Near-infrared Estimation of Blood Volume and Skeletal Muscle Oxygenation During 4RM and 10RM Resistance Exercise Protocols in Trained and Untrained Males

by
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We accept this thesis as conforming to the required standard

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ABSTRACT

The purpose of this study was to observe muscle oxygenation and blood volume changes elicited by 10RM and 4RM weight lifting in trained (ages 23.8 ± 3.5 years, sum of 4 arm skinfolds 15.2 ± 3.7 mm, arm girth 35.7 ± 3.0 cm) and untrained trained (ages 25.3 ± 3.6 years, sum of 4 arm skinfolds 16.6 ± 3.2 mm, arm girth 28.4 ± 3.1 cm) subjects. 24 college aged males volunteered for the study and were assigned to the trained (N=14) or untrained (N=10) group based on training experience. Each subject completed a 10RM, 4RM and muscle ischemia protocol in random order on separate days. The 10RM protocol consisted of 3 sets of right arm curl exercise at a 10RM load. The 4RM protocol consisted of 4 sets of arm curls at a 4RM load. Each set of exercise was separated by 3 min rest. In the muscle ischemia protocol, subjects had blood flow occluded for 8min by a pressure cuff inflated to 250mmHg. Blood volume and tissue oxygenation of the right biceps brachii muscle were monitored non-invasively in all protocols using a near-infrared spectrophotometer placed on the mid-point of the medial muscle belly of the biceps brachii. Both trained and untrained subjects showed marked decreases in blood volume and tissue oxygenation during each set of exercise in both the 4RM and 10RM protocols. No differences existed between trained and untrained groups in a set-wise comparison of muscle oxygenation and blood volume in the 10RM and 4RM protocols. No set-wise differences were found for the 10RM or 4RM protocol for either the trained or untrained subjects. Further, no differences existed between 10RM and 4RM deoxygenation and blood volume values for the trained and untrained groups. It was concluded that blood volume and oxygenation decrease during resistance exercise

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Gladys, Norma, and Jill. I could not have graduated without your help and humour. Thank you.

Thanks to my partner in crime Dave Harrison for the endless hours of data collection, analysis, and brainstorming (often in my absence).

Thanks to all those who helped in the study, to my roommates, family and friends who believed I would someday finish even when I did not. I could not have done this without you.

DEDICATION

To my parents: Your unfaltering support through all my endeavours, both academic and athletic allowed me to achieve all I have. I could not have done any of this without you.

INTRODUCTION

Strength is defined as the peak force or torque a muscle or muscle group can generate during a maximal voluntary contraction (Goldspink, 1992). The ability of an athlete to generate force depends on the size of the involved muscles and the ability of the nervous system to activate these muscles (Sale, 1992). Increases in strength can be realized by stimulation of the neuromuscular system through resistance training exercise. Adaptations to resistance exercise resulting in strength increases occur through neural and/or muscular mechanisms (Sale, 1988; Moritani, 1992). There is a wide variation in the design of resistance training programs and different sports employ variations in the exercises and the training parameters of type, volume, rest interval and load. As a result, a variety of adaptations in the muscular and neural systems are observed corresponding to the different training protocols.

Alterations in the distribution of fiber types in human skeletal muscle have been observed following training. Endurance training and high intensity intermittent training have resulted in increases in the percentage distribution of ST or type I muscle fibers (Jansson, Sjodin, & Tesch, 1978; Simoneau, Lortie, Boulay, Marcotte, Thibault, & Bouchard, 1985). Jansson and Kaiser (1977) and Andersen and Henriksson (1977) found endurance training caused a decrease in percentage of type IIb fibers and an increase in percentage of type IIa fibers suggesting a shift from type IIb to IIa. Staron, Malicky, Leornardi, Falkel, Hagerman, and Dudley (1989) suggested this fiber conversion occurred to increase the oxidative capacity of the muscle as IIa fibers are more oxidative than IIb fibers.

Several strength training studies have also shown a conversion of IIb to IIa fibers over the course of training. Staron et al. (1989) examined the effects of a heavy lower body resistance training program that was performed twice per week for 20 weeks on female subjects. Subjects performed two sub-maximal warm-up sets (40-60% of 1 RM) followed by three sets of 6-8 RM for four lower body exercises each training session. The training resulted in significant hypertrophy in all three fiber types (I, IIa, IIb) and a decrease in type IIb fiber percentage with a concomitant increase in mean type IIa percentage. This suggests that there was a conversion of IIb to IIa fibers over the course of training. In a follow up study, Staron et al. (1991) required the same population to detrain for 30 to 32 weeks then retrain for either 6 or 13 weeks. In the course of detraining there was an increase in the percentage of IIb fibers and a decrease in the percentage of IIa fibers toward pre-training values. After the 6 week retraining period there was a conversion of IIb to IIa fibers. There were no type IIb fibers found in the group completing 13 weeks of retraining. Similarly, Adams, Hather, Baldwin, and Dudley (1993) observed a 12% decrease in myosin heavy chain (MHC) IIb content and a 12% increase of MHC IIa content in men performing multiple sets (4-5) of high repetitions (6-12) of concentric and eccentric resistance exercise over 19 weeks. In addition, Kraemer et al. (1995) found a decrease in percent IIb fibers (17.3%) following three months of heavy resistance exercise consisting of two heavy (5RM) and two moderate (10RM) training sessions per week. These studies showed a lower percentage of IIb fibers and a higher percentage of IIa fibers after resistance exercise that suggested a IIb to IIa fiber conversion.

Although a fiber shift can occur in resistance training, all resistance trained subjects do not exhibit lower percentages of type IIb fibers. Bodybuilders were found to possess a lower percentage of type II fibers compared to powerlifters (Tesch & Larsson, 1982). However, Klitgaard, Zhou, and Richter (1990) found bodybuilders had a higher percentage of fibers with a greater MHC IIa content and a lower percentage of fibers expressing both MHC IIa and IIb compared to sedentary men. Further, only the bodybuilders demonstrated a trend toward a lower proportion of IIb fibers and higher proprotion of type IIa fibers. Conversely, powerlifters have been shown to contain a high percentage of fast twitch fibers (Tesch & Karlsson, 1985;Tesch, Thorsson, & Kaiser, 1984). However, Tesch et al. (1984) found no difference in fiber distribution between the quadriceps of powerlifters and non-athletes

It appears that a fiber conversion may occur in bodybuilders and not power-lifters. This may be the result of the type of training used by the two groups of athletes. The exercise regimens of powerlifters are variations of a common theme. Typical exercises include the snatch, front squat, dead-lift, power-clean, and clean and jerk (Garhammer & Takano, 1992). Training includes approximately 5-12 sessions per week, 3-5 exercises per session, with loads between 1 and 5 RM. Rest intervals between sets are approximately four to five minutes (Garhammer & Takano, 1992). Bodybuilding routines are highly varied in loading, volume, frequency and rest. Generally programs include multiple exercises per muscle group, multiple sets per exercise (3-5), high repetitions (6-12) and short rest intervals between sets (1-2 minutes) (Tesch, 1992). Each exercise is typically performed to muscle failure. All the studies previously discussed in which fiber type conversions were observed employed bodybuilding-like loading parameters.

The cause of the IIb to IIa fiber conversion is unclear. Lind and Williams (1979) reported a decrease in muscle blood flow in the forearm at contractions of 60% and 100% MVC. Hulten, Thorstensson, Sjodin, and Karlsson (1975) suggested that blood flow at tensions greater than 50% MVC is severely restricted allowing little or no oxygen delivery to the tissues. Therefore, it is possible that the loads used by power-lifters and bodybuilders are large enough to severely restrict blood flow that may impair tissue oxygenation. Tamaki, Uchiyama, Tamura, and Nakano (1994) found that an exercise protocol involving multiple sets of 10 RM arm curls resulted in a rapid decrease in oxy-Hb/Mb saturation and an increase in deoxy Hb/Mb. Further, total Hb/Mb levels increased during exercise and did not return to resting level between sets. The blood volume changes and lack of oxygen supply was interpreted as muscle anoxia that was accelerated by short rest intervals. They suggested that the high number of repetitions, multiple sets and exercises, and short rest periods seen in the training sessions of bodybuilders may expose the exercising muscle to insufficient oxygen supply for extended periods of time. They stated that this lack of oxygen supply may be a cause of the conversion of IIb to IIa fibers associated with resistance exercise. No studies to date have examined muscle oxygenation changes during power-lifter type training involving high load, low repetition, long rest protocols.

Near infrared spectroscopy (NIRS) is a non-invasive method that can be used to measure relative changes in tissue oxygenation in humans. The NIRS method relies on ability of NIR light (700-1000nm) to pass through biological tissues (Hampson & Piantadosi, 1988). Light emitted by the NIR probe is primarily scattered and absorbed as it passes through body tissues except for a portion of the original incident light. This light is reflected and picked up by the photo detectors of the probe (Belardinelli, Barstow, Porszasz, & Wasserman, 1995). NIRS use in the monitoring of tissue oxygenation is based on the difference in absorption spectra between the oxygenated and deoxygenated forms of hemoglobin (Hb) and myoglobin (Mb), the oxygen carrying molecules in blood and muscle (Sahlin, 1992) and has been shown to be a valid indicator of muscle oxygenation (Mancini et al., 1994).

NIRS has been used successfully to monitor Hb/Mb oxygenation in a variety of applications in humans. These include tissue monitoring in ischemic conditions (Hampson & Piantadosi, 1988), in patients with arterial disease and heart failure (Wilson et al., 1989), and as a non-invasive instrument to monitor newborns (Tamura, Eda, Takada, & Kubodera, 1988). More recently, NIRS has been used to evaluate muscle oxygenation under a variety of exercise conditions. Belardinelli, Barstow, Porszasz and Wasserman (1995a: 1995b) used NIRS to monitor Hb/Mb desaturation during incremental cycle exercise. Others have used NIRS to monitor Hb/Mb saturation during hypoxic arm crank exercise (Jensen-Urstad, Hallback, & Sahlin, 1995; Bhambhani, Maikala, & Buckley, 1998) and to measure resaturation/recovery times in elite rowers on both cycle and rowing ergometers (Chance, Dait, Zhang, Hamaoka, & Hagerman, 1992).

Although a number of studies have used NIRS to evaluate oxygenation changes of Hb/Mb during exercise, few studies have examined oxygenation changes during resistance type exercise. Homma, Eda, Ogasawara, and Kugaya (1996) investigated post-exercise oxygenation changes in rhythmically contracting forearm muscles exercising at loads between 5% and 30% MVC. However, deoxygenation was not monitored throughout the exercise bouts. Because blood flow is restored shortly after the cessation of exercise (Lind & Williams, 1979), any oxygenation changes during exercise may not be represented. Tamaki et al. (1994) examined Hb/Mb oxygenation in contracting muscle during resistance-training exercise. They employed a ten repetition maximum (10RM) arm curl protocol and found increased Hb/Mb and decreased HbO_2/MbO_2 over the course of the exercise protocol. Further, increased blood volume with the performance of multiple sets of exercise with short rest periods (manifested as an increase in total Hb/Mb) was observed. This is the only study to date examining tissue oxygenation under modern resistance training protocols.

Although the technology is available, no studies to date have examined tissue oxygenation changes during powerlifter-type training involving high load, low repetition, long rest protocols. Further, no research has compared tissue oxygenation and blood volume changes during different weight lifting protocols. Therefore, it is not known whether protocols involving the powerlifter type of loading elicit similar oxygenation changes to bodybuilding type loading. Similarities or differences in muscle oxygenation patterns under the two loading conditions may provide insight into the underlying cause of the fiber-type shift that is observed in bodybuilders but absent in powerlifters. The purpose of this study is to observe tissue oxygenation and blood volume changes consequent to loading protocols typically used by powerlifters (4RM) and bodybuilders (10RM) using NIRS technology.

Statement of the Problem

Four major problems addressed in the present study were:

1. To identify changes in tissue oxygenation and blood volume during a dynamic 4RM resistance exercise protocol on the right biceps brachii muscle

2. To identify changes in tissue oxygenation and blood volume during a dynamic 10RM resistance exercise protocol on the right biceps brachii muscle
3. To determine if changes in tissue oxygenation and blood volume during 4RM and 10RM resistance exercise protocols are significantly different
4. To determine if changes in tissue oxygenation and blood volume during 4RM and 10RM resistance exercise protocols are significantly different between trained and untrained subject groups

Hypotheses

The following null hypotheses were tested:

Ho1: The 4RM resistance protocol will have no significant effect on the desaturation of Hb/Mb

Ho2: The 10RM resistance protocol will have no significant effect on the desaturation of Hb/Mb

Ho3: No significant difference will exist between the effects of the 4RM and 10RM resistance protocols on tissue oxygenation for each subject

Ho4: No significant difference in tissue oxygenation will exist between trained and untrained subjects in the 4RM resistance protocol

Ho5: No significant difference in tissue oxygenation will exist between trained and untrained subjects in the 10RM resistance protocol

Ho6:The 4RM resistance exercise protocol will have no significant effect on blood volume

Ho7: The 10RM resistance exercise protocol will have no significant effect on blood volume

Ho8:No significant difference in blood volume changes will exist between trained and untrained subjects in the 4RM protocol

Ho9: No significant differences in blood volume changes will exist between trained and untrained subjects in the 10RM protocol

Ho10: No significant differences in tissue deoxygenation or blood volume will exist between sets for trained and untrained subjects in the 10RM protocol

Ho11: No significant differences will exist in tissue oxygenation or blood volume between sets for trained and untrained subjects in the 4RM protocol

Ho12: No significant differences will exist set-wise between trained and untrained subjects in tissue oxygenation or blood volume for the 10RM protocol

Ho13:No significant differences will exist set-wise between trained and untrained subjects in tissue oxygenation or blood volume for the 4RM protocol

Assumptions

1. Near infared spectroscopy (NIRS) is a valid and reliable measure of Hb/Mb deoxygenation and blood volume in human skeletal muscle
2. The exercise protocols will elicit measurable changes in Hb/Mb oxygenation and blood volume
3. The 4RM and 10RM resistance exercise protocols are representative of powerlifter- and bodybuilder-type training, respectively
4. Desaturation of Hb/Mb is a valid and reliable indication of tissue oxygen consumption
5. Subjects are giving maximal effort during testing and abide by study restrictions

6.The rest interval provided between sets was enough to allow blood volume and tissue oxygen levels recover toward baseline levels

Delimitations

1.Only heavy resistance trained, college-aged, male volunteers were eligible to participate in the study.

2.The use of NIRS provided a non-invasive estimation of Hb/Mb oxygenation changes and blood volume changes in muscle. Consequently, values for the amount of oxygen consumed or blood volume change were unavailable through this technique

3.Only the biceps brachii of the right arm was monitored during the exercise protocols. This may limit the generalizability of results to other muscle groups in the body and excluded other agonists involved in elbow flexion.

Limitations

1.Muscle fiber composition of individual subjects may have differed. Consequently, individuals may have possessed different levels of muscle oxygen consumption.

2.Subjects may not have been able to complete the exercise protocol.

3.Testing must be completed in the limited time frame the NIRS equipment is available. This may have limited the subjects available to participate in the study.

Operational definitions

1) 10RM: The maximum amount of weight that can be lifted for 10 repetitions

2) 4RM: The maximum amount of weight that can be lifted for 4 repetitions

3) Repetitions: The number of times a weight is lifted consecutively without rest

4) Set: Unit consisting of the number of repetitions performed consecutively

5) Hemoglobin(Hb): Complex molecule found in red blood cells containing iron and protein. It serves as an oxygen carrying compound in blood (Fox, Bowers, & Foss, 1993).

6) Myoglobin(Mb): Complex oxygen binding molecule, similar to Hb, found in muscle. It acts as an oxygen store and aids in transport (Fox et al., 1993).

7) Trained subject: Individual who has been involved in heavy resistance training a minimum of twice a week for at least 1 year

8) Near Infrared Spectroscopy (NIRS): Non-invasive method of monitoring tissue oxygenation in human skeletal muscle. It is based on the differential absorption spectra for oxygenated and deoxygenated forms of Hb and Mb at different wavelengths of NIR light. By comparing differences in reflected light intensity at the monitored wavelengths, relative measures of Hb/Mb oxygen saturation are obtained (Sahlin, 1992).

METHODS

Subjects

24 college-aged males volunteered for the study. Subjects were divided into two groups. The trained group had a minimum of 1 year of heavy resistance training experience including at least 2 sessions per week while the untrained group had no previous resistance training experience. Each subject performed both the 10RM and 4RM resistance exercise protocols. Approval from the University of Victoria Human Ethics committee was obtained prior to the commencement of data gathering. Further, all subjects signed an informed consent form agreeing to participate in the study.

Exercise Protocols

One week prior to the start of testing, all subjects were assessed for right forearm (elbow) flexion 10RM. This was performed on a seated preacher curl bench with a progressively loaded dumbbell until 10RM was determined. The 4RM weight was predicted from the 10RM value using a strength continuum (Baechle, 1994). Subjects moved the weight between 110 and 45 degrees, determined by hand held goniometer, to constitute a successful repetition. The concentric and eccentric phases of each contraction were 1.5s, determined by metronome, totalling 3s for 1 repetition of exercise. Five min rest was given between each attempt.

The 10RM and 4RM exercise protocols were performed on the seated preacher curl bench. Joint angles for the range of motion and contraction lengths were the same as the protocol used to determine 10RM. A loaded dumbbell, equivalent to the predicted 10RM weight for the subject, was placed in the right hand at the 45° angle in the range of motion. The subject then performed 10 contractions to the metronome tempo. At completion of the exercise, the subject rested for 3 min then repeated the procedure. This protocol was repeated for 3 sets of exercise, each separated by 3 min of rest. The 4RM protocol used the same range of motion, contraction time, apparatus, and procedure as the 10RM protocol. However, instead of performing 3 sets of 10RM separated by 3 min of rest, 4 sets of 4RM separated by 3 min of rest were performed. Therefore, 12s of exercise per set were performed for the 4RM protocol and 30s of exercise per set for the 10RM protocol. Order of protocols was randomized and 2 days rest was given between testing sessions.

Muscle Oxygenation Measures

Tissue oxygen saturation was monitored using a commercially available continuous wave near infrared spectrophotometer (RUNMAN, NIM Inc., Philadelphia, Pa.). The desaturation signal was monitored on a dual chart recorder.

During the exercise protocols, the NIRS probe was placed on the middle of the medial head of the right biceps brachii muscle over a layer of cellophane wrap to prevent moisture contamination (Bhambhani et al., 1998). The optical probes were spaced 3 cm apart. It was covered by a black shield band that secured the probe to the muscle without causing compression of the exercising muscle. This position was marked with permanent ink on each subject. Prior to the commencement of exercise a stable optical baseline was obtained. This was followed by a warm-up of 10 repetitions at 50% of the predicted 4RM.The 4RM or 10RM protocol was then performed. All values for blood volume and muscle oxygenation for each subject were expressed as a percentage of the desaturation and blood volume cuff occlusion range and recorded as a percentage. An ischemic condition was performed by each subject as well as the exercise protocols. A pressure cuff was placed on the right upper bicep over the NIRS probe and shield band. A stable optical baseline was found and the subjects rested in with their arm extended for 2 min. After the baseline period, the cuff was inflated to 250mmHg, completely occluding blood to the tissue. The cuff was left inflated for 8 min and then released (Hampson & Piantadosi, 1988). The values taken at the end of the 8min occlusion period were considered maximum tissue desaturation.

Absorbances at two wavelengths, 760nm and 850nm, were monitored. Absorbance at 760nm represents the peak of the absorbance difference spectrum of deoxygenated Hb/Mb while 850nm is near the isosbestic point between oxygenated and deoxygenated Hb/Mb (Chance et al., 1992). Changes in the difference between the wavelengths [Δ(760-850)] estimates the changes in relative Hb/Mb desaturation while blood volume changes are estimated by the sum of the wavelengths [Δ(760+850)]. All values were converted to optical densities (OD) through Microsoft Excel using Lambert-Beers' Law. Deoxygenation and total Hb/Mb values (blood volume) were expressed as changes in OD (ΔOD). The mean peak OD was determined for both the desaturation and blood volume signals for each exercise protocol. These values were compared within each subject.

Statistics

Independent t-tests were conducted using SPSS version 10.0 to determine significant differences within and between the means trained and untrained groups for the 10RM and 4RM exercise protocols. An ANOVA was conducted to determine if any differences occurred within and between trained and untrained subjects in a set-wise comparison for the 10RM and 4RM exercise protocols. Significance was set at $p<0.05$.

RESULTS

Blood Volume

The raw data of a representative subject is presented in Figure 1. Blood volume data, the sum of deoxygenated and oxygenated Hb/Mb, is expressed in optical density (OD). A full exercise protocol is shown in the graph consisting of a warm-up followed by three sets of exercise, each separated by a rest interval. The fall of the data points at the onset of exercise for set 1, a decrease in optical density, indicates a decrease in blood volume during the exercise set. At the cessation of exercise optical density increases, indicating an increase in blood volume. These values returned toward baseline values during the rest interval. This pattern was consistent for subsequent exercise sets and for all subjects in both the 4RM and 10RM exercise protocols.

The set-wise comparison of blood volume changes between subject groups for the 10RM exercise protocol is shown in Figure 2. Changes in blood volume were calculated by comparing the range of blood volume change for each subject for each set of exercise to the range achieved in the cuff occlusion protocol. These values were then expressed as a percentage of the cuff occlusion range. Both the trained and untrained groups showed decreased blood volume in each set of exercise. Values for each set are presented in Table 1. Blood volume ranged from 119% to 135% for the trained group and 115% to 126% for the untrained group in relation to the occluded state.

Figure 3 shows the set-wise comparison of blood volume changes for the 4RM protocol between trained and untrained subjects. Percentage blood volume changes were calculated in the same manner as in the 10RM protocol. Both subject groups showed a decreased blood volume during each set of exercise. These values are presented in Table

1. The trained group had blood volume changes ranging from 119% to 129% while the untrained group had values ranging from 113% to 118% in relation to the occluded state. The mean values for changes in blood volume for the trained 10RM and 4RM protocols and untrained 10RM and 4RM protocols are shown in Table 1.

No statistically significant differences were found between sets for either the trained or untrained groups in both the 10RM and 4RM protocols. Further, no significant differences were found between 10RM and 4RM exercise protocols for both trained and untrained subject groups. Finally, no significant differences existed in a set-wise comparison between trained and untrained groups in both the 10RM and 4RM exercise protocols.

Muscle Oxygenation

The raw data of a representative subject is presented in Figure 4. Oxygenation data for deoxyhemoglobin/myoglobin (Hb/Mb) is expressed in optical density (OD). The graph shows a warm-up set and three subsequent sets of exercise interspersed by rest intervals. The rise in the data points at the onset of exercise for Set 1, or an increase in optical density, indicates increasing muscle tissue deoxygenation. At the cessation of exercise, represented by the peak indicated on the graph, the data points return toward starting values, indicating rapid tissue re-oxygenation. These values return toward baseline values during the course of the rest period. This pattern continues for subsequent exercise sets and was present for all subjects in both exercise protocols.

Table 1.

Mean physical characteristics (SD), age (years), and sum of biceps brachii skinfold of anterior, posterior, medial, and lateral aspects of the upper arm (mm), relaxed arm girth (cm), 10RM (lbs) and 4 RM (lbs) of trained (N=14) and untrained (N=10) subjects

Group	Age	Skinfold	Arm Girth	10RM	4RM
Untrained	23.84(3.48)	15.20(3.65)	28.37(3.09)	28.24(3.92)	34.25(5.41)
Trained	25.30(3.61)	16.56(3.18)	35.68(3.00)	39.81(10.48)	48.85(13.10)

Figure 1. Muscle blood volume raw data for a representative subject expressed in optical density (OD)

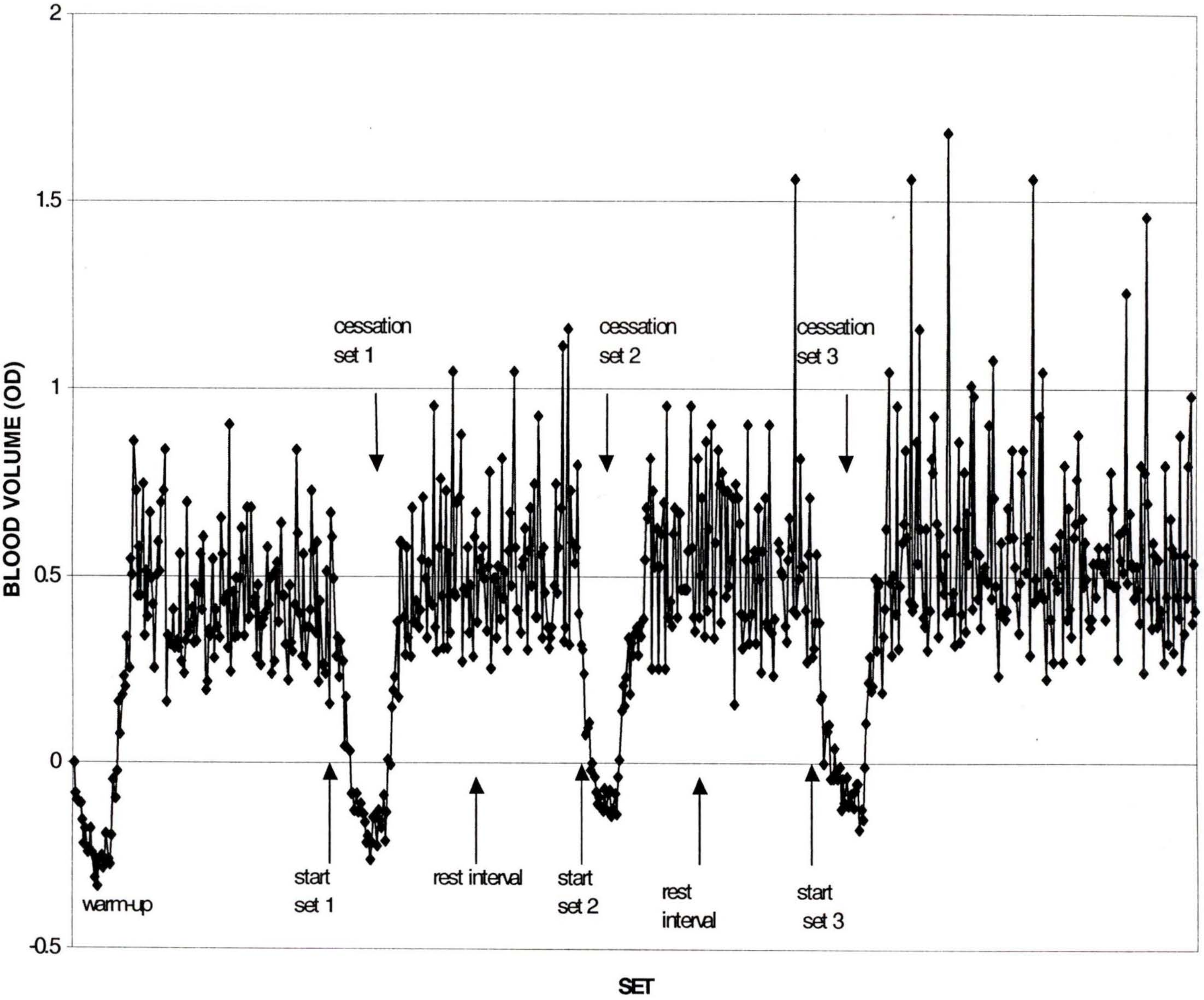

2
1.5
1
0.5
0
-0.5
BLOOD VOLUME (OD)
SET
cessation set 1
cessation set 2
cessation set 3
warm-up
start set 1
rest interval
start set 2
rest interval
start set 3

Figure 2. Mean change (%) in blood volume values (SEM) for trained (N=14) and untrained (N=10) subject groups for each set of the 10RM exercise protocol

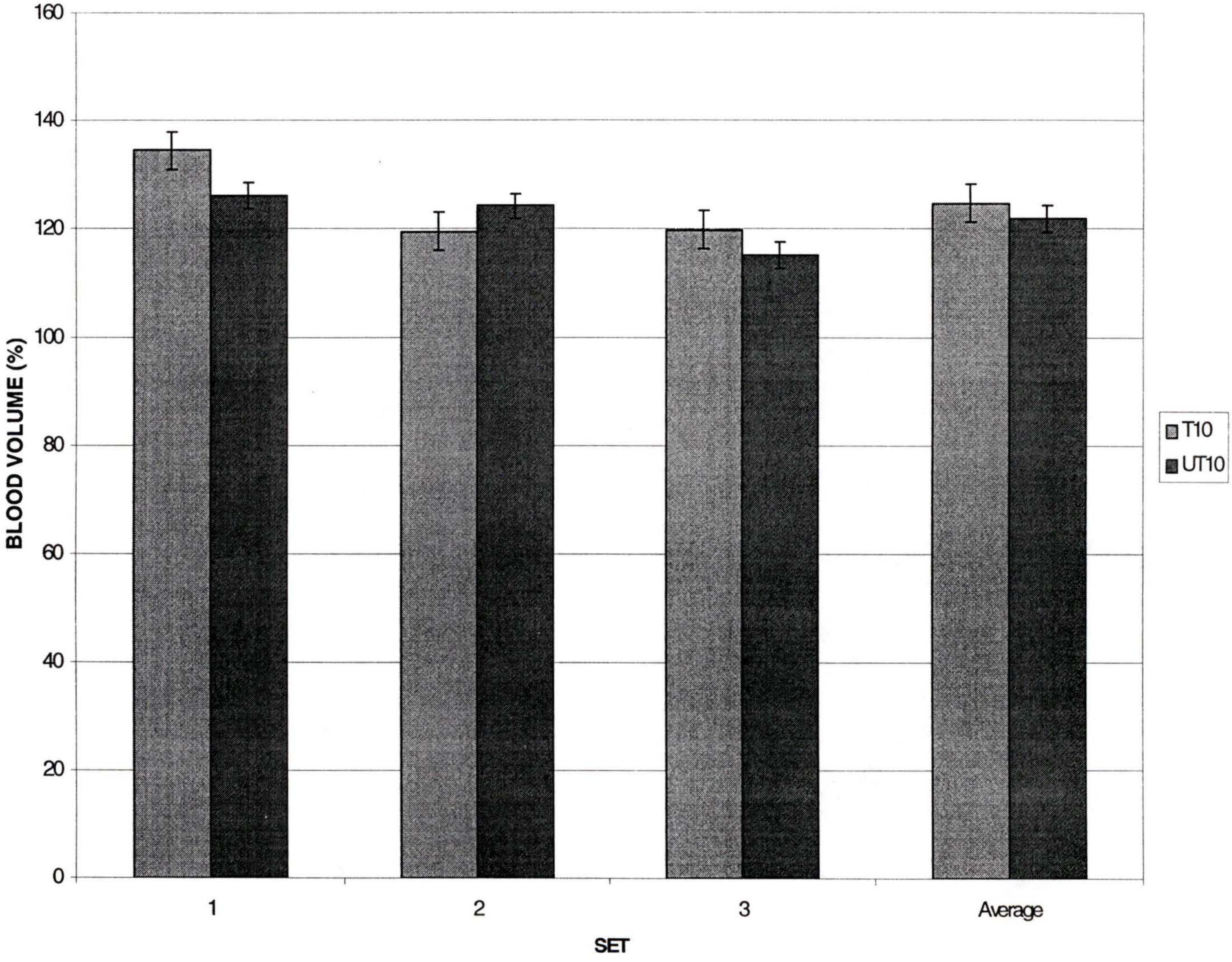

160
140
120
100
80
60
40
20
0
BLOOD VOLUME (%)
1
2
3
Average
SET
T10
UT10

Table 2:

Mean (SD) values for relative blood volume (BV) change for trained (N=14) and untrained (N=10) males in the 10RM and 4RM exercise protocols.Values are expressed as a % of the cuff occlusion protocol.

Group	Set				
	1	2	3	4	Average
Trained					
BV(%)					
10RM	135(45.3)	119(43.5)	119(54.1)	X	125
4RM	129(52.7)	121(49.5)	119(55.4)	121(49.0)	123
Untrained					
BV (%)					
10RM	126(52.7)	124(45.3)	115(43.0)	X	122
4RM	114(41.0)	118(37.9)	117(38.7)	113(36.9)	115

Figure 3. Mean change (%) in blood volume values (SEM) for trained (N=14) and untrained (N=10) subject groups for each set of the 4RM exercise protocol

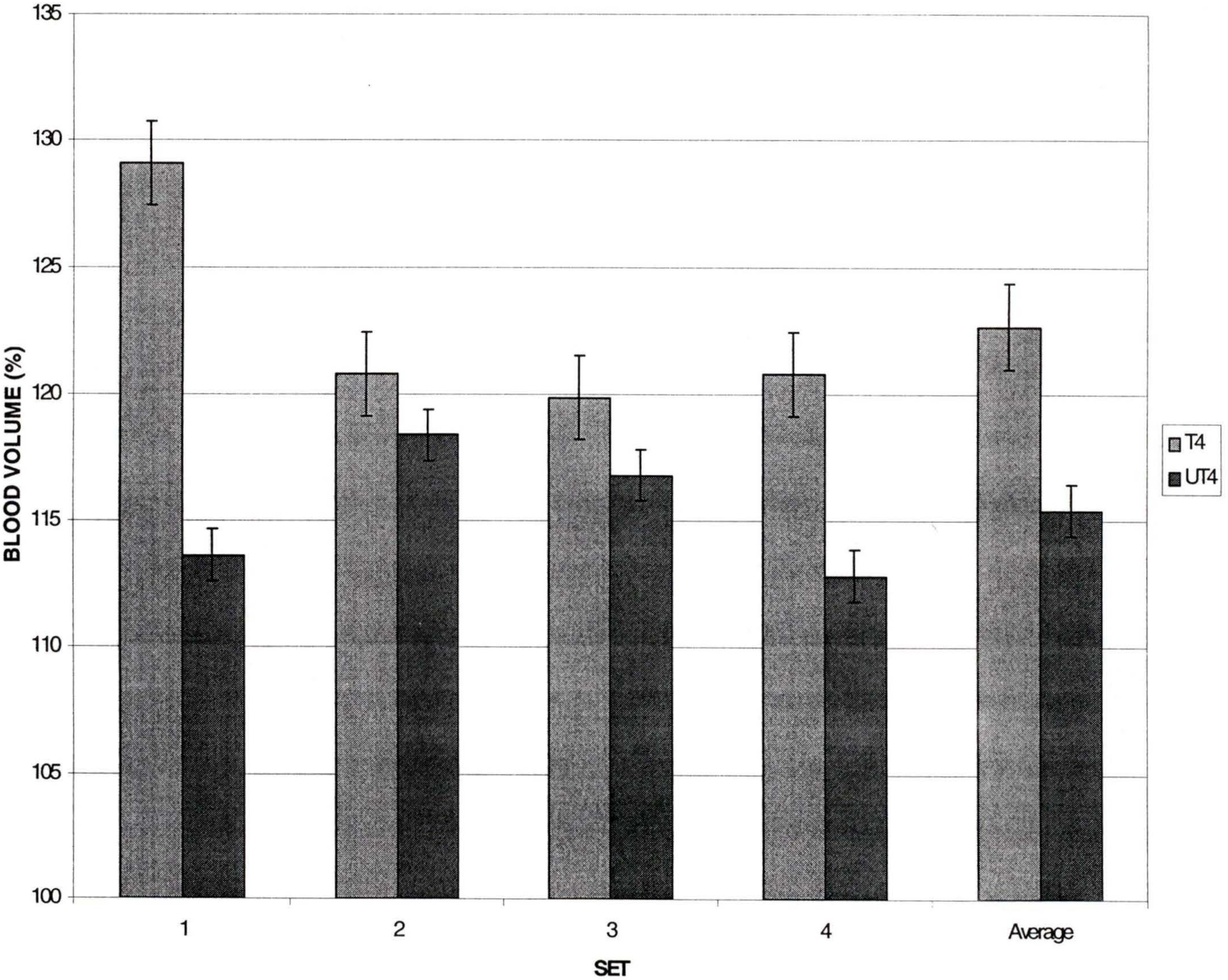

BLOOD VOLUME (%)
135
130
125
120
115
110
105
100
1
2
3
4
Average
SET
T4
UT4

Figure 5 shows the set-wise comparison of muscle deoxygenation for the 10RM exercise protocol between trained and untrained subjects. Deoxygenation values were calculated by comparing the range of deoxygenation for each subject for each exercise set to that of 100% deoxygenation range achieved by the cuff occlusion protocol. These values were then expressed as a percentage of maximum deoxygenation. Both the trained and untrained subject groups displayed marked muscle deoxygenation in each set of exercise. Values for each set are presented in Table 2. Muscle deoxygenation ranged from 58.2% to 78.6% in the trained group and from 66.7% to 92.1% in the untrained group for the 10RM protocol.

The set-wise comparison between trained and untrained subject groups for the 4RM protocol is shown in Figure 6. Percentage deoxygenation values for the 4RM protocol were calculated in the same manner as that of the 10RM group. Both subject groups showed tissue deoxygenation during each set of exercise(these average values are presented in Table 2). The trained group had deoxygenation values ranging form 50.1% to 67.2 while the untrained group had values ranging from 58.8% to 85.7%.

The mean deoxygenation value for the trained 10RM protocol was 65.4% while the trained 4RM protocol was 56.8%. The values for the untrained 10RM and 4RM were 81.6% and 77.7% respectively(these values are presented in Table 2).

However, no statistical significance was found between sets for either the trained or untrained groups in both the 10RM and 4RM protocols. Further, no significant differences were found between 10RM and 4RM exercise protocols for both trained and untrained subject groups. Finally, no significant differences existed in a set-wise comparison between trained and untrained groups in both the 10RM and 4RM exercise protocols.

Figure 4. Muscle deoxygenation raw data for a representative subject expressed in optical density (OD)

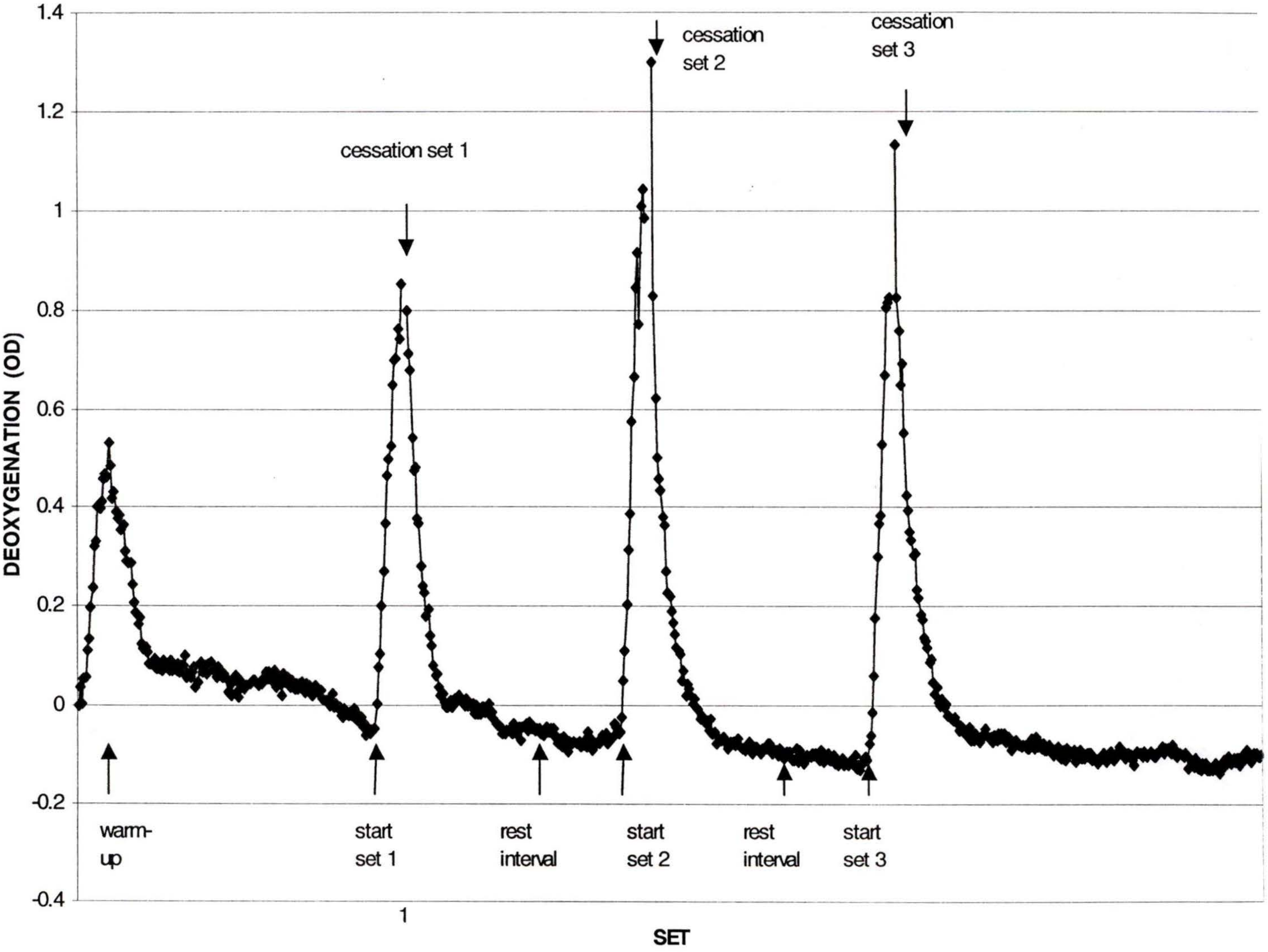

1.4
1.2
1
0.8
0.6
0.4
0.2
0
-0.2
-0.4
DEOXYGENATION (OD)
cessation set 1
cessation set 2
cessation set 3
warm-up
start set 1
rest interval
start set 2
rest interval
start set 3
1
SET

Figure 5. Mean relative (%) deoxygenation values (SEM) for trained (N=14) and untrained (N=10) subject groups for each set of the 10RM exercise protocol.

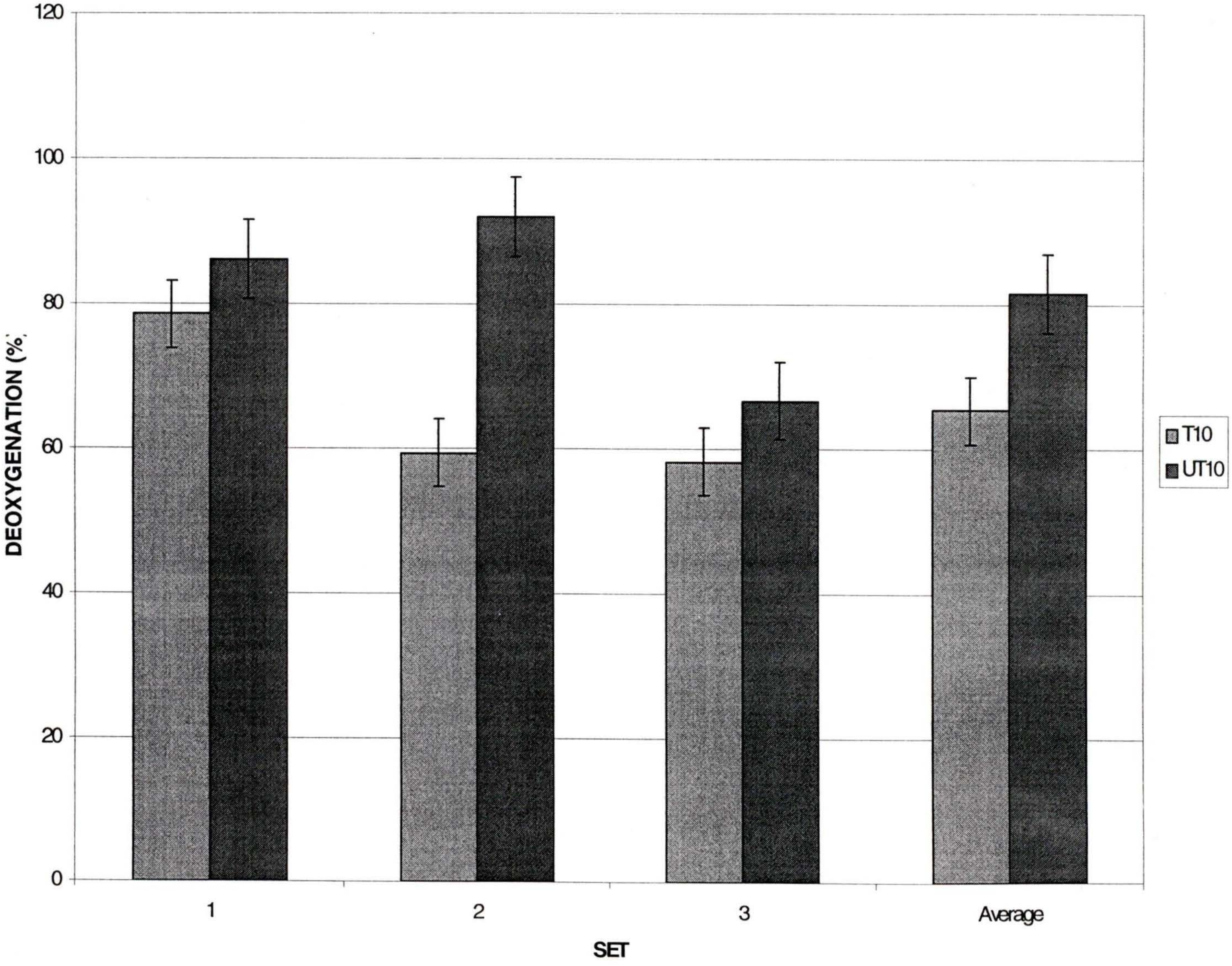
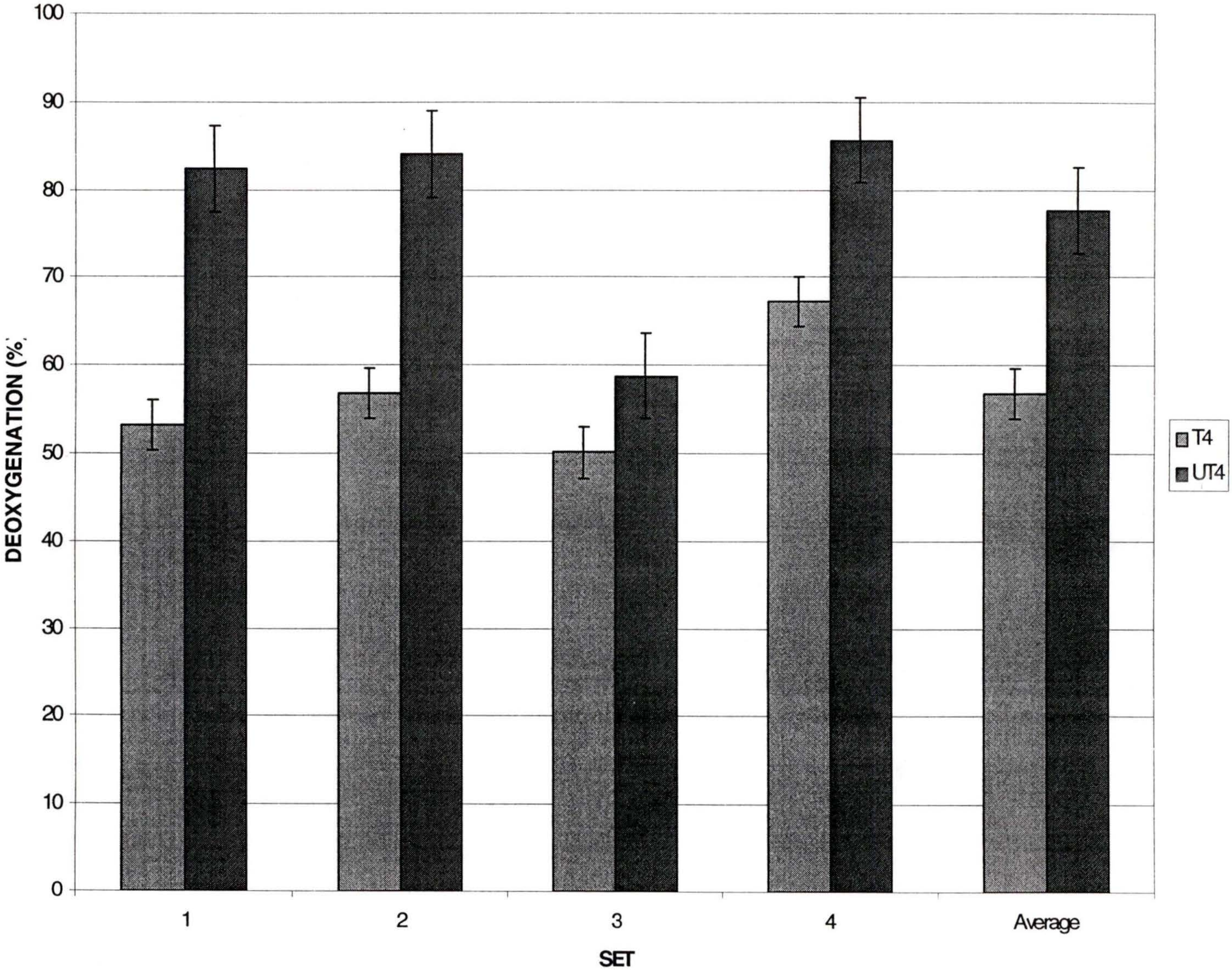

120
100
80
60
40
20
0
DEOXYGENATION (%
1
2
3
Average
SET
T10
UT10

Table 3:

Mean (SD) values for relative muscle deoxygenation (De02) change for trained (N=14) and untrained (N=10) groups in the 10RM and 4RM exercise protocols. These values were expressed as a percentage of the range of the cuff occlusion protocol.

Group	Set				
	1	2	3	4	Average
Trained					
De02(%)					
10RM	78.6(38.5)	59.4(31.5)	58.2(30.8)	X	65.4
4RM	53.5(25.3)	56.8(34.8)	50.1(17.2)	67.2(43.3)	56.8
Untrained					
De02(%)					
10RM	86.2(50.6)	92.1(69.5)	66.7(25.3)	X	81.6
4RM	82.3(84.8)	84.1(49.4)	58.8(19.8)	85.7(73.1)	77.7

Figure 6. Mean relative (%) deoxygenation values (SEM) for trained (N=14) and untrained (N=10) subject groups for each set of the 4RM exercise protocol.


100
90
80
70
60
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DEOXYGENATION (%)
1
2
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Average
SET
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Discussion

Blood Volume

It has been proposed that the hemodynamics during resistance training are similar to what occurs when a tourniquet is applied to produce venous and arterial blood occlusion. Several studies have investigated the effect of both venous and arterial occlusion on muscle. A significant deoxygenation of Hb was found to occur as a result of venous and arterial occlusion without significant increases in muscle blood volume during an 8 min forearm ischemia protocol (Hampson and Piantadosi 1988). Upon tourniquet release blood volume increased above resting levels before returning to near baseline. A decrease in tissue oxygenation and a small increase in blood volume was also found to occur during a 20 min tourniquet protocol involving the thigh (Sahlin, 1992). The slight increase was attributed to a shift in muscle geometry during contraction that influenced light penetration depth from the NIRS probe that was used. Like the former study, once the pressure cuff was released both tissue oxygenation and blood volume values increased above baseline. Homma, Eda, Ogasawara, and Kagaya (1996) investigated blood volume changes during a venous occlusion protocol. A pressure cuff was inflated to 60mmHg around the upper arm and forearm blood volume was determined using NIRS. They reported significant increases in blood volume during this protocol. Mancini et al. (1994) also reported increased blood volume during a similar venous occlusion protocol.

It has been shown that blood flow is impaired during resistance exercise. Lind and Williams (1979) found that muscle blood flow decreased during 60% and 100% MVC isometric handgrip contractions but increased, as a result of the reactive hyperemia to exercise, immediately following the cessation of the handgrip exercise. Similarly, Hulten, Thorstensson, Sjodin, and Karlsson (1975) reported that blood flow at tensions corresponding to 50% MVC is severely restricted, which would limit oxygen delivery to the working tissue. The blood volume increases subsequent to resistance training (Tamaki, Uchiyama, Tamura, & Nakano, 1994) resemble those seen in the venous occlusion protocols. Therefore, blood flow impairment during resistance exercise is likely a consequence of exercise-induced venous occlusion.

Resistance-type exercise has been shown to elicit increases in blood volume during training. Tamaki, Uchiyama, Tamura, and Nakano (1994) reported increases in blood volume during weight lifting exercise. Subjects performed 3 consecutive sets of 10RM exercise, separated by 1min rest, using a single arm curl protocol. Blood volume decreased slightly at the start of exercise and then increased gradually to a peak at the cessation of lifting. This occurred in all sets. Tamaki et al. (1994) suggested that the increase in blood volume observed during exercise was the result of exercise-induced venous occlusion without complete arterial occlusion. Consequently, arterial inflow occurred during exercise without an equally large venous outflow resulting in an increase in blood in the muscle. This is not consistent with the findings of the present study that found a decrease in blood volume from the start to cessation of exercise (Figure 1). This was consistent for all subjects across all sets for both the 4RM (N=10) and 10RM (N=14) exercise protocols. Although the decrease in blood volume seen in the present study is not in agreement with others, some explanations exist. Both Sahlin (1992) and Tamaki et al. (1994) observed a temporary decrease in muscle blood volume after the start of resistance exercise. This was attributed to a redistribution of blood or a squeezing of venous blood out of the muscle by the contraction thereby increasing venous return and temporarily decreasing blood volume. Similarly, Vankan et al. (1998) suggested venous outflow during contraction is strongly increased. Lind and McNicol (1967), Lind and Williams (1979), and Hulten et al. (1975) suggested that blood flow is severely impaired at exercise intensities above 25% MVC. The exercise intensities in the present study were approximately 73% MVC for the 10RM protocol and 88% MVC for the 4RM protocol (Baechle, 1994). Therefore, it can be assumed that there was a large degree of blood flow impairment during exercise. Vankan et al. (1998) also stated that arterial inflow to the muscle during contraction is strongly decreased at the onset of contraction. Therefore, blood volume could decrease in a multiple contraction exercise protocol if venous outflow exceeded arterial inflow and blood flow to the muscle was compromised. This would result in a decrease in blood volume over the course of the set as each contraction forced more blood out by venous outflow than came into the muscle through arterial inflow. This may have been the case in the present study.

The trend of rapid recovery of blood volume at the completion of exercise and subsequent overshoot of blood volume past resting values observed in the present study mirrored that reported in occlusion (Hampson & Piantadosi, 1988; Sahlin, 1992; Homma, Eda, Ogasawara, & Kagaya, 1996; Mancini et al., 1994) and in exercise studies (Tamaki et al., 1994; Chance, Dait, Zhang, Hamaoka, & Hagerman, 1992). This increase in blood volume following exercise has been attributed to increased blood flow resulting from reactive hyperemia (Belardinelli, Barstow, Porszasz, & Wasserman, 1995; Hampson & Piantadosi, 1988).

No other studies have compared inter-set differences in blood volume for resistance exercise protocols. Further, no studies have compared trained and untrained subject groups in different exercise protocols with respect to blood volume. No significant differences in blood volume were found between sets in either the 10RM or 4RM exercise protocols. Further, no significant differences in blood volume were found between trained and untrained subjects in a set-wise comparison for both the 10RM and 4RM exercise protocols. Both training groups produced similar changes in muscle blood volume over the course of each exercise set in both the 10RM and 4RM training sessions (Table 2; Figure 2,3). As a result, the blood volume changes observed in the present study may be the result of intense weight lifting and appear independent of training status and magnitude of the load.

Muscle Oxygenation

It has been established that non-resistance type exercise protocols elicit decreases in muscle oxygenation. Using a cycle ergometer, Belardinelli, Barstow, Porszasz and Wasseman (1995a) found that Hb/Mb desaturation in the vastus lateralis muscle reached maximum levels as the subjects approached VO2max. In a similar study (Belardinelli et al., 1995b) Hb/Mb saturation of the vastus lateralis decreased over the course of incremental cycle exercise at work rates greater than lactate threshold. Jensen-Urstad, Hallback, and Sahlin (1995) investigated the effects of hypoxia on muscle oxygenation during arm crank exercise. They reported similar levels of Hb/Mb desaturation in both normoxic and hypoxic subjects during the exercise session. Similarly, Bhambhani, Maikala, and Buckley (1998) investigated the effects of both incremental arm crank and incremental leg cycling exercise on muscle oxygenation in men and women. They reported greater deoxygenation with increased exercise intensity until exercise intensity approached VO2max at which point tissue deoxygenation leveled off despite increased power output. Further, Chance, Dait, Zhang, Hamaoka, and Hagerman (1992) found decreases in tissue oxygenation with increasing power output levels for elite male and female rowers in both cycle and rowing ergometer tests.

In the present study both the 4RM and 10RM exercise protocols elicited decreases in tissue oxygenation during exercise followed by a rapid re-oxygenation at the cessation of exercise. This occurred for both trained and untrained subjects. These findings support the trend in exercise deoxygenation reported during non-resistance type exercise and are in agreement with the few studies that have examined muscle oxygenation changes during resistance type exercise. Mancini et al. (1994) investigated the effect of 30 repetitions, performed every 4 s, of incrementally loaded forearm flexion on muscle oxygenation in four normal subjects. They reported forearm desaturation ranging from 70%-83% of the physiologic range. Kahn et al. (1998) examined the effect of isometric contractions of varying percentages of MVC on forearm flexor oxygenation. They reported varying degrees of brachioradialis muscle deoxygenation depending on exercise intensity. Greatest desaturation was observed at 50% MVC while lowest desaturation occurred at 40% MVC. Tamaki, Uchiyama, Tamura, and Nakano (1994) examined Hb/Mb oxygenation in actively contracting muscle. They employed an arm-curl exercise protocol in which 3 successive sets of 10RM exercise, with interspersed rest intervals, were performed. Like the present study, they observed a decrease in tissue oxygenation during exercise and a rapid re-oxygenation at the cessation of the exercise.

In the present study, desaturation during each set of exercise ranged from approximately 50% to 92% (Table 3). These findings are similar to those reported in the literature. Mancini et al (1994) reported forearm desaturation values ranging from 70% to

83% during 30 contractions of increasing intensity over a 2 min exercise period. Kahn et al. (1998) found brachioradialis muscle desaturation values ranging from 20% to 60% muscle deoxygenation. It should be noted that the lower desaturation values were produced by lower loads than used in the present study while the higher desaturation values similar to those obtained in the present study were produced by similar loads.

The cause behind the observed resistance exercise-induced hypoxia or exercise induced-ischemia is unclear. Tamaki et al. (1994) suggested that the phenomenon of muscle tissue deoxygenation is brought about by restricted arm blood flow, specifically restricted venous outflow, causing a blood or oxygen debt. This blood debt or lack of oxygen supply is increased with repeated high-intensity contractions. Consequently, the working tissue further depletes an already decreased oxygen supply with each successive contraction in each exercise set. Kahn et al. (1998) suggest that deoxygenation of tissue during isometric forearm flexion at exercise intensities with high relative forces like those used in the present study resulted from a tourniquet effect (ischemia due to high muscle pressure) coupled with the oxygen consuming activity of exercising muscle fibers. The findings of the present study partially support these results. No significant differences in muscle oxygenation were found within sets for trained and untrained groups in either the 10RM or 4RM exercise protocols. The 3min rest interval between sets may have allowed for sufficient ATP replenishment and re-synthesis to meet the energy demands of the subsequent set of exercise without any oxygen debt carried over from the previous set. Consequently, no difference in muscle oxygenation between sets would occur. As well, no significant differences were found in a set-wise comparison between groups in the 10RM and 4RM protocols (Table 3; Figure 5,6). Because of the intensity of exercise involved in the present study it can be assumed that exercise-induced ischemia occurred to some extent. While aerobic glycolysis accounts for some of the ATP utilized during short duration high-intensity exercise, the primary source of muscle ATP for exercise lasting less than 1 min is provided by anaerobic-alactic and anaerobic-lactic sources (Fox, Bowers, & Foss, 1993). However, it is unlikely that oxygen used as a fuel for muscle work was a large contributor to the deoxygenation observed considering each set in the 4RM and 10RM protocols which lasted for 12s and 30s respectively. Because of the lack of differences observed between exercise protocols and groups, it is more likely that tissue deoxygenation was brought about by the decremental decrease in blood volume observed in each set of exercise. With a decreasing blood volume during each repetition of exercise, less oxygenated blood was available to the muscle during each contraction. Consequently, the NIRS probe registered this as increasing tissue deoxygenation. Coupled with the oxygen consuming activity of the muscle during exercise, overall muscle deoxygenation increased over the course of each set of exercise.

Although no statistically significant differences existed in muscle oxygenation for either exercise protocol, the untrained group demonstrated a 20% greater tissue deoxygenation than the trained group in the 4RM protocol (Table 3). This difference may be attributable to variations in motor unit recruitment between trained and untrained subjects. With training the muscle becomes more efficient. The synchronous firing of motor units brought about by training allows the muscle to generate the same amount of force with fewer activated fibers (Sale, 1992). Further, training increases the firing frequency of each motor unit. An increase in firing frequency increases the force output of the motor unit. Training also allows high threshold, highly glycolytic fast twitch muscle fibers (type IIb) to be activated (Sale, 1992). Untrained populations do not benefit from training induced synchrony of motor unit recruitment, elevated firing frequency, and may not be able to recruit high threshold motor units. Therefore they cannot fully activate the agonist muscle (Sale, 1992). In order to move the load imposed by the 4RM protocol, the untrained subject group would need to use more of the motor units within the muscle to overcome the load without the benefit of the highly glycolytic type IIb fibers. As a result, a more oxidative pool of motor units would be utilized to lift the weight than that used by the trained subject group. The trained group could obtain more force from each motor unit (increased firing frequency), simultaneously activate pools of motor units simultaneously (synchrony of recruitment allowing fewer motor units to be used to lift the 4RM weight), and activate high force, highly glycolytic fibers. Consequently, the greater number and more oxidative profile of motor units recruited by the untrained group during the 4RM protocol may have led to a greater oxygen-use during contraction in relation to the trained group who used fewer and more glycolytic motor units. This could account for the 20% difference in deoxygenation observed.

Fiber Type Shift

Alterations in the distribution of oxidative and glycolytic fiber types in human skeletal muscle have been observed in endurance training and high intensity intermittent training (Jansson, Sjodin, & Tesch, 1978; Simoneau, Lortie, Boulay, Marcotte, Thibault, & Bouchard, 1985; Jansson & Kaiser, 1977; Andersen & Henriksson, 1977). Increases in both the percentage distribution of ST or type I muscle fibers and/or type IIa muscle fibers (with a concomitant decrease in type IIb fibers) have been observed. It has been suggested that the redistribution of fiber types or fiber conversion occurred to increase the oxidative capacity of the muscle (Staron, Malicky, Falkel, Hagerman, & Dudley, 1989). Fiber type conversions have also been observed in strength training studies. In a 20 week lower body resistance training program where subjects trained twice a week using 3 sets of 6-8RM exercise, a decrease in type IIb fiber percentage with a concomitant increase in type IIa percentage was observed (Staron et al., 1989). Others have reported similar decreases in type IIb fiber percentage with accompanying increase in type IIa percentage over the course of long term resistance exercise protocols involving multiple sets and reps (Staron et al.,1991; Staron et al., 1994;Adams, Hather, Baldwin, & Dudley, 1993; Kraemer et al., 1995). However, all resistance-trained subjects do not demonstrate fiber type shift. Body builders and endurance athletes tend to have a higher percentage of type IIa fibers than do strength/power athletes. (Tesch & Larsson, 1982; Klitgaard, Zhou, & Richter, 1990; Tesch & Karlsson, 1985; Tesch, Thorsson, & Kaiser, 1984). Tamaki et al. (1994) reported that a multiple set 10RM resistance exercise protocol, similar to that used in bodybuilding type training, elicited rapid increases in deoxygenated blood similar to application of a tourniquet. They suggested that bodybuilding training, in which a moderately high load, a high number of repetitions, and multiple exercises are used, causes long-term exposure of the muscles to a lack of oxygen supply. Further, they conclude that the relative lack of oxygen supply to muscles may be a cause of fiber type redistribution observed in bodybuilders. However, strength and power athletes use training regimens emphasizing high loads, low repetitions, and longer rest intervals between sets. The present study is the only study to date that investigates the effect of this type of training on muscle oxygenation and blood volume. The high load, low repetition 4RM resistance protocol yielded similar blood volume and deoxygenation values to the high repetition, moderate load 10RM protocol (Table 1,2). Therefore, it would appear that strength/power athletes may be exposed to similar levels of deoxygenation as body-builders. However, although the present study investigated load and repetition parameters similar to those used in strength/power training regimens, the exercise selected (arm curl) and rest interval used (3min) are not typical of those in strength/power training. Strength and power athletes typically use multi-joint exercises such as cleans, squats, bench press, jerk, and the snatch. The arm-curl exercise used is a single joint exercise. As well, rest intervals in strength and power training generally exceed 3 min and are often greater than 4 min. The lack of inclusion of these other parameters may be the cause of the similarity observed between the 10RM and 4RM exercise protocols in the present study.

Tesch, Thorsson and Kaiser (1984) reported no difference in fiber type distribution in the quadriceps of power-lifters and non-athletes. Others have reported bodybuilders possess a higher percentage of type IIa fibers than sedentary men (Klitgaard, Zhou, & Richter, 1990) and that resistance training according to bodybuilder-like parameters can alter fiber-type distribution (Staron, Malicky, Leonardi, Falkel, Hagerman, & Dudley, 1989). Therefore, it was anticipated that the trained subjects in the present study would have experienced some form of fiber conversion consequent to training where the untrained group, not being resistance trained, would not. As a result of the training induced fiber conversion, the trained group would be able to use more oxygen during both the 10RM and 4RM lifting protocols and therefore exhibit a greater level of deoxygenation than the untrained group. However, no differences were found in muscle deoxygenation between trained and untrained groups in either exercise protocol.

Muscle biopsies were not taken in the present study. Therefore fiber distribution of the subjects was not established. The results suggest there may not have been a fiber-type distribution difference between the trained and untrained subjects. This could account for the lack of exercise-induced muscle deoxygenation difference observed between the two groups

Summary

Both the 4RM and 10RM resistance protocols elicited decreases in muscle oxygenation and blood volume in both the trained and untrained groups. There was no set-wise difference for the 10RM or 4RM protocol for either the trained or untrained groups for both deoxygenation and blood volume. No difference existed set-wise between trained and untrained groups in the 4RM or 10RM protocols. Further, no differences existed between 10RM and 4RM deoxygenation and blood volume for both the trained and untrained groups. Therefore, it was concluded that 4RM and 10RM resistance training protocols elicit similar decreases in muscle oxygenation and blood volume in both trained and untrained males and is consistent between sets.

Directions for Future Research

Although this study has given some insight into muscle oxygenation and blood volume trends during different resistance training protocols there are many areas that could be explored. Future research should concentrate on combining muscle oxygenation and blood volume measures with histochemical analyses over the course of full training sessions and long term training periods. Further, comparisons of the effects of different loading parameters on blood volume and muscle oxygenation should be investigated. What is the effect of different rest intervals between sets? What is the effect of different loads? What are the effects of different numbers of repetitions? What is the effect of going to failure on each set? What effect does unloading between repetitions have on muscle oxygenation? Are there differences between multi-joint and single joint lifts? What is the effect of different amounts of time that tension is placed on the muscle? Are there differences between males and females? Does level of aerobic fitness affect tissue deoxygenation during weight training? There are also many questions regarding the amount of oxygen used by the muscle during weight lifting under a number of parameters. Future research should also attempt to quantify oxygen use, blood volume and blood flow during weight lifting.

REFERENCES

Adams, G.R., Hather, B.M., Baldwin, K.M., & Dudley, G.A. (1993). Skeletal muscle myosin heavy chain composition and resistance training. Journal of Applied Physiology, 74(2), 911-915.

Andersen, P. & Henriksson, J. (1977). Training induced changes in subgroups of human type II skeletal muscle fibers. Acta Physiologica Scandinavica, 99, 123-125.

Baechle, T.R.(1994). Essentials of strength training and conditioning.Champaign, Il: Human Kinetics.

Belardinelli,R.,Barstow, T.J., Porszasz, J. & Wasserman, K. (1995). Skeletal muscle oxygenation during constant work rate exercise. Medicine and Science in Sports and Exercise,27(4), 512-519.

Belardinelli,R.,Barstow, T.J., Porszasz, J. & Wasserman, K. (1995). Changes in skeletal muscle oxygenation during incremental exercise measured with near infrared spectroscopy. European Journal of Applied Physiology, 70, 487-492.

Bhambhani, Y., Maikala, R., & Buckley, S. (1998). Muscle oxygenation during incremental arm and leg exercise in men and women. European Journal of Applied Physiology, 78, 422-431.

Chance, B., Dait, M.T., Zhang, C., Hamaoka, T. & Hagerman, F. (1992). Recovery from exercise –induced desaturation in the quadriceps muscles of elite competitive rowers. American Journal of Physiology, 262, C766-C775.

Fox, E., Bowers, R., & Foss, M. (1993). The Physiological Basis for Exercise and Sport.(5th ed.). Madison, WI: Brown & Benchmark.

Garhammer, J. & Takano, B. (1992). Training for weightlifting. In P.V. Komi (ed.), Strength and Power in Sport (pp.357-369). Oxford: Blackwell Scientific.

Goldspink, G. (1992). Cellular and molecular aspects of muscle adaptations in skeletal muscle. In P.V. Komi (ed.), Strength and Power in Sport (pp.211-229). Oxford: Blackwell Scientific.

Hampson, N.B. & Piantadosi, C.A. (1988). Near infrared monitoring of human skeletal muscle oxygenation during forearm ischemia. Journal of Applied Physiology, 64(6), 2449-2457.

Homma, S., Eda, H., Ogasawara, S., & Kagaya, A. (1996). Near-infrared estimation of O2 supply and consumption in forearm muscles working at varying intensity. Journal of Applied Physiology, 80(4), 1279-1284.

Hulten, B., Thorstensson, A., Sjodin, B., & Karlsson, J. (1975). Relationship between isometric endurance and fibre types in humans. Acta Physiologica Scandinavica, 93, 135-138.

Jansson, E. & Kaijser, L. (1977). Muscle adaptation to extreme endurance training in man. Acta Physiologica Scandinavica, 100, 315-324.

Jansson, E., Sjodin, B., & Tesch, P. (1978). Changes in muscle fibre type distribution in man after physical training. Acta Physiologica Scandinavica, 104, 235-237.

Jensen-Urstad, M., Hallback, I. , & Sahlin, K. (1995). Effect of hypoxia on muscle oxygenation and metabolism during arm exercise in humans. Clinical Physiology,15, 27-37.

Kahn, J.F., Jouanin, J.C., Bussiere, J.L., Tinet, E., Avrillier, S., Ollivier, J.P., & Monod, H. (1998). The isometric force that induces maximal surface muscle deoxygenation. European Journal of Applied Physiology, 78, 183-187.

Klitgaard, H., Zhou, M., & Richter, E.A. (1990). Myosin heavy chain composition of single fibers from m. biceps brachii of male body builders. Acta Physiologica Scandinavica, 140, 175-180.

Kraemer, W.J., Patton, J., Gordon, S.E., Harman, E.A., Deschenes, M.R., Reynolds, K., Newton, R.U., Triplett, N.T., & Dziados, J.E. (1995). Compatability of high intensity strength and endurance training on hormonal and skeletal muscle adaptations. Journal of Applied Physiology, 78(3), 976-989.

Lind, A.R & McNicol, G.W. (1967). Circulatory responses to sustained contractions and the effect of free or restricted arterial inflow on post-exercise hyperemia. Journal of Physiology, 192, 529-547.

Lind, A.R. & Williams, C.A. (1979). The control of blood flow through human forearm muscles following brief isometric contractions. Journal of Physiology, 288, 529-547.

Mancini, D.M., Bolinger, L., Li, H., Kendrick, K., Chance, B., & Wilson, J.R. (1994). Validation of near-infrared spectroscopy in humans. Journal of Applied Physiology, 77(6), 2740-2747.

.Moritani, T. (1992) Time course of adaptations during strength and power training. In P.V. Komi (ed.), Strength and Power in Sport (pp.266-278). Oxford: Blackwell Scientific.

Sahlin, K. (1992). Non-invasive measurements of O2 availability in human skeletal muscle with near-infared spectroscopy. International Journal of Sports Medicine, 13(suppl. 1), S157-S160.

Sale, D.G. (1988). Neural adaptations to resistance training. Medicine and Science in Sports and Exercise, 20, S135-S145.

Sale,D.G. (1992). Neural adaptation to resistance training. In P.V. Komi (ed.), Strength and Power in Sport (pp.249-265). Oxford: Blackwell Scientific.

Simoneau, J.A., Lorite, G., Boulay,M.R., Marcotte, M., Thibault, M.-C., & Bouchard,C. (1985). Human skeletal muscle fiber type alteration with high-intensity intermittent training. European Journal of Applied Physiology, 54, 250-253.

Staron, R.S., Malicky, E.S., Leonardi, M.J., Falkel, J.E., Hagerman, F.C., & Dudley, G.A. (1989). Muscle hypertrophy and fast fiber type conversions in heavy resistance-trained women. European Journal of Applied Physiology, 60, 71-79.

Staron, R.S., Leonardi, M.J., Karapondo, D.L., Malicky, E.S., Falkel, J.E., Hagerman, F.C., & Hikida, R.S. (1991). Strength and skeletal muscle adaptations in heavy resistance-trained women after detraining and retraining. Journal of Applied Physiology, 70(2), 631-640.

Staron, R.S., Leonardi, M.J., Karapondo, D.L., Malicky, E.S., Falkel, J.E., Hagerman, F.C., & Hikida, R.S. (1994). Skeletal muscle adaptations during early phase heavy resistance-training in men and women. Journal of Applied Physiology, 76(3), 1247-1255.

Tamaki, T., Uchiyama, S., Tamura, T., & Nakano, S. (1994). Changes in muscle oxygenation during weight-lifting exercise. European Journal of Applied Physiology, 68, 465-469.

Tamura, T., Eda, H., Takada, M., & Kubodera, T. (1988). New instrument for monitoring hemoglobin oxygenation. Advances in Experimental Medicine and Biology, 103-107.

Tesch, P.A. (1992). Training for bodybuilding. In P.V. Komi (ed.), Strength and Power in Sport (pp.371-380). Oxford: Blackwell Scientific.

Tesch, P.A. & Karlsson, J. (1985). Muscle fiber types and size in trained and untrained muscles of elite athletes. Journal of Applied Physiology, 59(6),1716-1720

Tesch, P.A. & Larsson, L. (1982). Muscle hypertrophy in bodybuilders. European Journal of Applied Physiology, 49, 301-306.

Tesch, P.A., Thorsson, A., & Kaiser, P. (1984). Muscle capillary supply and fiber type characteristics in weight and power lifters. Journal of Applied Physiology, 56(1), 35-38.

Vankan, W.J., Huyghe, J.M., van Donkelaar, C.C., Drost, M.R., Janssen, J.D., & Huson, A. (1998). Mechanical blood-tissue interaction in contracting muscles: a model study. Journal of Biomechanics, 31, 401-409.

Wilson, J.R., Mancini, D.M., McCully, K., Ferraro, N., Lanoce, V., & Chance, B. (1989). Noninvasive detection of skeletal muscle under perfusion with near-infrared spectroscopy in patients with heart failure. Circulation, 80, 1668-1674.

Appendix A: Review of Literature

Introduction

Strength, defined as the peak force or torque a muscle or muscle group can generate during a maximal voluntary contraction (Goldspink, 1992), is an important component of sport today. As athletes strive to compete in the "bigger, stronger, faster" world of modern sport, their strength, and therefore ability to generate force, is an important consideration in performance and injury prevention. An athlete's ability to generate force depends on the size of the involved muscles and the ability of the nervous system to activate these muscles (Sale, 1992). Increases in strength can be realized by stimulation of the neuromuscular system through resistance training exercise. Adaptations to resistance exercise resulting in increases in strength occur through neural and/or muscular mechanisms (Sale, 1988; Moritani, 1992).

Neural mechanisms thought to be responsible for increases in strength include a) increased recruitment of motor units, b) increased firing frequency of motor units, c) synchronization of contracting motor units, and d) decrease in inhibition by muscular protective mechanisms (Sale, 1988; Sale, 1992; Kraemer Fleck & Evans, 1996). The motor unit is the functional unit of the neuromuscular system and consists of the motor neuron and the muscle fibers it innervates (Kraemer et al., 1996). The magnitude of force a muscle can generate is dependent on the number of motor units activated (Sale, 1988). Recruitment of motor units occurs according to recruitment threshold (Kraemer et al., 1996). Motor units with low twitch force and low thresholds (slow twitch or Type I motor units) are recruited at low force levels and before high twitch force/high threshold motor units (fast twitch or type II motor units). The latter are required for maximal force production (Sale, 1992; Kraemer et al., 1996). This orderly recruitment pattern is referred to as the "size principle" (Sale, 1992). It has been suggested that untrained individuals are incapable of activating or recruiting the highest threshold motor units, thereby limiting their ability to generate force (Sale, 1992). One of the adaptations to resistance exercise is development of the ability to recruit all motor units, therefore attaining the ability for maximal force production.

Increases in firing frequency of motor units results in the increased ability of motor units to generate force. Firing frequency is the number of impulses from the motor neuron of a motor unit received by muscle fibers (Sale, 1992). Type II motor units require high firing rates to generate maximal force. Therefore, by increasing the discharge of these high force/high threshold motor units through resistance training, the force-generating capability of the muscle is increased (Kraemer *et al.*, 1996).

Sale (1988) describes synchronization as the simultaneous firing of motor units in contracting muscle. As all motor units fire at once, activation of motor units is maximized, resulting in a greater rate of force development and possibly a greater peak force (Sale, 1992).

Because the central nervous system (CNS) is capable of limiting force through protective inhibitory mechanisms, removal of these through training can increase force production (Kraemer et al., 1996). The Golgi tendon organ, located in the musculotendonous junction, is considered to adapt during resistance exercise. It sends inhibitory impulses, during the production of high tension, to the CNS preventing maximal motor unit recruitment and firing rates thereby limiting force production (Kandel, Schwartz, & Jessel, 1991; Wilson, 1995). Exposure of the Golgi tendon organ to high tension during resistance exercise may dampen its inhibitory effects resulting in an increased potential for force development (Wilson, 1995).

Muscular mechanisms that increase the force generating capability of muscle include an increase in muscle fiber size (increased muscle cross-sectional area) (MacDougall, 1997) and increases in connective tissue (Stone, 1992). These both fall under the category of muscle hypertrophy or an increase in muscle size. Increased muscle fiber size occurs due to an increase in the myofibrillar content of each muscle fiber (Goldspink, 1992). Through the addition of myofibrils, the number of actin-myosin cross-bridges that can be formed in the muscle increases. Because each cross-bridge is an independent force generator and the magnitude of force generated in the muscle depends on the number of cross-bridges formed (Goldspink, 1992; Huijing, 1992), and increase in myofibrillar cross-sectional area increases the potential for force generation (Goldspink, 1992). Therefore, increases in myofibrillar content consequent to resistance exercise increases the force generating capability of the muscle. Further, connective tissue, which participates in force transmission from muscle to bone, increases in response to resistance training. As a result, greater tension can be transferred from the muscle to the bone, increasing the force production potential of the muscle (Jones & Rutherford, 1987; Stone, 1992).

The application of resistance training programs is varied. Different sports employ variations in the exercises and training parameters of, volume, rest interval and load. As a result, a variety of adaptations are observed corresponding to different training protocols. One adaptation observed in many studies is a fiber type shift from highly glycolytic type IIb fibers to more oxidative type IIa fibers (Tesch, 1992). The cause of this shift is unknown, however Tamaki, Uchiyama, Tamura and Nakano (1994) suggest the shift may be the product of long-term muscle anoxia resulting from training. Only one study to date (Tamaki et al., 1994) has monitored tissue oxygenation during resistance exercise. Tissue oxygenation changes can be monitored non-invasively in humans through the use of Near Infared Spectroscopy (NIRS).

Fiber Type Shift

Human skeletal muscle has two primary fiber types, type I and type II. Type I fibers, also known as slow twitch muscle fibers, possess a higher oxidative capacity and lower contraction velocity than type II or fast twitch fibers (Billeter & Hoppeler, 1992). Conversely, type II fibers possess low oxidative capacity and high contraction velocity. Type I fibers and, consequently, type I motor units, are more resistant to fatigue than type II motor units. Within each main fiber types are subtypes. The major fiber subtypes of human muscle include type I, type IIa and type IIb fibers (Staron, Leonardi, Karapondo, Malicky, Falkel, Hagerman, & Hikida, 1991). Other minor fiber types include type Ic, type IIc and type IIab. The continuum of human fiber types progresses from type I, the most oxidative, to type IIb, the least oxidative fibers (I>Ic>IIc>IIac>IIa>IIab>IIb) (Staron et al., 1991). Classification of fiber types is primarily based on myosin heavy chain and myosin ATPase histochemical analysis (Kraemer, Fleck, & Evans, 1996) although biochemical techniques are being used more frequently.

Alterations in the distribution of fiber types in human skeletal muscle have been observed following exercise. Endurance training and high intensity intermittent training have resulted in increases in the percentage distribution of ST or type I muscle fibers

(Jansson, Sjodin, & Tesch, 1978; Simoneau, Lortie, Boulay, Marcotte, Thibault, & Bouchard, 1985). Further, Jansson, and Kaiser (1977) and Andersen and Henriksson (1977) showed endurance training caused a decrease in percentage of type IIb fibers and an increase in the percentage of type IIa fibers suggesting a shift form type IIb to IIa. Staron, Malicky, Leornardi, Falkel, Hagerman, and Dudley (1989) suggested this fiber conversion occured to increase the oxidative capacity of the muscle because IIa fibers are more oxidative than IIb fibers.

Several strength training studies have also shown a conversion of IIb to IIa fibers over the course of training. In a study by Staron et al. (1989) a heavy lower body resistance training program was performed two times per week for 20 weeks in women. Subjects performed two sub-maximal warm-up sets (40-60% of 1 RM) followed by three sets of 6-8 RM for four lower body exercises each training session. The training resulted in significant hypertrophy in all three fiber types (I, IIa, IIb) and a decrease in mean type IIb fiber percentage with a concomitant increase in mean type IIa percentage. This suggests a conversion of IIb to IIa fibers occurred over the course of training. In a follow up study, Staron et al. (1991) had the same population detrain for 30 to 32 weeks then retrain for either 6 or 13 weeks. In the course of detraining, an increased percentage of IIb and decreases percentage of IIa fibers was seen toward pre-training values. After the 6 week retraining period conversion of IIb to IIa fibers was seen. In the group completing 13 weeks of retraining no type IIb fibers were found and only a few IIab fibers could be identified. This suggests long-term resistance exercise may result in a major shift from IIb to IIa fibers. Further, Staron et al. (1994) found similar results in an 8 week high intensity weight training study involving men and women. Subjects trained twice a week using 5 sets (2 warm-up sets, 3 sets to failure) of 6-8 RM one session and 10-12 RM the following session for three quadriceps exercises. Over the duration of the program, a 7% decrease in type IIb fibers was observed. Significant decreases in histochemically assessed type IIb fibers were observed after two weeks of training in the women and four weeks of training in the men. These trends corresponded to a concomitant increase in type IIa fibers. Similarly, Adams, Hather, Baldwin, and Dudley (1993) observed a decrease in myosin heavy chain (MHC) IIb content of 12% and an increase of MHC IIa content of 12 % in men performing multiple sets (4-5) of high repetitions (6-12) of concentric and eccentric resistance exercise over 19 weeks. As well, Kraemer et al. (1995) saw a decrease in percent IIb fibers (17.3%) following three months of heavy resistance exercise consisting of two heavy (5RM) and two moderate (10RM) training sessions per week. All of these studies show a lower percentage of IIb fibers and a higher percentage of IIa fibers after resistance exercise. This suggests a IIb to IIa fiber conversion.

Although a fiber shift can occur in resistance training, not all resistance trained subjects exhibit lower percentages of type IIb fibers as a result of training. Tesch and Larsson (1982) observed a lower percentage of type II fibers (50%) in high caliber body builders when compared to competitive power/weight lifters (69%) in the deltoid and quadriceps muscles. In a study comparing bodybuilders with sedentary men, Klitgaard, Zhou and Richter (1990) found bodybuilders had a higher percentage of fibers with MHC IIa and a lower percentage of fibers expressing both MHC IIa and IIb. Further, the bodybuilders possessed practically no fibers containing only MHC IIb. Only the bodybuilders demonstrated a trend toward a lower proportion of IIb fibers (18% vs. 26%

in sedentary) and higher proprotion of type IIa fibers (31% vs. 25% in sedentary). In a study involving power-lifters, endurance athletes and non-athletes, Tesch, Thorsson, and Kaiser (1984) found no difference in fiber distribution in the quadriceps between the power-lifters (59% FT) and non-athletes (61%) but a difference between power-lifters and endurance athletes (40% FT). However, the mean fiber area in the power-lifters was 72% larger than in the non-athletes and was accounted for by specific quadriceps hypertrophy. Tesch and Karlsson (1985) and Tesch et al. (1984) also reported high percentages of FT fibers in power-lifters.

It appears that a fiber conversion may occur in bodybuilders and not power-lifters. This may be the result of the type of training used by each athlete. Power-lifters' routines are variations of a common theme. Typical exercises include the snatch, front squat, dead-lift, power, clean, and clean and jerk (Garhammer & Takano, 1992). Training includes approximately 5-12 sessions per week, 3-5 exercises per session, with loads between 1 and 5 RM. Rest intervals between sets are approximately four to five minutes (Garhammer and Takano, 1992). Bodybuilding routines are highly varied in loading, volume, frequency and rest. Generally programs include multiple exercises per muscle group, multiple sets per exercise (3-5), high repetitions (6-12) and short rest intervals between sets (1-2 minutes) (Tesch, 1992). Each exercise is typically performed to muscle failure. All previously discussed studies in which fiber type conversions were observed employed bodybuilder-like loading parameters.

The cause of the IIb to IIa fiber conversion is unclear. Lind and Williams (1979) show a decrease in muscle blood flow in the forearm at contractions of 60% and 100% MVC. Further, Hulten, Thorstensson, Sjodin, and Karlsson (1975) suggest that blood flow at tensions greater than 50% MVC is severely restricted allowing little or no oxygen delivery to the tissues. If this holds true, the loads used by power-lifters and bodybuilders are large enough to severely restrict blood flow and may impair tissue oxygenation. Tamaki et al. (1994) found that multiple set, 10 RM arm exercise protocol resulted in a rapid decrease in oxy-Hb /Mb saturation and an increase in deoxy Hb/Mb. Further, total Hb/Mb levels increased during exercise and did not return to resting level between sets. The blood volume changes and lack of oxygen supply was interpreted as muscle anoxia accelerated by short rest intervals. They suggest that the high number of repetitions, multiple sets and exercises, and short rest periods common in bodybuilder training may expose the exercising muscle to insufficient oxygen supply for extended periods of time. They further state that this lack of oxygen supply may be a cause of the conversion of IIb to IIa fibers seen with resistance exercise and especially among bodybuilders.

No studies to date have examined muscle oxygenation changes during power-lifter type training involving high load, low repetition, long rest protocols. A non-invasive method of measuring tissue oxygenation changes, more specifically Hb/Mb saturation characteristics, is near infrared spectroscopy (NIRS).

Near Infrared Spectroscopy (NIRS)

Near infrared spectroscopy (NIRS) is a non-invasive method that can be used to measure relative changes in tissue oxygenation in humans. The NIRS method relies on the ability of NIR light (700-1000nm) to pass through biological tissues (Hampson & Piantadosi, 1988). Light emitted by the NIR probe is primarily scattered and absorbed as it passes through body tissues except for a portion of the original incident light. This light is reflected and picked up by the photo detectors of the probe (Belardinelli, Barstow, Porszasz, & Wasserman, 1995). NIRS use in monitoring of tissue oxygenation is based on the difference in the absorption spectra between the oxygenated and deoxygenated forms of hemoglobin (Hb) and myoglobin (Mb), the oxygen carrying molecules in blood and muscle (Sahlin, 1992). The iron-porphyrin complexes of the oxygenated and deoxygenated forms of Hb and Mb absorb NIR light. Because of differences in the absorption spectra of the iron-porphyrin complexes, tissue illumination at different wavelengths of light yields varying absorption/reflection patterns proportional to deoxyhemoglobin/myoglobin (Hb/Mb) and oxyhemoglobin/myoglobin (HbO2/MbO2) relative concentrations (Hampson & Piantadosi, 1988; Belardenelli, Barstow, Parszasz, & Wasserman, 1995). Because the absorption spectra for Hb/Mb and HbO_2/MbO_2 are similar, the contribution of each to the NIRS signal cannot be separated. As a result, oxygenated and deoxygenated forms of Hb/Mb are summed when monitoring, skeletal muscle desaturation (Hampson & Piantadosi, 1988). There is debate as to the contribution Mb makes to the de-saturation signal. However, because of the absorbance spectra similarity to Hb, many studies include Mb in their studies (Belardinelli et al., 1995; Belardinelli, Barstow, Porszasz, & Wasserman, 1995; Wilson, Mancini, McCully, Ferraro, Lanoce, & Chance, 1989; Sahlin, 1992; Chance, Dait, Zhang, Hamakoa, & Hagerman, 1992). Wilson et al. (1989) suggest that myoglobin desaturation does not occur until a PO2 of below 20 mmHg is experienced intracellularly. Further, in the same experiment, when Mb was inactivated with ethyl hydrogen peroxide (thereby removing the Mb contribution to the signal) the absorption did not change substantially with exercise, indicating deoxygenation of Hb was the main contributor to the NIR signal. As well, Mancini et al. (1994) discovered, when combining NIRS with nuclear magnetic spectroscopy (allowing for in vivo Mb monitoring), that a high degree of Hb deoxygenation occurred without Mb deoxygenation in exercise. They concluded that the major NIR signal was the result of Hb. Despite these findings neither group exclude Mb as a contributor to the NIR signal. Instead, they suggest it is a minor contributor to the NIR signal.

The number and value of wavelengths used to study deoxygenation of Hb/Mb varies amongst studies. Typically two wavelengths are used on either side of the HbO_2/MbO_2 and Hb/Mb isosbestic point (Mancini, et al., 1994). Most studies involved in human monitoring use values of 760 nm and 800nm or 850 nm (Chance et al, 1992; Belardinelli et al., 1995; Wilson et al., 1989;Sahlin, 1992; Mancini et al., 1994). Others use wavelengths between 700 and 900nm (Homma, Eda, Ogasawara, & Kagaya, 1996; Tamaki et al., 1994; Hampson & Piantadosi, 1988). Regardless of the number or value of wavelength, changes in oxygenation of Hb/Mb are determined according to the same principles. Using the most frequent values as an example, at 760 nm, light absorption is due primarily to Hb/Mb. At 850 nm, the absorption is proportional to the total amount of Hb/Mb (as oxy and deoxy forms of Hb/Mb exhibit similar absorption patterns) (Wilson, 1989). Trends in deoxygenation of Hb/Mb are measured through changes in the difference between the two wavelengths (changes in 760-850nm). Changes in the sum of light intensity (changes in 760 +850 nm) give a relative measure of blood volume changes (Belardinelli et al., 1995; Chance et al., 1994; Sahlin, 1992). All values are converted to optical densities (OD) through Lambert-Beer's Law (Belardinelli et al., 1995; De Blasi, Quaglia, Gasparetto, & Ferrari, 1992). As a result values are interpreted as changes in OD (ΔOD).

NIRS use has been validated in humans in a series of experiments. Mancini et al. (1994) found a significant correlation ($r=0.83-0.97$) between venous saturation and absorption (760nm –800nm) changes at rest and during ischemic exercise. As well, they found skin blood flow changes, similar to those experienced in maximal exercise as body temperature increases, had no significant effect on changes in NIR absorption. Further, they discovered that decreased forearm blood flow, achieved through norepinephrine administration, resulted in greater deoxygenation of Hb/Mb while nitroprusside induced increased forearm blood flow at exercise whereas rest resulted in less Hb/Mb deoxygenation. As a result, changes in 760-800nm absorption parelled changes in forearm blood flow. Because of these findings, Mancini et al. (1994) suggest NIR provided localized tissue oxygenation data. Further, Chance et al. (1992) suggested the optical signal is derived primarily from the absorption of light in capillaries and small vessels. This is because large vessels almost completely absorb light (representing only a small fraction of the absorbance)(DeBlasi et al.,1992)) and NIR light penetration into the tissue is approximately 2-3 cm.

NIRS has been used successfully to monitor Hb/Mb oxygenation in a variety of applications in humans including tissue monitoring in ischemic conditions (Hampson & Piantadosi, 1988) in patients with arterial disease and heart failure (Wilson et al., 1989) and as a non-invasive instrument to monitor newborns (Tamura et al., 1988). More recently, NIRS has been used to evaluate muscle oxygentaion under a variety of exercise conditions. Belardinelli et al. (1995) found that Hb/Mb desaturation reached maximum levels as the subjects approached VO_2 max during incremental cycle exercise. They also discovered that the rate of tissue desaturation increased after lactate threshold. In a follow up study, Belardinelli et al., (1995) found, using NIRS, that changes in Hb/Mb oxygenation were dependent on exercise intensity. In an incremental cycling protocol, HbO_2/MbO_2 saturation remained constant or increased at workloads less than lactate threshold. Conversely, HbO_2/MbO_2 saturation declined throughout exercise at work rates greater than lactate threshold. Jensen-Urstad, Hallback, & Sahlin (1995) compared the effects of hypoxia on muscle oxygenation using NIRS. They discovered similar decreases in desaturation for normoxia and hypoxia subjects during arm crank exercise but found hypoxic subjects were slower to attain steady state values than normoxic exercisers. Further, Chance et al. (1992) used NIRS to measure the extent of deoxygenation and resaturation (recovery) time in the leg muscles of elite male and female rower following maximal efforts on cycle and rowing ergometers.

Despite many studies involving the use of NIRS to evaluate oxygenation changes of Hb/Mb during exercise, few have examined oxygenation changes during resistance type exercise. Homma et al. (1996) investigated post-exercise oxygenation changes in rhythmically contracting forearm muscles exercising at loads between 5% and 30% MVC. However, they did not monitor deoxygenation throughout the exercise bouts. Because blood flow is restored shortly after the cessation of exercise (Lind & Williams, 1979), any oxygenation changes during exercise may not be represented. Therefore, this data did not provide any information in regard to the changes in Hb/Mb oxygenation during exercise. In addition, the loads and exercise protocols that were used are not representative of more common resistance training practices. Tamaki et al. (1994) have conducted the only study to date that examined Hb/Mb oxygenation in contracting muscle during resistance-training exercise. They employed a ten repetition maximum (10RM) arm curl protocol and found an increase in Hb/Mb and a decrease in HbO_2/MbO_2 over the course of the exercise protocol. Further, they observed an increase in blood volume with the performance of multiple sets of exercise with short rest periods (manifested as an increase in total Hb/Mb).

Conclusions

Near infrared spectroscopy is a valid measure of the changes in oxygenation of Hb/Mb at both rest and during exercise (Mancini et al., 1994). This technique can also be used to measure oxygenation changes in muscle during resistance exercise (Tamaki et al., 1994). Despite this application, only one study to date has used NIRS technology to study oxygenation changes in muscle consequent to resistance exercise. Therefore, more research is needed to examine changes in Hb/Mb saturation with resistance exercise in humans under a variety of training conditions.

REFERENCES

Adams, G.R., Hather, B.M., Baldwin, K.M., & Dudley, G.A. (1993). Skeletal muscle myosin heavy chain composition and resistance training. Journal of Applied Physiology, 74(2), 911-915.

Andersen, P. & Henriksson, J. (1977). Training induced changes in subgroups of human type II skeletal muscle fibers. Acta Physiologica Scandinavica, 99, 123-125.

Belardinelli,R.,Barstow, T.J., Porszasz, J. & Wasserman, K. (1995). Skeletal muscle oxygenation during constant work rate exercise. Medicine and Science in Sports and Exercise,27(4), 512-519.

Belardinelli,R.,Barstow, T.J., Porszasz, J. & Wasserman, K. (1995). Changes in skeletal muscle oxygenation during incremental exercise measured with near infrared spectroscopy. European Journal of Applied Physiology, 70, 487-492.

Billeter, R. & Hoppeler, H. (1992). Muscular Basis of strength. In P.V. Komi (ed.), Strength and Power in Sport (pp.39-63). Oxford: Blackwell Scientific.

Chance, B., Dait, M.T., Zhang, C., Hamaoka, T. & Hagerman, F. (1992). Recovery from exercise –induced desaturation in the quadriceps muscles of elite competitive rowers. American Journal of Physiology, 262, C766-C775.

DeBlasi,R.A., Quaglia,E., Gasparetto, A., & Ferrari, M. (1992). Muscle oxygenation by fast near infared spectrophotometry (NIRS) in ischemic forearm. Advances in Experimental Medicine and Biology, 316, 163-172.

Garhammer, J. & Takano, B. (1992). Training for weightlifting. In P.V. Komi (ed.), Strength and Power in Sport (pp.357-369). Oxford: Blackwell Scientific.

Goldspink, G. (1992). Cellular and molecular aspects of muscle adaptations in skeletal muscle. In P.V. Komi (ed.), Strength and Power in Sport (pp.211- 229). Oxford: Blackwell Scientific.

Hampson, N.B. & Piantadosi, C.A. (1988). Near infrared monitoring of human skeletal muscle oxygenation during forearm ischemia. Journal of Applied Physiology, 64(6), 2449-2457.

Homma, S., Eda, H., Ogasawara, S., & Kagaya, A. (1996). Near-infrared estimation of O2 supply and consumption in forearm muscles working at varying intensity. Journal of Applied Physiology, 80(4), 1279-1284.

Huijing, P.A. (1992). Elastic potential of muscle. In P.V. Komi (ed.), Strength and Power in Sport (pp.151-168). Oxford: Blackwell Scientific.

Hulten, B., Thorstensson, A., Sjodin, B., & Karlsson, J. (1975). Relationship between isometric endurance and fibre types in humans. Acta Physiologica Scandinavica, 93, 135-138.

Kandel,E.R., Schwartz, J.H., & Jessell, T.M. (1991). Principles of Neural Science. Norwalk, CT: Appleton & Lange.

Jansson, E. & Kaijser, L. (1977). Muscle adaptation to extreme endurance training in man. Acta Physiologica Scandinavica, 100, 315-324.

Jansson, E., Sjodin, B., & Tesch, P. (1978). Changes in muscle fibre type distribution in man after physical training. Acta Physiologica Scandinavica, 104, 235-237.

Jensen-Urstad, M., Hallback, I. , & Sahlin, K. (1995). Effect of hypoxia on muscle oxygenation and metabolism during arm exercise in humans. Clinical Physiology,15, 27-37.

Jones, D.A. & Rutherford, O.M. (1987). Human muscle strength training: the effects of three different regimes and the nature of the resultant changes.Journal of Physiology, 391, 1-11.

Klitgaard, H., Zhou, M., & Richter, E.A. (1990). Myosin heavy chain composition of single fibers from m. biceps brachii of male body builders. Acta Physiologica Scandinavica, 140, 175-180.

Kraemer, W.J., Patton, J., Gordon, S.E., Harman, E.A., Deschenes, M.R., Reynolds, K., Newton, R.U., Triplett, N.T., & Dziados, J.E. (1995). Compatability of high intensity strength and endurance training on hormonal and skeletal muscle adaptations. Journal of Applied Physiology, 78(3), 976-989.

Kraemer, W.J., Fleck, S.J., & Evans, W.J. (1996). Strength and power training: physiological mechanisms of adaptation. In J.O.Holloszy (ed.), Exercise and Sport Sciences Reviews, 24, (pp.363-397). Baltimore: Williams & Wilkens.

Lind, A.R. & Williams, C.A. (1979). The control of blood flow through human forearm muscles following brief isometric contractions. Journal of Physiology, 288, 529-547.

MacDougall, J.D. (1992). Hypertrophy or hyperplasia? In P.V. Komi (ed.), Strength and Power in Sport (pp.230-237). Oxford: Blackwell Scientific.

Mancini, D.M., Bolinger, L., Li, H., Kendrick, K., Chance, B., & Wilson, J.R. (1994). Validation of near-infared spectroscopy in humans. Journal of Applied Physiology, 77(6), 2740-2747.

Moritani, T. (1992) Time course of adaptations during strength and power training. In P.V. Komi (ed.), Strength and Power in Sport (pp.266-278). Oxford: Blackwell Scientific.

Sahlin, K. (1992). Non-invasive measurements of O2 availability in human skeletal muscle with near-infrared spectroscopy. International Journal of Sports Medicine, 13(suppl. 1), S157-S160.

Sale, D.G. (1988). Neural adaptations to resistance training. Medicine and Science in Sports and Exercise, 20, S135-S145.

Sale,D.G. (1992). Neural adaptation to resistance training. In P.V. Komi (ed.), Strength and Power in Sport (pp.249-265). Oxford: Blackwell Scientific.

Simoneau, J.A., Lorite, G., Boulay,M.R., Marcotte, M., Thibault, M.-C., & Bouchard,C. (1985). Human skeletal muscle fiber type alteration with high-intensity intermittent training. European Journal of Applied Physiology, 54, 250-253.

Staron, R.S., Malicky, E.S., Leonardi, M.J., Falkel, J.E., Hagerman, F.C., & Dudley, G.A. (1989). Muscle hypertrophy and fast fiber type conversions in heavy resistance-trained women. European Journal of Applied Physiology, 60, 71-79.

Staron, R.S., Leonardi, M.J., Karapondo, D.L., Malicky, E.S., Falkel, J.E., Hagerman, F.C., & Hikida, R.S. (1991). Strength and skeletal muscle adaptations in heavy resistance-trained women after detraining and retraining. Journal of Applied Physiology, 70(2), 631-640.

Staron, R.S., Leonardi, M.J., Karapondo, D.L., Malicky, E.S., Falkel, J.E., Hagerman, F.C., & Hikida, R.S. (1994). Skeletal muscle adaptations during early phase heavy resistance-training in men and women. Journal of Applied Physiology, 76(3), 1247-1255.

Stone, M.H. (1992). Connective tissue and bone response to strength training. In P.V. Komi (ed.), Strength and Power in Sport (279-290). Oxford: Blackwell Scientific.

Tamaki, T., Uchiyama, S., Tamura, T., & Nakano, S. (1994). Changes in muscle oxygenation during weight-lifting exercise. European Journal of Applied Physiology, 68, 465-469.

Tesch, P.A. (1992). Short- and long-term histochemical and biochemical adaptations in muscle. In P.V. Komi (ed.), Strength and Power in Sport (pp.239-248).Oxford: Blackwell Scientific.

Tesch, P.A. (1992). Training for bodybuilding. In P.V. Komi (ed.), Strength and Power in Sport (pp.371-380). Oxford: Blackwell Scientific.

Tesch, P.A. & Karlsson, J. (1985). Muscle fiber types and size in trained and untrained muscles of elite athletes. Journal of Applied Physiology, 59(6),1716-1720

Tesch, P.A. & Larsson, L. (1982). Muscle hypertrophy in bodybuilders. European Journal of Applied Physiology, 49, 301-306.

Tesch, P.A., Thorsson, A., & Kaiser, P. (1984). Muscle capillary supply and fiber type characteristics in weight and power lifters. Journal of AppliedPhysiology, 56(1), 35-38.

Tamura, T., Eda, H., Takada, M., & Kubodera, T. (1988). New instrument for monitoring hemoglobin oxygenation. Advances in Experimental Medicine and Biology, 103-107.

Wilson, G.J. (1995). Disinhibition of the neural system: uses in programming, training, and competition. Strength and Conditioning Coach, 3(3), 3-5.

Wilson, J.R., Mancini, D.M., McCully, K., Ferraro, N., Lanoce, V., & Chance, B. (1989). Noninvasive detection of skeletal muscle underperfusion with near-infrared spectroscopy in patients with heart failure. Circulation, 80, 668-1674.

Appendix B: Informed Consent

Informed consent form for the research projects:

Near infrared estimation of muscle oxygenation during 4RM and 10RM weight lifting exercise

And

Acute skeletal muscle oxygenation during resistance training using NIRS

The purpose of these studies is to determine the influence of 4 repetition maximum (4RM) and 10 repetition maximum (10RM) resistance exercise protocols on skeletal muscle oxygenation and blood volume and to examine changes in acute muscle oxygenation and blood volume during acute resistance training that may act as a stimulus for a muscle fiber type shift.

Total duration of the study will be three days including an orientation session. During the orientation session, you will be instructed on proper lifting technique, have 4RM and 10RM values determined and introduced to the Near infrared spectrometer (NIRS; a surface probe used in the determination of muscle oxygenation). All exercise involves seated biceps curls performed on the preacher curl bench. Six exercise variations will be performed over the course of the three testing sessions. Prior to each exercise protocol a baseline will be established. This consists of a pressure cuff applied to the arm at 250 mmHg, occluding blood flow to the arm for a period of 8 minutes. Single set 4 repetition and 10 repetition contractions without a load will be completed as will a set unloaded 10 repetitions where blood flow is occluded throughout the exercise. As well, loaded 4RM, where four repetitions are performed to failure, and 10RM, where ten repetitions are performed to failure, will be performed. You will also complete a 10RM set with occluded blood flow. Trained subjects and untrained subjects will participate in both exercise protocols.

Muscle oxygenation and blood volume data will be collected using a portable near infrared spectrometer probe (NIRS). NIRS utilizes white light to analyze oxygen content of blood. It is a non-invasive procedure and poses no danger to the subject.

I acknowledge that the research procedures described in this form have been explained to me to my satisfaction. I am aware that muscle oxygenation will be measured for 10RM and/or 4RM protocols non-invasively with a NIRS probe. I understand that the 4RM and 10RM protocols may result in some discomfort in the form of delayed onset muscle soreness. I am aware that I can terminate my participation in any research procedures at any point without penalty and that any data collected will be destroyed within a period of five years. I understand that my participation or non-participation has no effect on my grades or standing and acknowledge no coercion of any kind has been used. I have been guaranteed anonymity as a subject and assured that all data will be kept confidential in a secured file.

I voluntarily consent to participate in this project and hereby release the University of Victoria and all personnel involved in and around this research project from any and all liability for any injury that may result from my participation in this study.

Participant name:	Participant signature:
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Title of Thesis:

NEAR-INFRARED ESTIMATION OF BLOOD VOLUME AND MUSCLE OXYGENATION DURING 4RM AND 10RM RESISTANCE-EXERCISE PROTOCOLS IN TRAINED AND UNTRAINED MALES.

Author: [redacted]

Sean Campbell

July 11, 2000