sale et al. 1983; Hakkinen & Komi, 1986), the synchronization of motor unit firing (Milner-Brown et al. 1975) and the ability to co-ordinate the activation of all muscles involved both directly in the movement and as stabilizers of agonistic and synergistic muscles (Rutherford & Jones, 1986). Speed development is a function of the activities of myofibrillar adenosine triphosphatase (ATPase) (Thorstensson et al. 1975; Komi, 1986) creatine kinase, myokinase and enzymes associated with the phosphorylation of ATP through anaerobic glycolysis (Thorstensson et al. 1975; Cadefau et al. 1990). These enzymes are present in Type II muscle fibers to a greater extent than in Type I (Gollnick, 1972). The ability to quickly activate the motor units (Hakkinnen et al. 1985), and preferentially recruit fast-twitch motor units (Nardone et al. 1989) may also impact the rate of force development and velocity of muscle contraction. Nerve conduction velocity may also be a factor in quickly activating motor units but this is controversial and remains speculative (Kamen et al. 1984; Upton & Radford, 1975).

The product of strength (force) and speed (velocity) is power (Edgerton, 1986) and it follows that improvements in either or both of these factors should enhance maximal power. Both the strength and speed components of power are frequently trained separately, usually with strength training occurring before speed training. This is done in an attempt to improve the quantity and quality of the muscle mass with strength training prior to utilizing a speed overload in training the nervous system to activate the stronger muscles more quickly (Sale & MacDougall, 1981), however, improving strength may not necessarily improve power (Bell et al. 1989). Strength acquisition is highly specific to the movement pattern (Thorstensson & Karlson, 1974;

MacDougall et al. 1977;1979) and single rather than multiple-joint strength exercises are commonly prescribed. Since a large part of the strength training effect is neural (Moritani & DeVries, 1979;Sale et al. 1983; Rutherford & Jones, 1986), strength improvements through sport-specific multi-joint strength exercises could translate to greater power improvements than single-joint strength exercises. Further, it is also possible that if multi-joint speed training occurred subsequent to strength training the power gains would be greater than if speed training was done in isolation. Muscle hypertrophy also accounts for a large part of the strength training response (Goldspink, 1974; MacDougall et al. 1980) therefore single-joint strength training, through the isolation of specific muscle groups, could cause greater hypertrophy than multi-joint strength training, where load is distributed over a series of muscle groups. This greater muscular development with single versus multi-joint strength training could improve subsequent power development with sprint training. Since little research has investigated these questions the purposes of this study were:

1. To determine whether muscular and neural adaptations to single versus multi-joint strength training are different, and whether training induced adaptations in these modes are transferable to other strength and power movements.

2. To determine if there are differences in adaptations to strength versus sprint work.

3. To determine whether prior strength training (and consequent adaptations) enhances multi-joint power acquisition with subsequent sprint training over sprint training alone, and whether the mode of strength training (single vs multi-joint) influences this power acquisition.

Methods

Following University of Victoria Human Subjects Committee approval, 32 male volunteers, age 20-28, were oriented to the laboratory and testing and training procedures, prior to signing informed consent and agreeing to participate in the study. They were randomly assigned to one of 4 groups.

Training Procedures

The training program was 14 weeks in duration and split into 2 training phases (Figure 1). All subjects were asked to maintain their regular activity levels outside of the study.

Controls (C)

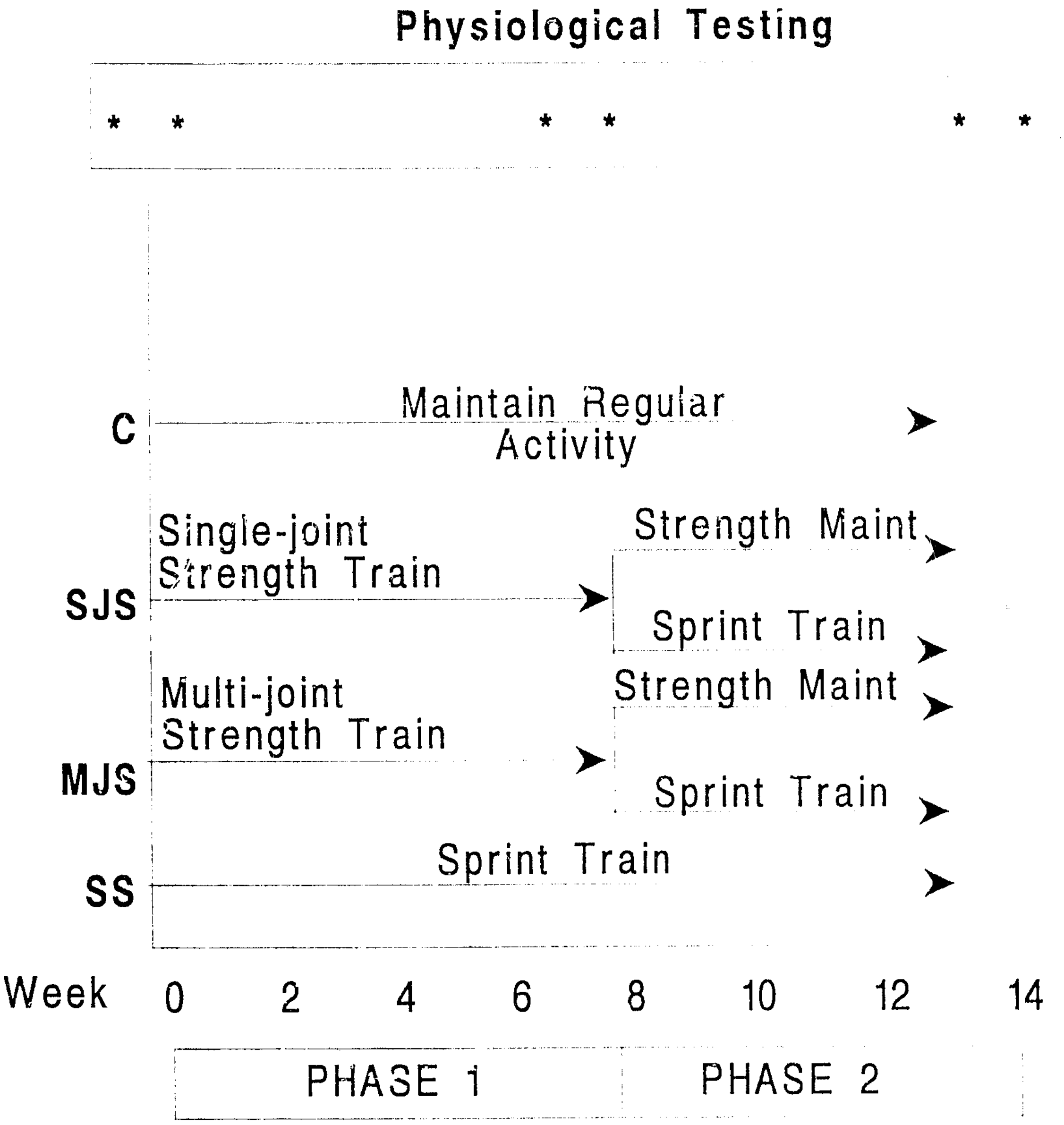
Controls were asked to maintain their regular level of activity without starting or stopping any present activities during the time frame of the study (14 weeks).

Sprint-Sprint Training Group (SS)

Training Phase 1 (8 weeks) & 2 (6 weeks)

Three times per week, separated by 48 hours, subjects performed 10s:120s work:rest intervals on a cycle ergometer with the load set at 65g/kg of body mass. The number of repetitions/workout started at 8 (2 sets of 4 with 5 minutes active recovery between sets) and increased by 2 repetition every two weeks, so that by week 14 subjects were completing 20 repetitions/workout (4 sets of 5 repetitions with 5 minutes active recovery between sets). Subjects were encouraged to sprint with 100% effort during the work intervals.

Figure 1: Experimental design of the 14 week training study, and training protocols for control (C), single-joint strength (SJS), multi-joint strength (MJS) and sprint (SS) groups.


Physiological Testing
* *
* *
* *
C
Maintain Regular Activity
SJS
Single-joint Strength Train
Strength Maint
Sprint Train
MJS
Multi-joint Strength Train
Strength Maint
Sprint Train
SS
Sprint Train
Week
0
2
4
6
8
10
12
14
PHASE 1
PHASE 2

Single-Joint Strength-Sprint (SJS) and Multi-joint Strength-Sprint (MJS) Groups

Training Phase 1 (8 weeks)

Three days per week, separated by 48 hours, subjects trained on variable resistance strength training equipment (Universal). The SJS group trained using the isolated single-joint movements of plantar flexion, leg extension and hip extension while the MJS group trained using a multi-joint leg press movement (proximal-distal sequencing of hip extension, leg extension, plantar flexion). In addition, each group trained with 6 general upper body strength exercises. For each exercise, 3 sets of 8-12 repetitions were performed to failure at a load of approximately 10 repetitions maximum (RM), with 1-2 minutes rest between sets. When 12 repetitions could be performed without failure on the second or third set the load was increased. In addition, at the first session of each week's training a 10 RM test was performed in order to encourage progressive increases in training weights.

Training Phase II (6 weeks)

Both SJS and MJS groups performed maintenance strength training 1 day per week (identical load, sets, repetitions and rest intervals to last week of strength training in Phase I). Additionallly, they sprint trained in a similar program to that followed by the SS group. That is, three times per week, separated by 48 hours, subjects performed 10s:120s work: rest intervals on a cycle ergometer with the load set at 65g/kg of body mass. The number of repetitions per workout started at 8 (2 sets of 4 with 5 minutes active recovery between sets) and increased by 2 repetition every two weeks, so that by week 14 subjects were completing 12 repetitions per workout (3 sets of 4 repetitions with 5 minutes active recovery between sets).

Experimental Procedures

Subjects were tested twice during the pre-testing phase, twice after training phase 1 (8 weeks), and twice at the conclusion of training phase 2 (14 weeks)

(Figure 1). For each test administered, the maximal score from either of the two testing days was retained for data analysis. Subjects were asked to refrain from vigorous exercise during the 24 hour period before testing sessions and not to eat in the 2 hours immediately preceding testing. During the first testing session, their body height, mass, sum of 8 skinfolds (Ross & Marfell-Jones,1991), and left thigh volume (Katch et al. 1974) were measured anthropometrically.

Nerve Conduction Velocity

Subsequent to the anthropometry, tibial nerve conduction velocity was measured. Subjects lay prone on a padded table with the lower limb supported so that there was 120 degrees flexion at the knee and 90 degrees flexion at the ankle (Vecchierini-Blineau & Guiheneuc, 1979). Skin temperature was measured at two sites using skin thermistors taped to the lateral retro-malleolar groove and medial popliteal fossa (Halar et al. 1983). The thermistors were interfaced with a telethermometer (Yellowsprings). Maximal tibial nerve conduction velocity was obtained on the left leg using the traditional double stimulation technique (Smorto & Basmajian, 1979) and corrected for temperature effects (Halar et al. 1983) using the mean of the two leg temperatures. Briefly, square pulses of 0.1 ms duration and sufficient intensity to evoke a supramaximal compound muscle action potential were applied to the tibial nerve 1 cm lateral to the mid-line of the popliteal fossa, and at the medial retro-malleolar groove. The motor response was detected by two surface electrodes on the abductor hallucis muscle (Chu-Andrew, 1986; Dorfman & Bosley, 1979). All electrode positions were marked on the skin with indellible ink so that electrode placement was consistent between testing days. The difference in the latency of the motor response between the proximal and distal stimulation sites, together with the distances between the two sites of stimulation, was used to calculate nerve conduction velocity (Chu-Andrews, 1986). An integrated neurostimulator/amplifier/storage oscilloscope (Cadwell 5200) was used for all nerve stimulation and latency response measurements. Nerve distance was measured using metal calipers. The closest two values of three trials were averaged and taken to represent nerve conduction velocity for each session (Kamen et al. 1984).

Strength Measurements

In order to track strength improvements in the weight room, 10 RM performance tests were administered weekly on the resistance training apparatus to the SJS group for leg extension, plantar flexion and hip extension, and to the MJS group for leg press. In addition controls performed 10 RM tests on the leg press, and leg extension machines at 0, 8 and 14 weeks.

Force exerted by the left leg was also measured on an isokinetic dynamometer (Cybex) interfaced with a microcomputer using ATCODAS signal processing software (DATAQ). Maximal voluntary contractions at 0, 1.05, 2.10, 3.14 and 4.19 rad$\cdot$s^{-1} were measured for leg press, leg extension and plantar flexion in order to construct a force velocity curve for each movement. For all Cybex tests, straps were used to immobilize the upper body and the command "ready-set-go" was used for each contraction. Subjects were instructed to perform each movement as fast and as hard as possible upon hearing "go" and for isometric contractions were required to hold maximal force for a period of 3s. Three repetitions were performed at each speed, with peak torque from the strongest repetition, taken to represent velocity specific strength. Half the subjects completed the testing in the order of leg press-plantar flexion-leg extension, while the other half tested in the opposite order.

Leg Press: Peak torque exerted in the leg press exceeds the maximal torque capacity of the Cybex, therefore it was modified with a gear and chain system as previously described by Vandervoort et al. (1984). Subjects were in a seated position and for isometric tests, strength was measured with the hip and knee at 100°. For concentric contractions subjects started with the knee at 90° (Vandervoort et al. 1984).

Leg extension: Leg extension was measured in the seated position with the hips at 90° and the knee set at 100° for isometric contractions.

Plantar Flexion: Subjects were secured to a Cybex UBXT in the supine position. All contraction velocities were performed with the knee set at 100° and for the isometric contractions the ankle was also set at this angle.

Rate of Torque Development

For each movement the rate of torque development (RTD) was calculated from the isometric force-time curve. Force data was sampled at 2000 Hz and the first derivative of each force curve, smoothed by a factor of 7 was taken to provide a measure of RTD expressed as $Nm \cdot s^{-1}$. The smoothing factor of 7 resulted in slopes being calculated from 7 points over a duration of 3.5 ms. Mean RTD between 30 and 60% of peak torque was calculated.

Electromyography (EMG)

The motor point areas of the vastus lateralis (VL), rectus femoris (RF) and the medial (MG) and lateral (LG) heads of the gastrocnemius muscles were determined using an electrical stimulator. After reducing the skin impedance with sandpaper and rubbing alcohol, bipolar silver/silver chloride surface electrodes (3M) were placed over the motor point along the longitudinal axis of the muscle, 20 mm apart rim to rim. Electrode position was marked on the skin with indelible ink and subjects were asked to maintain these marks between testing sessions to ensure the same electrode positioning for each session (Hakkinen et al. 1991).

For each movement, EMG was collected for the isometric contractions as well as the 1.05 $rad \cdot s^{-1}$ concentric contraction. The myoelectric signal was sampled at 2000 Hz, amplified, and low pass (20Hz, 3rd order response) and high pass (1.5KHz, 2nd order response) filtered. The signal was subsequently rectified, integrated and averaged with ATCODAS signal processing software (DATAQ) for each muscle during the maximal force phase of the isometric contraction (1 s) and over the duration of the 1.05 rad$\cdot$s^{-1} concentric contraction. For both isometric and isokinetic contractions the average IEMG values for each of the muscles monitored was summed and then averaged in order to provide a single EMG value for each movement (Hakkinen et al. 1992). Thus mean IEMG activity for each movement was calculated as follows:

Mean Leg extension IEMG = (VL-IEMG + RF-IEMG)/2
Mean Plantar flexion IEMG = (MG-IEMG + LG-IEMG)/2
Mean Leg Press IEMG = (VL-IEMG + RF-IEMG + MG-IEMG + LG-IEMG)/4

These average IEMG values represent quantitative measures of the amount of electrical activity produced by the muscle fibers of activated motor units during each maximal contraction (Sale,1991).

Multi-joint Power

Fifteen minutes after strength testing, subjects performed a 15s power test on a cycle ergometer (Monark) equipped with toe clips and racing bars. A photoelectric cell registered every 45 degrees of flywheel revolution and was interfaced with an electronic timing device in order to accurately count flywheel revolutions during the test. Subjects were given a two minute warm-up at 50 Watts after which time they were told to increase their revolutions to a maximum as fast as possible. Simultaneously, the flywheel resistance was set at 95 g/kg of body weight, rounded to the nearest quarter kp. Once the resistance was set a verbal signal was given and the 15s test began. Peak 1s power, and mean 5, 10 and 15s power values were calculated in Watts (W).

Muscle Biopsy

Five SJS, 5 MJS, 6 SS and 5 C subjects reported to the lab on a third testing day for the muscle biopsy procedure, pre, mid and post-training. A muscle sample was taken from the vastus lateralis of the left leg using the percutaneous needle-biopsy technique (Bergstrom, 1962) with the addition of suction to facilitate the acquisition of an adequate muscle sample (Evans et al. 1982). The biopsy procedure was administered by a physician experienced in this technique. The depth of the biopsy sample was standardized so that the error associated with generalizing a single biopsy to the entire muscle was minimized (Lexell et al. 1985; Elder et al. 1982).

Immediately following the biopsy, the muscle sample was mounted on a cork with the fibers oriented in a vertical position. The muscle samples were then rapidly frozen in isopentane cooled to -160 °C with liquid nitrogen and stored at -70 °C until histochemical analysis (Martin et al. 1988).

Histochemistry

The properties of single fibers in the biopsy sample were determined from tissue sections cut (10 um thick) using a cryostat maintained at a temperature of -20 °C. Fibers were histochemically stained for myofibrillar ATPase activity using an alkaline preincubation (pH 10.0) as described by Brooke & Kaiser (1970). Visual inspection of the staining intensity allowed subsequent classification of the muscle fibers as Type I (light) or Type II (dark). The fiber-type composition of the biopsy sample was then calculated and corresponding cross-sectional areas determined for each fiber type by video-scanning each biopsy preparation into a microcomputer using image analysis software (Optimas). The areas of all Type I and II fibers present in one field of view were calculated by tracing each fiber with a mouse that had been previously calibrated to a stage micrometer.

Statistics

For the strength, power and EMG variables a 4 x 3 ANOVA with repeated measures on one factor (time) followed by paired t-test planned comparisons was utilized to test for differences between groups and over time. A similar model was utilized for motor nerve conduction velocity, however height and age were used as covariates since these factors account for inter-individual differences in nerve conduction velocity (Chu-Andrews, 1986; Dorfman & Bosley, 1979). The histochemical data were compared between groups and across time using a 4 x 3 ANOVA with repeated measures on one factor (time) and planned comparisons utilizing paired t-tests. For all statistics, significance was set at $p < 0.05$.

Results

The physical characteristics of the subjects were not significantly different between groups, and did not change over time (Table 1).

Absolute 1s, 5s and 10s power outputs on the cycle ergometer significantly improved during training phase 1 for SJS and SS groups, and improvements approaching statistical significance were also noted in MJS (1s:p=0.08, 5s:p=0.09, 10s:p=0.07) (Table 2). Only SJS significantly increased mean 15 s absolute power, although similar trends and magnitudes of improvement were evident in the other training groups, and statistical significance was approached (MJS: p=0.12, SS: p=0.09). By the end of phase 2 all training groups had significantly elevated absolute power outputs above pre-test values, however for 5, 10 and 15s power outputs, only SJS and SS had further improved from the mid-test at the end of phase 1 (MJS 5s: p=0.11, 10s: p=0.07, 15s: p=0.07) (Table 2). Body mass and leg volume did not change over the course of the training program, therefore, when expressed relative to these measures, power followed the same patterns of change over time as absolute power outputs.

For both the SJS and MJS groups mean 10 RM strength significantly increased during training phase1 (Figure 2) so that by week 4 both SJS (21.7%) and MJS (22.2%) were stronger than pre-training, and by week 8, significantly stronger than week 4 (43.6 and 41.1% stronger than pre-test, respectively). There was no difference in the rate of 10 RM strength improvement between these groups. During strength maintenance (Phase 2), 10 RM strength did not increase further, but was maintained at week 8 levels for both MJS and SJS groups. 10 RM strength did not change over 14 weeks for control subjects (Figure 2). Changes in isometric and isokinetic force generation did not follow the same patterns as 10 RM strength. No changes were noted for any group over time in either leg extension (Table 3) or leg press (Table 5), however SJS significantly improved isometric plantar flexion strength (Table 4). The rate of isometric torque development (RTD) did not change in any group for either leg extension (Figure 3) or plantar flexion (Figure 4).

Both Type I and II fiber cross-sectional area increased in all training groups by the end of training phase 1, and did not increase further during the sprint training of phase 2 (Table 6). The extent of fiber hypertrophy was not different between fiber types, therefore no changes were evident in the Type II/I fiber area ratio for any group. Similarly, the percentage of Type II fibers present in the vastus lateralis biopsy sample did not change in any group over time (Table 6).

Mean IEMG values for isometric and 1.05 rad/s leg extension, plantar flexion and leg press did not change in any group over the course of the training program (Table 7). Nerve conduction velocity (NCV) improved in MJS by the mid-test at the end of phase1, and by the post-test at the end of phase 2 in both SJS and SS (Figure 5).

Table 1: Mean (SE) physical characteristics of the experimental and control groups pre, mid and post training: single-joint strength-sprint training (SJS); multi-joint strength-sprint training (MJS); sprint-sprint training (SS) and controls (C), n=8 each group.

Training Group	Time	Height (cm)	Mass (kg)	Sum8 (mm)	Leg Volume (cm^3)
SJS	Pre	180.2 (3.6)	79.1 (4.3)	107.6 (12.8)	5682 (425)
	Mid	-	79.7 (4.2)	102.6 (11.5)	5664 (444)
	Post	-	79.3 (4.2)	103.2 (11.9)	5631 (465)
MJS	Pre	176.6 (2.9)	79.2 (4.1)	97.1 (17.8)	5123 (292)
	Mid	-	79.4 (4.0)	94.6 (18.9)	5060 (340)
	Post	-	79.4 (3.9)	92.1 (18.9)	5132 (306)
SS	Pre	179.3 (1.6)	77.2 (3.1)	92.9 (14.1)	5309 (292)
	Mid	-	77.7 (2.7)	92.9 (13.8)	5285 (234)
	Post	-	78.2 (2.5)	92.8 (13.4)	5400 (288)
C	Pre	177.7 (1.9)	72.6 (1.7)	69.8 (7.8)	4996 (299)
	Mid	-	72.4 (1.7)	69.7 (7.8)	4945 (311)
	Post	-	72.2 (1.6)	69.9 (8.1)	5035 (323)

Table 2: Mean (SE) power outputs (W) from the 15s cycle ergometer sprint test for the experimental and control groups pre, mid and post training: single-joint strength-sprint training (SJS); multi-joint strength-sprint training (MJS); sprint-sprint training (SS) and controls (C), n=8 each group.

	1s			5s		
	Pre	Mid	Post	Pre	Mid	Post
SJS	987 (58)	1045* (57)	1060* (62)	936 (54)	974* (50)	993*† (53)
MJS	1031 (46)	1083 (51)	1106* (50)	976 (44)	1017 (47)	1045* (50)
SS	943 (40)	1055* (42)	1056* (38)	894 (36)	960* (35)	996*† (34)
C	996 (30)	968 (38)	969 (38)	957 (31)	920 (33)	923 (33)

	10s			15s		
	Pre	Mid	Post	Pre	Mid	Post
SJS	866 (58)	900* (54)	930*† (54)	806 (55)	839* (50)	877*† (51)
MJS	902 (43)	941 (44)	977* (50)	850 (44)	880 (42)	915* (48)
SS	850 (36)	892* (35)	941*† (35)	807 (34)	836 (28)	891*† (32)
C	924 (31)	863 (31)	868 (26)	867 (29)	812 (30)	819 (24)

Note: *significantly higher than pre-test ($p < 0.05$), † sig.higher than mid-test ($p < 0.05$).

Figure 2: The mean (SE) percentage change over 14 weeks in 10 repetition maximum strength for the two strength groups and control group pre, mid and post training. The single-joint strength-sprint group (SJS) results represent average of leg extension, plantar flexion, hip extension; multi-joint strength-sprint training group (MJS) results are for leg press; control (C) results represent average of leg extension and leg press, n=8 each group. Note: *significantly higher than pre-training (Week 0), + significantly higher than week 4.

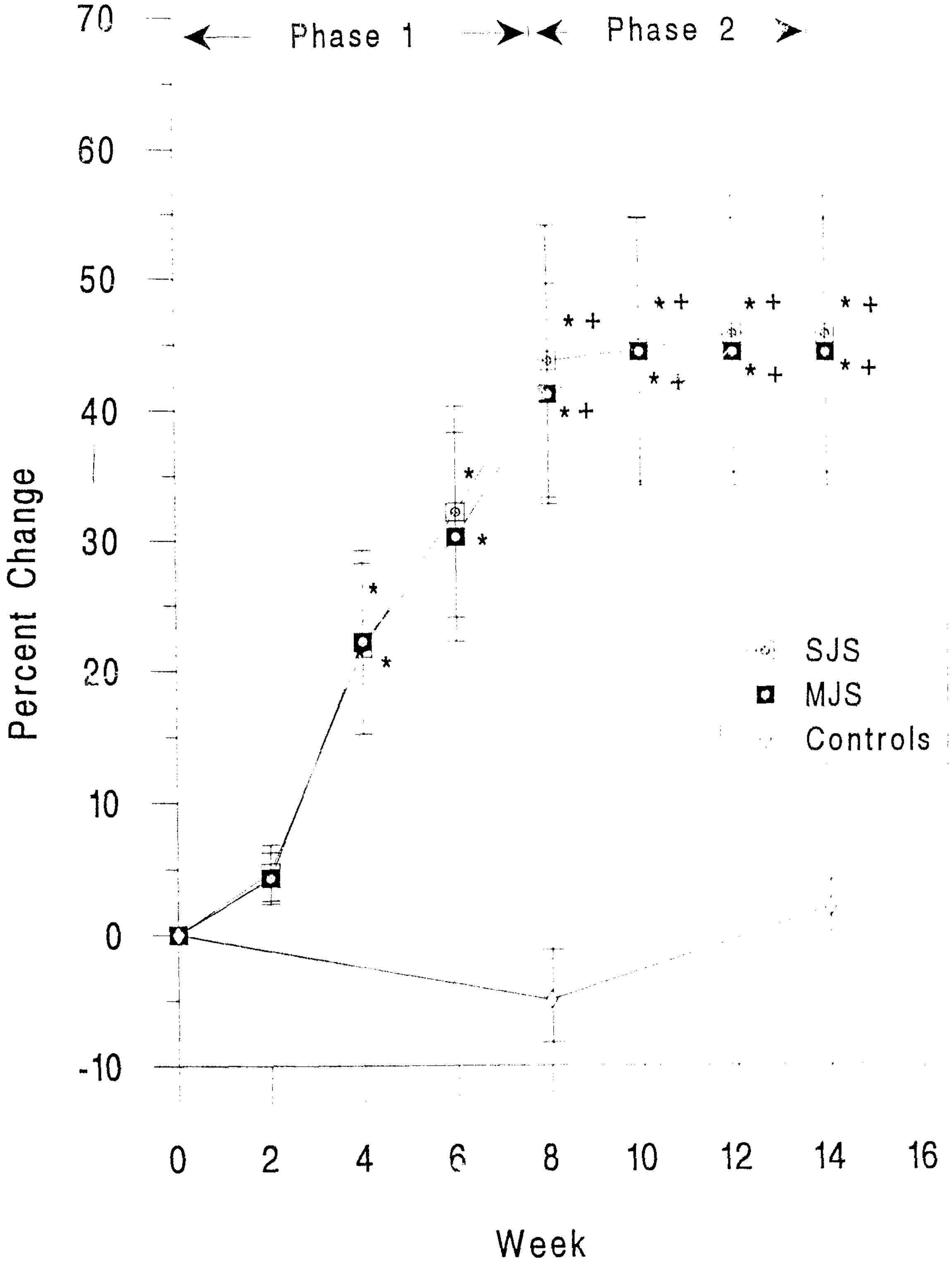

Phase 1
Phase 2
70
60
50
40
30
20
10
0
-10
Percent Change
0
2
4
6
8
10
12
14
16
Week
SJS
MJS
Controls
* +

Table 3: Mean (SE) peak torque values (Nm) for isometric and isokinetic Cybex leg extension in the experimental and control groups pre, mid and post training: single-joint strength-sprint training (SJS); multi-joint strength-sprint training (MJS); sprint-sprint training (SS) and controls (C), n=8 each group.

		SJS			MJS	
Rad/s	Pre	Mid	Post	Pre	Mid	Post
0	307 (24)	311 (28)	306 (25)	297 (18)	303 (19)	301 (20)
1.05	230 (17)	236 (19)	220 (18)	224 (12)	221 (15)	216 (12)
2.10	184 (13)	192 (15)	189 (14)	182 (10)	185 (11)	181 (12)
3.15	149 (12)	158 (13)	154 (11)	143 (7)	152 (9)	150 (10)
4.18	124 (10)	130 (11)	128 (10)	115 (6)	128 (7)	125 (9)

		SS			C	
Rad/s	Pre	Mid	Post	Pre	Mid	Post
0	300 (15)	294 (13)	295 (14)	306 (23)	302 (19)	303 (21)
1.05	227 (12)	221 (11)	223 (9)	217 (14)	218 (12)	213 (14)
2.10	179 (10)	177 (9)	176 (8)	181 (11)	183 (10)	181 (10)
3.15	145 (7)	145 (7)	147 (7)	151 (9)	149 (9)	150 (8)
4.18	118 (7)	123 (7)	122 (5)	123 (6)	121 (8)	125 (7)

Table 4: Mean (SE) peak torque values (Nm) for isometric and isokinetic Cybex plantar flexion in the experimental and control groups pre, mid and post training: single-joint strength-sprint training (SJS); multi-joint strength-sprint training (MJS); sprint-sprint training (SS) and controls (C), n=8 each group.

		SJS			MJS	
Rad/s	Pre	Mid	Post	Pre	Mid	Post
0	115 (9)	144 (16)*	133 (15)*	116 (8)	124 (9)	113 (8)
1.05	104 (5)	106 (7)	98 (7)	103 (6)	96 (6)	91 (4)
2.10	70 (4)	70 (5)	65 (5)	73 (5)	69 (4)	62 (3)
3.15	49 (3)	46 (4)	42 (4)	51 (5)	52 (4)	43 (2)
4.18	35 (2)	30 (3)	27 (2)	36 (4)	35 (3)	29 (3)

		SS			C	
Rad/s	Pre	Mid	Post	Pre	Mid	Post
0	112 (8)	123 (7)	123 (7)	108 (8)	101 (9)	118 (8)
1.05	91 (4)	99 (5)	93 (4)	90 (5)	89 (3)	89 (5)
2.10	62 (3)	66 (4)	61 (4)	60 (3)	58 (3)	58 (3)
3.15	43 (2)	44 (2)	40 (3)	38 (3)	43 (3)	41 (3)
4.18	31 (2)	29 (3)	26 (2)	27 (2)	31 (3)	30 (3)

Note: *significantly higher than pre-test ($p < 0.05$).

Table 5: Mean (SE) peak torque values (Nm) for isometric and isokinetic Cybex leg press in the experimental and control groups pre, mid and post training: single-joint strength-sprint training (SJS); multi-joint strength-sprint training (MJS); sprint-sprint training (SS) and controls (C), n=8 each group.

		SJS			MJS	
Rad/s	Pre	Mid	Post	**Pre**	**Mid**	**Post**
0	531 (50)	517 (37)	496 (42)	602 (25)	625 (28)	671 (55)
1.05	436 (23)	459 (32)	446 (31)	432 (27)	471 (27)	456 (33)
2.10	201 (10)	213 (11)	206 (8)	197 (21)	206 (18)	204 (12)
3.15	164 (9)	170 (14)	173 (11)	152 (19)	165 (17)	163 (12)
4.18	112 (4)	127 (12)	123 (12)	115 (14)	128 (16)	126 (14)

		SS			C	
Rad/s	Pre	Mid	Post	**Pre**	**Mid**	**Post**
0	487 (30)	477 (24)	488 (31)	599 (38)	614 (34)	602 (38)
1.05	408 (28)	469 (19)	448 (17)	469 (18)	406 (21)	408 (23)
2.10	197 (12)	219 (8)	213 (10)	242 (17)	184 (10)	200 (10)
3.15	156 (11)	169 (8)	166 (14)	186 (13)	149 (12)	152 (11)
4.18	122 (11)	132 (10)	136 (7)	145 (12)	111 (11)	108 (8)

Figure 3: Mean (SE) rate of isometric torque development(RTD) for Cybex leg extension in the experimental and control groups pre, mid and post training: single-joint strength-sprint training (SJS); multi-joint strength-sprint training (MJS); sprint-sprint training (SS) and controls (C), n=8 each group.

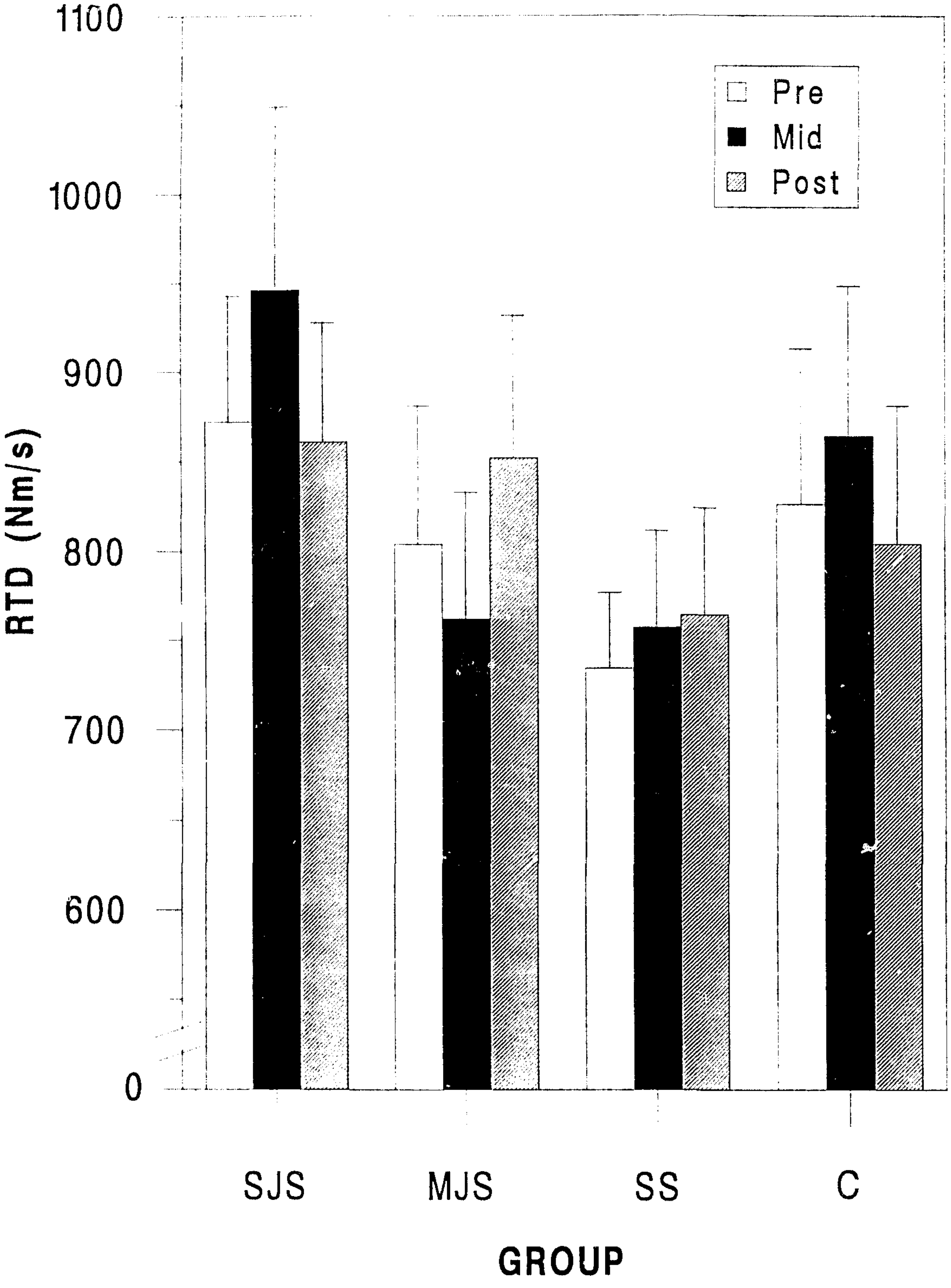

1100
1000
900
800
700
600
0
RTD (Nm/s)
Pre
Mid
Post
SJS
MJS
SS
C
GROUP

Figure 4: Mean (SE) rate of isometric torque development (RTD) for Cybex plantar flexion in the experimental and control groups pre, mid and post training: single-joint strength-sprint training (SJS); multi-joint strength-sprint training (MJS); sprint-sprint training (SS) and controls (C), n=8 each group.

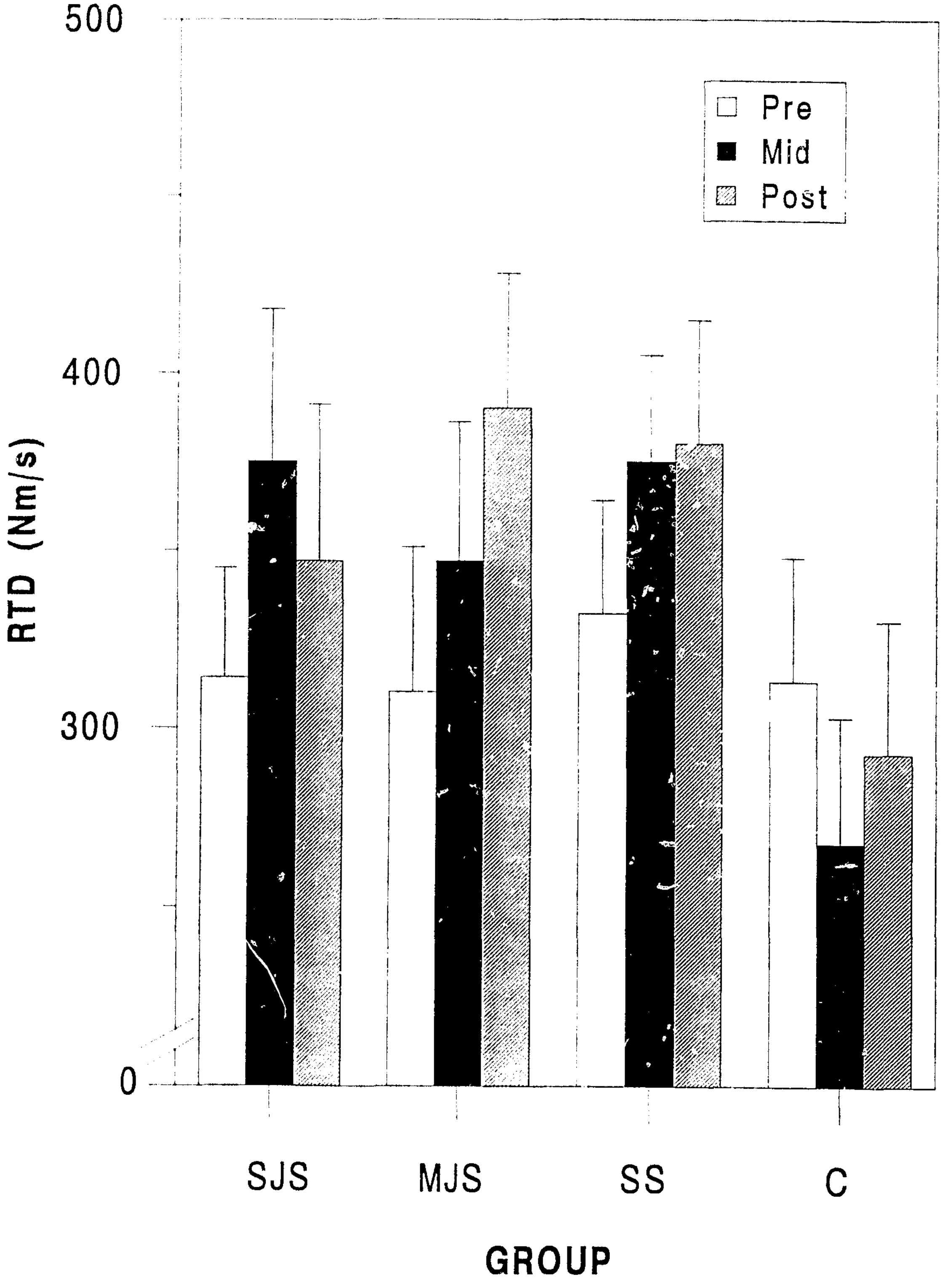

500
400
300
0
RTD (Nm/s)
☐ Pre
■ Mid
▨ Post
SJS
MJS
SS
C
GROUP

Table 6: Mean (SE) Type I and II muscle fiber cross-sectional areas, area ratios and distributions in the experimental and control groups pre, mid and post training: single-joint strength-sprint training (SJS,n=5); multi-joint strength-sprint training (MJS,n=5); sprint-sprint training (SS,n=6) and controls (C,n=5).

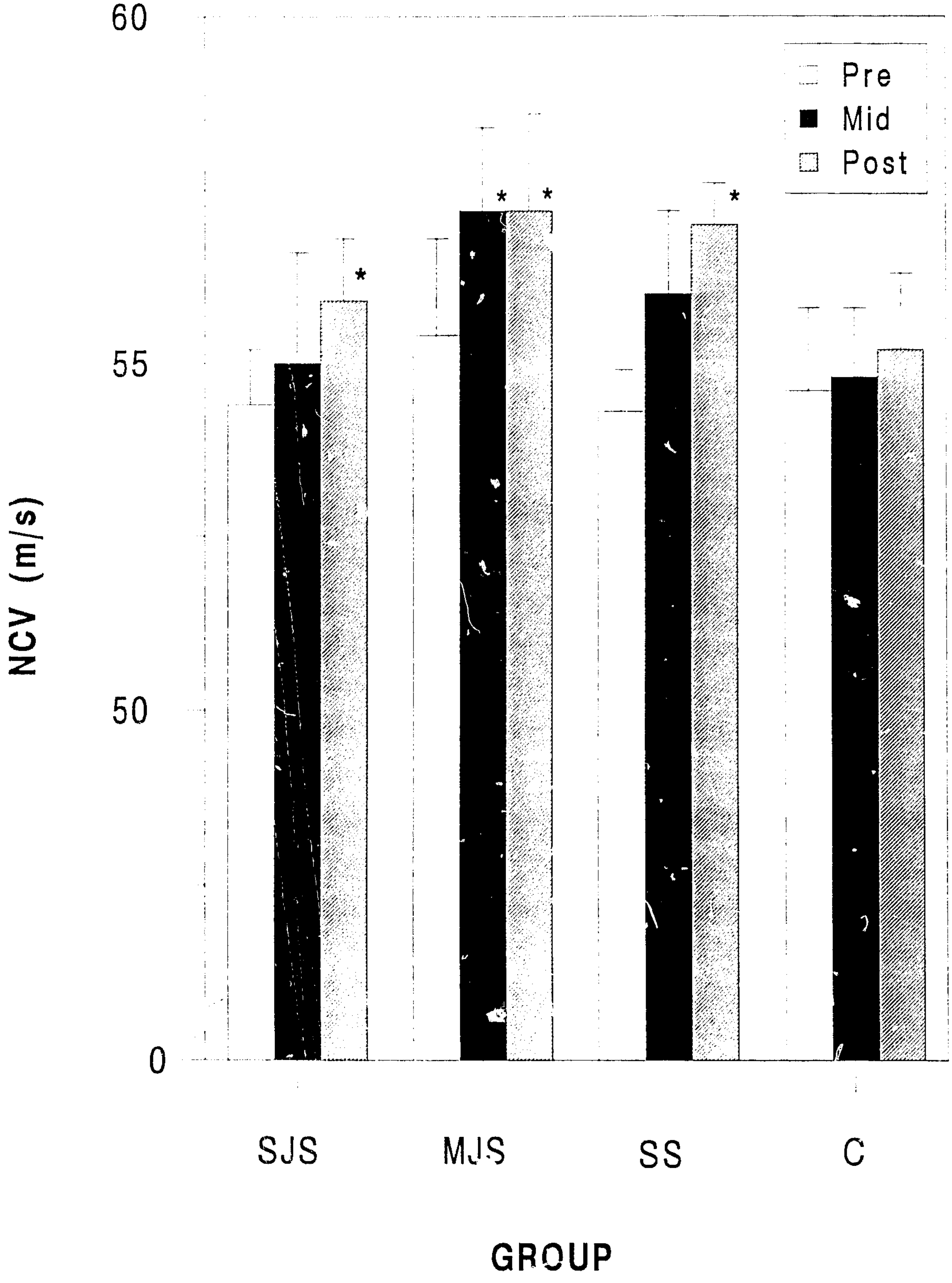
		Type I Fiber Cross-sectional Area x 10^2 (um^2)	
Group	**Pre**	**Mid**	**Post**
SJS	43.1 (4.5)	49.3 (5.1)*	50.5 (4.7)*
MJS	43.2 (1.0)	46.6 (1.7)*	48.3 (1.3)*
SS	47.9 (3.2)	55.1 (2.7)*	56.9 (2.1)*
C	49.5 (3.9)	-	50.7 (4.9)
		Type II Fiber Cross-sectional Area x 10^2 (um^2)	
Group	**Pre**	**Mid**	**Post**
SJS	45.8 (5.2)	51.6 (4.8)*	51.0 (3.7)*
MJS	49.9 (5.6)	57.7 (5.0)*	55.9 (5.8)*
SS	55.1 (3.9)	60.4 (2.8)*	59.9 (3.6)*
C	54.2 (7.1)	-	47.2 (5.5)
		Type II/I Fiber Area Ratio	
Group	**Pre**	**Mid**	**Post**
SJS	1.07 (0.05)	1.08 (0.05)	1.06 (0.06)
MJS	1.16 (0.15)	1.24 (0.12)	1.15 (0.11)
SS	1.16 (0.05)	1.10 (0.03)	1.12 (0.07)
C	1.06 (0.12)	-	0.99 (0.08)
		Type II Fiber Percentage	
Group	**Pre**	**Mid**	**Post**
SJS	53.6 (2.4)	53.9 (2.0)	56.0 (2.9)
MJS	46.2 (4.5)	51.1 (6.2)	52.2 (6.1)
SS	46.4 (3.7)	44.8 (3.1)	52.6 (5.3)
C	52.0 (4.5)	-	51.4 (4.5)

Note: *significantly higher than pre-test ($p<0.05$).

Table 7: Mean (SE) IEMG values (μv) for isometric and isokinetic Cybex plantar flexion in the experimental and control groups pre, mid and post training: single-joint strength-sprint training (SJS); multi-joint strength-sprint training (MJS); sprint-sprint training (SS) and controls (C), n=8 each group.

Leg Extension	0 rad/s			1.05 rad/s		
	Pre	Mid	Post	Pre	Mid	Post
SJS	648 (50)	687 (78)	671 (66)	561 (44)	582 (57)	565 (45)
MJS	659 (117)	646 (71)	707 (66)	635 (101)	501 (61)	490 (55)
SS	653 (99)	611 (62)	650 (69)	484 (38)	464 (34)	471 (39)
C	726 (80)	827 (84)	721 (80)	644 (68)	616 (57)	600 (57)
Plantar Flexion	**0 rad/s**			**1.05 rad/s**		
	Pre	Mid	Post	Pre	Mid	Post
SJS	348 (47)	280 (25)	341 (49)	338 (41)	335 (43)	381 (54)
MJS	422 (44)	372 (39)	380 (35)	430 (51)	373 (71)	391 (59)
SS	343 (28)	322 (29)	361 (36)	381 (30)	316 (33)	378 (33)
C	403 (44)	375 (38)	405 (37)	428 (50)	402 (39)	365 (23)
Leg Press	**0 rad/s**			**1.05 rad/s**		
	Pre	Mid	Post	Pre	Mid	Post
SJS	343 (24)	328 (20)	300 (20)	331 (23)	339 (32)	318 (22)
MJS	362 (36)	326 (35)	344 (55)	304 (33)	307 (30)	299 (31)
SS	388 (46)	339 (44)	308 (31)	312 (25)	326 (26)	314 (27)
C	404 (48)	383 (32)	309 (29)	382 (32)	366 (19)	297 (31)

Figure 5: Mean (SE) tibial motor nerve conduction velocity (NCV) in the experimental and control groups pre, mid and post training: single-joint strength-sprint training (SJS); multi-joint strength-sprint training (MJS); sprint-sprint training (SS) and controls (C), n=8 each group. Note:* represents significantly different from pre-test.


60
55
50
0
NCV (m/s)
Pre
Mid
Post
*
*
*
*
SJS
MJS
SS
C
GROUP

Discussion

Major findings of this study were that multi-joint power on the cycle ergometer increased both with strength and speed training, the mode of strength training seemed to have little impact on power acquisition, and training strength prior to speed did not increase multi-joint power more than speed training alone. Secondly, the primary mechanism of adaptation in all modes of training appeared to be muscular hypertrophy, although some small neural adaptations (increased NCV) were noted. A secondary finding of this study concerns neural specificity. It appears that the training stimuli is specific to mode of contraction, since improvements in strength measured on variable resistance training equipment, did not transfer to isokinetic force production as measured on the Cybex, yet transferred to cycle ergometer power.

It was hypothesized that a single-joint movement (ie. leg extension) may isolate prime movers more effectively, cause greater muscle hypertrophy, and therefore result in greater strength improvement than a more integrated multi-joint movement, utilizing several muscle groups working across several joints (ie. leg press). However, both SJS and MJS increased type I and II fiber area and 10 RM strength equally over the 8 weeks of phase 1 strength training, with no concurrent increases in the amount of motor unit activation (IEMG). It therefore seems that the rate of strength gain does not depend on the exercise movement per se, and adaptation is similar when factors such as loading intensity (RM), volume of training, and rest intervals are consistent.

The muscle fiber hypertrophy observed in response to strength training is consistent with previous research (Thorstensson, 1976; Houston et al. 1983; Hather et al. 1991). These investigators also observed greater hypertrophy in the Type II versus Type I muscle fibers, yet this was not observed in the present study, or in others (MacDougall et al. 1979; Frontera et al. 1988). The greater hypertrophic response of Type II muscle fibers has been suggested to reflect greater activation levels of the high threshold Type II motor units in strength and power activities relative to daily activity (MacDougall, 1991). In the present study, the volunteers were untrained, but were primarily physical education students. They must be considered active, and may recruit Type II motor units more regularly than the average inactive subject. Therefore, the lack of preferential Type II hypertrophy may reflect these activity levels. That leg volume did not change, in light of muscle fiber cross-sectional area increases, supports a suggestion that one of the first responses to a strength training stimulus is consolidation of the tissue as the muscle fibers increase in girth at the expense of extracellular spaces (Goldspink, 1991). Others have also reported much larger changes in muscle fiber area with small changes observed in whole muscle cross-sectional area (Frontera et al. 1988). This could act to increase the amount of tension generated per unit of muscle cross-sectional area (specific tension), but whether or not increased packing density of muscle fibers occurs in response to training remains speculative since methodological limitations exist in measuring fiber area (Blomstrand et al. 1984), and in generalizing the size of the fibers in a biopsy of the vastus lateralis to other fibers in that muscle, or in the rest of the quadriceps (Elder et al. 1982; Lexell et al. 1985; Frontera et al. 1988).

In the present study isometric and isokinetic strength measured on the Cybex showed very little improvement, despite the large 10 RM increases shown in both strength training groups when tested on the training apparatus (43%), and despite muscle fiber hypertrophy (13%). The only significant increase on the Cybex was observed in the SJS group for isometric plantar flexion (25%). The large discrepancy between the percentage increase in strength and muscle fiber cross-sectional area suggests that adaptive changes in the nervous system (Komi, 1986; Sale, 1988) or qualitative changes within the muscle to increase force production per unit cross-sectional area (Jones & Rutherford, 1987) have taken place. Recently, Sale et al. (1992) also observed muscle hypertrophy (11%) and large increases in 1RM weight lifting performance (29%) without improvements in isometric knee extension strength. Similarly, Frontera et al. (1988) increased knee extensor cross-sectional area by 12%, but found the gain in weight lifting performance (1RM) to be approximately 10 times greater than strength gains measured isokinetically. If muscle recruitment patterns on the training apparatus were sufficiently different than the patterns required for force generation on the Cybex, minimal transfer might be expected due to a neural specificity effect related to motor learning. In support of this, Tax et al. (1990) examined motor unit recruitment patterns of elbow flexor muscles in response to isometric, voluntary slow velocity and imposed slow velocity muscle contractions. They observed altered recruitment thresholds, motor unit firing frequencies, and corresponding reversal of motor unit recruitment order between isometric and slow voluntary isotonic movement. However, when movement was imposed on the elbow flexors no differences in motor unit behaviour were evident. This demonstrated that there are marked differences in neural recruitment patterns between different types of contractions, and these differences have their origin in the central activation of the motoneuron pool. Others have also demonstrated that learning specific activation patterns, and learning to co-ordinate the fixators, stabilizers and prime movers accounts for a large part of the improvement in the ability to lift weights (Rutherford & Jones, 1986; Canon & Cafarelli, 1987). Therefore, muscle hypertrophy, although a primary strength adaptation, does not necessarily cause intrinsic strength improvements in muscle transferable to movements other than those that have been specifically trained.

That no increase in IEMG, and therefore motor unit activation, was observed is not surprising since the EMG response was measured on the Cybex, where strength did not improve. This is similar to the findings of Thorstennson et al. (1976), who trained subjects for 8 weeks using the squat exercise. They observed a large increase in 1 RM squat performance (73%) but smaller improvements in isometric leg press (31.5%) and non-significant improvements in isometric knee extension (4%). No corresponding IEMG changes were observed. It may be argued that, if EMG was tested in the specific training mode, improvements would have been detected, yet Garfinkel & Cafarelli (1992) isometrically trained the knee extensors of young sedentary women and observed no increases in maximum EMG response when measured in the identical exercise. Further, although these investigators observed a discrepancy between the amount of improvement in the isometric maximum voluntary contraction (28%) and knee extensor cross-sectional area (14.6%), the ratio between the two (specific tension) was not significantly different after training. It was therefore concluded that the increased strength was due solely to a hypertrophic muscle response, but the movement studied was a very simple isolated movement, and not generalizable. Similarly, Duchateau & Hainaut (1984) trained subjects adductor pollicis muscle for 3 months, either isometrically or dynamically, and noted no EMG changes. They concluded that peak torque generation and the rate of tension development were related to intrinsic changes within the muscle. Others have also reported that most individuals can fully activate their available muscle mass in simple single-joint movements if given sufficient practice (Belanger & McComas, 1981; Canon & Cafarelli; 1987). Full motor unit activation may be more difficult in complex, multi-joint movements such as the leg press (Sale, 1988), however no increases in IEMG were noted in MJS. It has also been suggested that various force inhibitory mechanisms (Caiozzo et al. 1981) such as agonist co-contraction (Tyler & Hutton, 1986) or golgi tendon organ involvement may interfere with maximal motor unit activation.

Several research groups have reported increased motor unit activation to be an early strength training response, and a primary contributor to early strength gains (Moritani & DeVries, 1979; Hakkinen & Komi, 1986; Hakkinen et al. 1991). However there are inconsistencies in some of these studies. For example, Moritani & DeVries (1979) reported maximal EMG in untrained contralateral muscles to increase twice as much as in the trained muscle. Other reasons for the discrepancies between those studies supporting increased motor unit activation (Moritani & DeVries; 1979; Hakkinen & Komi, 1986; Hakkinen et al. 1991), and those that do not (Thorstennson et al. 1976; Canon & Cafarelli, 1987; Garfinkel & Cafarelli, 1992) are not clear, and at present are controversial. They likely are the result of differences in techniques used for assessing motor unit activation (EMG vs interpolated twitch technique or tetanic stimulation technique) and differences in the movements (simple vs complex) and populations studied (trained vs untrained). The data of the present study suggest a neural training effect does occur in the form of learning and co-ordination, but not in the form of increasing motor unit activation, since it appears from the EMG data that individuals are able to sufficiently activate the motor units of prime movers involved in maximum voluntary contractions. Further, no differences were observed between SJS and MJS, suggesting that sufficient motor unit activation occured in both simple and complex movements.

Both SJS and MJS (NS) increased maximal power outputs on the cycle ergometer during phase 1 strength training, but not differentially. That a transfer of training effect occurred here, but not to isometric and isokinetic strength, probably represents a greater amount of neural specificity between the isotonic concentric/eccentric training in the weight room and the dynamic contractions required to overcome resistance on the cycle ergometer. This contraction type specificity may explain why Bell et al. (1989) failed to improve rowing ergometer maximal power output after 5 weeks of isokinetic strength training. Conversely, Petersen et al. (1984) did observe increases in maximal power measured on the cycle ergometer after 5 weeks of isokinetic high velocity resistance training (6%). The discrepancies between these two studies may represent test protocol differences, since Bell et al. 1989 used a different ergometer and a constant load of 29.4 N (approximately equal to 40 g / kg body mass), while in the study by Petersen et al. (1989) a load of 65g/kg body mass was used, which is closer to the relative load required to optimize maximal power output (Smith, 1987).

It was also expected that MJS would increase cycle ergometer power to a greater extent than SJS due to activation patterns in strength training more specific to cycling (proximal-distal sequencing of hip extension, leg extension, plantar flexion), since task specific motor unit recruitment (Desmedt & Godaux, 1981; Hoffer et al. 1987; Tax et al. 1990) and learning (Rutherford & Jones, 1986) play a large role in strength adaptation. This did not occur however, which agrees with the data of Chapter 2 showing correlations of equal magnitude between single-joint strength, multi-joint strength and maximal power output on the cycle ergometer. It appears then that in transferring strength to high power output multi-joint movements, other factors are more important determinants of performance than the specificity of movement pattern used in strength training. This is supported by the finding that the sprint training group (SS), who trained in a more neurally specific fashion (on the identical cycle ergometer used in testing maximal power, but at a lower load setting than power testing) improved maximal power, but not to a greater extent than the strength groups. The adaptations that occurred in SS were very similar to those observed in the two strength groups and may provide some insight into the factors responsible for increasing power output similarly in all training groups.

The muscle hypertrophy observed in both strength groups during phase 1 of training was expected, however marked hypertrophy was also noted in (SS) for both Type I (15%) and II fibers (10%). This response to sprint training is not unprecedented since Thorstensson et al. (1975) reported increases in leg volume and small increases in the size of both slow and fast twitch muscle fibers after 8 weeks of sprint training. He also reported glycogen depletion in both types of fibers suggesting that they are both recruited in sprint exercise. Cadefau et al. (1990) also observed Type I and II fiber hypertrophy (13% and 14.7% respectively) following 8 months of sprint training in young athletes. Larger muscle fibers and muscle cross-sectional areas have also commonly been reported in sprint athletes when compared to other populations (Costill et al. 1976; Gregor et al. 1981; Johansson et al. 1987; Hakkinen & Keskinen. 1989), although this could represent natural selection as discussed in Chapter 2 of this thesis. Therefore, it appears that muscle hypertrophy is a consistent adaptation in both sprint and strength exercise. Further it is probably partially responsible for increasing maximal power in these groups, since with larger and stronger muscles the resistance set on the cycle ergometer could be more easily overcome.

Even with muscle fiber hypertrophy in the vastus lateralis, leg volume and body mass did not significantly change in any training groups. When combined with the absolute cycle ergometer power increases, this suggests that more power could be produced per unit of muscle mass after either sprint or strength training. This is contrary to what was observed when comparing power and endurance athletes in Chapter 2. However, as previously mentioned one of the earliest responses to strength training is thought to be consolidation of the muscle fibers at the expense of extracellular space (Goldspink, 1991). If training was continued over longer durations it is likely that more contractile protein would accumulate than could be accounted for by increased muscle fiber packing density, and this could make the increased power relative to body mass a transient adaptation. However, qualitative changes in the muscle, or neural changes, could also account for the increased power generated relative to muscle mass.

Although not measured in this study, it is possible that metabolic changes within the muscle, apart from fiber type transformation, contributed to improving relative power. With respect to strength training, the literature is not clear since increases (Eriksson et al. 1981), decreases (Tesch et al. 1987), or little change (Thorstensson et al. 1976; Houston et al. 1983; Tesch et al. 1990) in enzyme activities have been reported, and increases (MacDougall et al. 1977) or no change (Tesch et al. 1990) have been observed in muscle concentrations of ATP, ADP, creatine, creatine phosphate and glycogen. The response to sprint training is mor consistent, with reported enzymatic increases (Thorstennson et al. 1975; Cadefau et al. 1990) but little change in substrate storage (Thorstennson et al. 1975).

Muscle fiber type changes have been extensively studied, and it has been reported that both large Type II fibers (Chapter 2) and a high percentage of Type II fibers (Bar -Or et al. 1980; Froese & Houston, 1987) are positively related to maximal power generation. Others have agreed with the findings of Chapter 2, and reported little relationship between Type II fiber composition and maximal power output (McCartney et al. 1983; Patton et al. 1990). Consistent with these observations, no changes in Type II fiber percentage were noted during phase 1 of this study in any group. Much of the literature has reported no fiber type changes with strength training (Costill et al. 1979; Houston et al. 1983; Hakkinen et al. 1988), or sprint training (Thorstensson et al. 1975) while others have reported mixed findings. For example, Cadefau (1990) reported an increased Type I percentage after 8 months of sprint type training in young athletes, an adaptation which does not seem consistent with the type of training these subjects were performing. Green et al.

(1979) reported decreases in Type IIb percentages with corresponding increases in IIa fiber percentage over a season of play in elite hockey players and similarly Hather et al. (1991) reported decreasing IIb and increasing IIa fiber percentages in response to 19 weeks of resistance training. Jacobs et al. (1987) reported an 8% decrease in the percentage of Type I fibers (NS) after 6 weeks of sprint training, with a concomitant increase in the proportion of IIa fibers. He also stated however that this could be due to variability between biopsy samples, which is one factor that may account for the conflicting research reports (Elder et al. 1982; Lexell et al. 1985). It may be possible for transformations to occur between IIb and IIa fibers, however, the present data support the majority of the literature in rejecting Type I to II fiber transformation in both strength and sprint work. Therefore, fiber type changes cannot be held responsible for the increased maximal powers relative to body mass and leg volume observed over phase 1 in both the strength and sprint groups.

It is difficult to estimate the magnitude of neural changes, and their impact on multi-joint power production in the three training groups over phase 1. That no EMG changes were observed in SS is not surprising since EMG was tested in a different mode of exercise from cycle ergometry, and as previously discussed, most individuals can fully activate their muscles. Learning plays a significant role in improving force generation, as observed in the strength groups, and there is no reason to expect that this is not also the case in multi-joint power production. Since SS showed hypertrophy, but no increases in Cybex strength, it would seem that this is the case.

Conduction velocity of the tibial nerve (NCV) was observed to increase by the end of phase 1 in MJS. A trend of improvement was also noted in SS (p=0.09) but no significant change occurred in SJS (p=0.36), due to high group variability at the mid-test. Others have also observed improvements in NCV with resistance training subsequent to immobilization in humans (Sale et al. 1982) and in spinalized cats exercised for 13-14 weeks (Edgerton et al. 1980). Indirectly, nerve fiber hypertrophy has been elicited as a result of exercise training (Eisen et al. 1973; Walsh et al. 1978) and this should result in faster NCV (Waxman, 1980). Changes in myelination, which could directly impact NCV, have also been observed with exercise (Roy et al. 1983).

Motoneuron enzymatic changes observed in response to exercise (Suzuki et al. 1991; Chalmers et al. 1991) and their impact on NCV is unknown, however if they influenced membrane potentials they could potentially increase NCV. Thus the literature suggests there are several adaptations in response to exercise in a motoneuron that could increase NCV. The training improvements noted in NCV do not concur with the data of Chapter 2 which showed no differences between power, endurance and non-athletes, and no relationship between NCV and power output. From the present data and the literature it seems that NCV responds to exercise, but not specifically to one form over another (sprint versus strength), and the magnitude of change is small, perhaps physiologically insignificant. This small effect size makes it likely that studies will continue to report conflicting results. Nevertheless, it is concluded that the faster NCV observed in the present study subsequent to strength or sprint training could provide a small advantage in increasing the rate at which muscles can be activated and therefore play a role in improving maximal power.

Since NCV increased in all training groups, one might expect that the rate of isometric torque development (RTD) would also increase due to faster muscle activation, however this was not observed for either leg extension or plantar flexion in any group. It is possible that the neuromuscular junction is a rate limiting step in this respect, and that increases in NCV are not translated into faster muscle activation and torque development due to slow transmission across the synaptic cleft. NCV was not related to RTD in the cross-sectional study (Chapter 2) however, so its influence on this parameter is questionable, and other neural factors such as increased motoneuron firing frequencies (Grimby, et al. 1981), increased onset of EMG (Hakkinen et al. 1985), preferential recruitment of Type II motor units (Grimby & Hannerz, 1977), or pre-movement silence (Mortimer et al. 1987) might be more important. Contrary to this study, others have shown increased rates of force development after strength training. Duchateau & Hainaut (1984), using supramaximal electrical stimulation showed that increases in the rate of force development after 3 months of training the adductor pollicis muscle were not a result of neural adaptation, but due to intrinsic changes within the muscle. They speculated that these changes may have been related to increased myofibrillar ATPase activity, or changes in Ca^{2+} movement via adaptation of the sarcoplasmic reticulum. Changes in both Ca^{2+} sensitivity and myofibrillar ATPase activity have been demonstrated with resistance (Bell et al. 1992) and power training (Thorstensson et al. 1975;Belcastro et al. 1981). Hakkinen et al. (1992) showed positive changes in the force-time curve after resistance training women for 3 weeks in a manner similar to that performed in this study. The main changes in the force-time curve occurred at higher force levels, which is similar to data reported by Thorstensson et al. (1976) who strength trained individuals for 8 weeks and noted faster rates of force development above 75% of pre-test peak torque. Others, using explosive types of training procedures have noted improvements earlier in the force-time curve (Hakkinen & Komi, 1985; Duchateau & Hainaut, 1984). In the present study RTD was measured between 30 and 60% of peak torque which may account for the lack of significant change since explosive strength training was not performed. That RTD differences were noted between power (explosive volleyball players) and endurance athletes in chapter 2, reinforces this specificity of training effect suggested in the literature. Thus, with explosive training, one can expect improvements in the early part of the force-time curve, but with more standard resistance training (eg. 10 RM loads), adaptations probably occur later in this curve. No studies have reported how sprint-training may influence RTD.

The influence of sequenced strength-sprint training on subsequent power acquisition has not been previously studied but is commonly utilized in elite sport in the form of periodized training models (Sale & MacDougall, 1981). The theory behind sequencing training in this fashion is as follows: the initial strength training phase is designed to cause muscle hypertrophy in those muscles utilized during the sports movement for which they are being trained (muscular adaptation), but not necessarily improve the ability of the athlete to utilize those muscles. The subsequent sprint phase is then introduced to teach the individual to activate the newly acquired muscle mass (neural adaptation), in a more sport-specific manner. One purpose of this study was to determine the effectiveness of this sequenced training approach. The data indicated that sequenced strength-sprint training was no more effective than sprint training alone in raising multi-joint power, since by the end of the 2nd training phase (6 weeks of sprint training) power had increased significantly, but not differentially in both the strength groups (SJS, MJS) that had previously strength trained for 8 weeks, and in the sprint group (SS) that had sprint trained for 14 straight weeks. As previously discussed, all groups showed both type I and II muscle fiber hypertrophy in the first 8 weeks of training, and increased NCV by the end of the 14 week training period. Thus, it appears that these two adaptations, or other changes similar in both sprint and strength training, contributed to no sequence effect being observed. An advantage of the sequential strength-speed training model is that for those athletes requiring strength in addition to power, this model will allow them to increase both, without compromising power development. Further, the sequenced training groups were at maximum performing 12 sprint repeats in a given workout (week 14), while SS performed 20 repeats by week 14, thus it would appear that the sequenced training groups received more return on their training time investment.

An additional variable that was introduced into this sequenced training model was the mode of strength training. It was hypothesized that single-joint strength training would cause primarily muscular adaptations relevant to power generation, but if a multi-joint movement was utilized to train strength, both neural and muscular adaptations could be expected to transfer to power movements, and hence more improvement should be observed. As previously discussed single versus multi-joint strength training had no differential impact on power acquisition prior to the sprint training period indicating that even though the muscle activation patterns were similar between leg press and cycling (proximal to distal sequencing of hip extension, leg extension, plantar flexion), neural differences probably still existed. In chapter 2 it was hypothesized that single-joint strength training might elicit a greater hypertrophic response than multi-joint strength training due to greater muscle isolation, and therefore with subsequent speed training elicit greater power improvements. This was not supported since both modes of training caused similar amounts of muscle fiber hypertrophy and when followed by sprint training similar power improvements were observed. Comparable increases in NCV were also evident by the end of the 14 week training period and any neural or learning differences between the two strength modes were not transferred to the cycle ergometer, thus the mode of strength training (single or multi-joint) has no influence on adaptation to sequenced strength-sprint training and it is concluded that their is no faster power improvement when sequenced (single or multi-joint) strength-speed training is utilized as opposed to straight sprint training over similar time-frames. This is because the neural and muscular adaptations to the two training modes were similar in nature and magnitude.

In summary, the present data have suggested that strength training, sprint training or sequenced strength-sprint training can increase maximal power and these increases in power are due to both muscular and neural changes. However, muscle hypertrophy and strength or power improvements caused by training in these modes does not necessarily cause intrinsic strength improvements in muscle transferable to other movements using different modes of contraction (ie. isokinetic strength on the Cybex). It appears however that strength may be transferred more readily to activities using similar contraction types since strength trained in both single and multi-joint movements utilizing concentric and eccentric isotonic exercises transferred very well to power output during constant load cycle ergometry.

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Appendix

Review of literature

The rapid application of force through muscular contraction across multiple joints to provide acceleration, deceleration, maintain velocity or change direction of the body mass is critical in many sports. Depending upon the sport, this work may be required instantaneously, continuously for several seconds, or intermittently for several minutes. The amount of physical work completed in a given unit of time is termed power (Knuttgen, 1978).

Theoretically, any improvement in the ability to generate force or in the rate of force development should improve power output capability (Edgerton, 1986). Consequently, the periodized training plans of many sports, such as alpine skiing and ice hockey, sequence a general strength training phase, to improve force development in muscle, prior to a speed or power training phase designed to improve the rate of force development. Strength training has been well investigated and it is known that strength improvement occurs due to both muscular and neural adaptations (Moritani & DeVries, 1979; Sale & MacDougall, 1981; Komi, 1986). Less is known about the influence of strength on power.

In untrained muscle, strength gains in the first six weeks of a training program have been attributed primarily to increased maximal activation of muscle due to changes in neural factors which may include an increased number and frequency of motor neurons firing (Moritani & DeVries, 1979) and increased motor unit synchronization (Milner-Brown et al. 1975). Possible explanations for these changes include increased sensory input to the dendrites of alpha-motoneurones and increased descending activity from higher motor centres (Komi, 1986).

Both electrophysiological and metabolic properties of motoneurones are consistent with the properties of the type of muscle fiber they innervate suggesting a degree of plasticity in the motoneurone itself. Slow motoneurones have lower maximal firing rates and axonal conduction velocities but greater excitability and fatigue resistance than fast motoneurones (Gardiner,1991). They also vary in their levels of energetic enzymes in a manner similar to the muscle fibers they innervate (Ishihara, 1988). These differences in the properties of motoneurones from different motor unit types may indicate adaptation that is specific to chronic activity levels.

There is some evidence that in order to co-ordinate optimum functioning of the motor unit as a whole the electrophysiological activity of motoneurones changes in conjunction with muscle when exposed to altered functional demands. For example, immobilization of the cat soleus leads to a slow to fast myosin transformation. This change is accompanied by a decrease in the afterhyperpolarization response of the motoneurone which effectively increases its minimum-maximum firing frequency (Gallego et al. 1979). Thus the altered force-frequency relationship of the muscle fibres are matched by the motoneurone. Metabolic changes such as increased protein synthesis and oxidative potential after chronic endurance exercise (Gerchman et al. 1975) and increased oxidative enzyme activities after compensatory overload in both the muscle fiber and motoneurone (Pearson & Sickles, 1987) also occur. The mechanisms of nerve/muscle coordination are unknown. Classically, re-innervation studies have shown that the motoneurone input to a muscle fiber determines phenotype expression (Komi, 1986), however motoneurone changes have been observed to precede muscle changes (Foehring et al. 1987). Therefore, adaptation in nerve and muscle may rely on both the amount and pattern of depolarization in the motoneurone as well as orthograde and retrograde axonal transport of trophic substances (Gardiner, 1991).

Nerve morphology and function may also change with exercise. Intense forced training has been demonstrated to decrease nerve fiber diameter, myelin area and mean axon diameter (Roy et al. 1983; Anderson & Edstrom, 1957) while voluntary training (Samorajski et al. 1975) and compensatory overload (Eisen et al. 1973; Walsh et al. 1978) have elicited nerve fiber hypertrophy. Since nerve axon diameter is highly related to nerve conduction velocity (NCV) (Waxman, 1980) any change in diameter would cause a concomitant change in NCV which could affect the rate of force production and therefore muscular power. In spinalized cats, those animals exercised for 13-14 weeks had faster motor NCV than those not exercised (Edgerton et al.

1980). In humans, motor NCV has been reported to be faster in strength than endurance athletes (Kamen et al. 1984). It has also been reported that elite track sprinters have slower NCV than untrained controls (Upton & Radford, 1975) which is surprising since fast conducting motorneurons should theoretically provide an advantage to sprinters by increasing the rate of muscle activation. Sale et al. (1982) found small (3%) but significant increases in NCV in a group that underwent immobilization prior to strength training. Other training studies have shown increases (Singh et al. 1977; Upton, 1976) or no change in NCV (Lehnert & Weber, 1975).

These findings on the influence of training on nerve fiber diameter and conduction velocity are somewhat equivocal. This may be due to inadequate statistical power caused by the small effect being investigated (eg. 3% increase in NCV; Sale et al. 1982) combined with the small sample sizes utilized in the majority of the studies ($n \leq 11$). Furthermore, whether a difference in NCV exists between endurance and power trained athletes remains an open question.

Neural adaptation may also occur within reflex arcs to facilitate activation of agonist or inhibit activation of antagonist muscles (Kamen et al. 1981; Hakkinnen & Komi, 1986; Koceja et al. 1991). Learning to co-ordinate the activation of all muscles involved directly and in stabilizing a movement also contributes to a strength training effect (Rutherford & Jones, 1986). Combined with task specific recruitment of motor units within a muscle (Hoffer et al. 1987) these factors may be responsible for the specificity of movement pattern commonly observed in strength movements (Sale, 1988). Similarly, specific neural adaptation may occur with speed and power training (Upton et al. 1975; Hakkinen et al. 1985; Hakkinen & Keskinen, 1989).

As strength training proceeds, muscle hypertrophy gradually contributes more to strength improvement than neural factors (Moritani & DeVries, 1979; Hakkinnen & Komi, 1986). Muscle hypertrophy subsequent to sprint training also occurs (Thorstensson et al. 1975; Hakkinnen & Keskinen, 1989; Cadefau et al. 1990) yet the magnitude of these changes is usually less than with strength training (Komi, 1986). It is generally agreed that the main contributor to muscular hypertrophy is an increase in myofibrillar volume (Luthi et al. 1986) and some evidence has suggested that this occurs more readily in type II fibers (Thorstennson et al. 1976; Kuno et al. 1990). The extent of skeletal muscle hypertrophy seems to be linked to the enhancement of repair mechanisms stimulated by repeated muscle damage occurring as a result of acute strength training sessions. Eccentric exercise seems to be especially potent at causing muscle damage since higher forces are generated at longer muscle lengths than other modes of contraction (Armstrong et al. 1991). These factors, combined with unequal lengthening of sarcomeres eliciting force mismatches within the muscle (Friden et al. 1992), appear to overload the cytoskeleton and cause focal disruptions in muscle ultrastructure. The cytoskeletal disruptions or metabolic disturbances (decreased ATP) may also damage the sarcoplasmic reticulum and sarcolemma (Byrd, 1992) leading to a disruption in calcium homeostasis. Increased intracellular calcium causes further muscular degradation by activating a variety of proteases and phospholipases (Armstrong, 1991).

Mechanisms to repair this exercise induced muscle damage are stimulated within hours (Clarkson, 1992; Smith, 1992). Neutrophils followed by monocytes migrate to the damaged areas and act to phagocytize pathogens and tissue debris and release an array of cytotoxic factors. The monocytes differentiate to become macrophages and secrete fibronectin and proteoglycans that help stabilize the extracellular matrix and promote cell adhesion. They also release the cytokines interleukin-1 and tumour necrosis factor (Evans & Cannon, 1991). Interleukin-1 activates the acute phase response which further acts to disrupt membranes of non-self cells and provide a pool of amino acids for cytokine-accelerated rates of hepatic protein synthesis (Evans & Cannon, 1991; Smith, 1992). Both transcription and translation of myofibril proteins increase after exercise induced muscle damage (Goldspink, 1991; Russell et al. 1992) and new contractile proteins are added to the periphery of myofibrils creating larger myofibrils without altering filament packing density or crossbridge spacing (MacDougall, 1991). Myofibril number also increases via longitudinal splitting of existing myofibrils (Goldspink, 1970; 1974). Thus hypertrophy seems to be the primary mechanism by which skeletal muscle adapts to force overload so that it is more resistant to damage.

Controversial evidence has also been presented supporting an increase in the number of muscle fibers (hyperplasia) with long term muscle overload since bodybuilders have been reported to have more muscle fibers than controls (Larsson & Tesch, 1986). This could however simply be a genetic predisposition in these athletes that was a factor in their decision to participate in bodybuilding. The dominant hand of young healthy adults has more fibers than the non-dominant hand however (Sjostrom et al. 1991) which is not as easily explained by genetic factors and suggests a possible long-term hyperplasia effect. The development of new myotubes in trained versus untrained muscle has been interpreted as evidence for new fiber formation and hyperplasia (Appell et al. 1988) yet MacDougall (1991) suggested that this could simply represent 1 to 1 replacement of necrotic fibers. Satellite cell proliferation in the absence of necrotic cells has been observed in compensatory hypertrophy (Kennedy et al. 1988) therefore the existence of a mechanism for hyperplasia is possible.

Enzymatic changes within the muscle due to strength training have been reported. Myofibrillar adenosine triphosphatase (ATPase) activity may decrease (Tesch et al. 1987), remain the same (Thorstensson et al. 1976; Houston et al. 1983; Tesch et al. 1990) or increase (Eriksson et al. 1981) and inconsistent findings in the activities of metabolic enzymes subsequent to strength training also exist (Houston et al. 1983; Thorstensson et al. 1976; Tesch et al. 1990; Chalmers et al. 1991). Tesch (1991) suggested that muscle hypertrophy induced by heavy-resistance low-repetition regimens can effectively decrease muscle enzyme activities via a dilution effect. This seems tenable since the stimulus for muscle hypertrophy (muscle damage) could easily be present without a stimulus for metabolic enzyme adaptation (high metabolic flux).

Acute bouts of strength training reduce the concentrations of ATP and creatine phosphate (CP) in muscle (Tesch, 1989). This may provide an adaptive stimulus in muscle for increased storage of these substrates. Indeed, increases in the muscle concentrations of ATP, adenosine diphosphate, creatine, creatine phosphate and glycogen (MacDougall et al. 1977) have been reported with strength training but in some cases no changes have occurred (Tesch et al. 1990).

Sprint or power training may elicit increases in myofibrillar ATPase (Thorstennson et al. 1975), creatine kinase, myokinase (Thorstennson et al. 1975; Cadefau et al. 1990) and enzymes associated with glycolysis (Cadefau et al. 1990). In general, sprint training causes less hypertrophy than strength training (Komi, 1986) but metabolic flux should be higher. These two factors could account for the more consistent observations of improvements in metabolic enzyme activities with sprint versus strength training. Acute sprint exercise also causes decreases in muscle glycogen, ATP and CP (Jacobs et al. 1983) however no increase in muscle concentration of ATP and CP (Thorstennson et al. 1975), but increases in muscle glycogen content (Cadefau et al. 1990) have been reported subsequent to sprint training.

The impact of strength training on sprint/power performance has not been extensively studied. Both slow and fast velocity isokinetic torque has been positively related to rowing (Bell et al. 1989) and cycling anaerobic power (Smith, 1987) as well as 40 m run time (Anderson et al. 1991). Bell et al. (1989) reported no change in 15 or 90s anaerobic power values after five weeks of isokinetic training however. As these investigators hypothesized, this may be due to the anaerobic rowing test involving a more complex movement pattern than utilized in strength training and/or insufficient strength training duration. As well, differences in the central activation of the motorneurone pool in movement and force tasks may cause differences in the co-ordination of synergistic muscles (Tax et al. 1990) which would account for a lack of strength transfer to anaerobic power. Nevertheless, Petersen et al. (1984) showed changes in 30 s anaerobic power on the cycle ergometer after five weeks of isokinetic strength training. Contrary to the study by Bell et al. (1989) where the anaerobic power of skilled rowers was tested on a rowing ergometer, the subjects in this study, swimmers, were not previously trained in the movement used for anaerobic power testing. It is therefore possible that strength training caused neural adaptation in the swimmers which impacted anaerobic power to a greater extent than would have been possible if they were already trained in cycling. However, the influence of strength training on anaerobic power remains equivocal.

Despite similarities in neural and muscular adaptation to strength and power exercise it may be hypothesized that prior strength training may not have an immediate effect on anaerobic power since no time for specific neural adaptation has occurred. As an exception to this, if strength was trained using a complex movement similar to that for which power was required then there may be a more complete power transfer. It may also be hypothesized that power training would be more successful after a period of strength training since an improved quality or quantity of muscle could then be trained neurally to contract faster. No research investigating these hypotheses has been reported.

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