0-315-01221-8

National Library of Canada
Canadian Theses Division
Ottawa, Canada
K1A 0N4

Bibliothèque nationale du Canada
Division des thèses canadiennes

49079

PERMISSION TO MICROFILM — AUTORISATION DE MICROFILMER

• Please print or type — Écrire en lettres moulées ou dactylographier

Full Name of Author — Nom complet de l'auteur

ROBERT JAMES PRICE

Date of Birth — Date de naissance

3 - DECEMBER 1947.

Country of Birth — Lieu de naissance

AUSTRALIA

Permanent Address — Résidence fixe

3 GLENDENNING COURT FRANKSTON 3199
VICTORIA
AUSTRALIA

Title of Thesis — Titre de la thèse

FATIGUE ALLEVIATION IN MAXIMAL ISOKINETIC SHORT DURATION MUSCULAR ACTIVITY.

University — Université

ALBERTA

Degree for which thesis was presented — Grade pour lequel cette thèse fut présentée

M. Sc.

Year this degree conferred — Année d'obtention de ce grade

1980.

Name of Supervisor — Nom du directeur de thèse

DR. MOHAN SINGH.

Permission is hereby granted to the NATIONAL LIBRARY OF CANADA to microfilm this thesis and to lend or sell copies of the film.

The author reserves other publication rights, and neither the thesis nor extensive extracts from it may be printed or otherwise reproduced without the author's written permission.

L'autorisation est, par la présente, accordée à la BIBLIOTHÈQUE NATIONALE DU CANADA de microfilmer cette thèse et de prêter ou de vendre des exemplaires du film.

L'auteur se réserve les autres droits de publication; ni la thèse ni de longs extraits de celle-ci ne doivent être imprimés ou autrement reproduits sans l'autorisation écrite de l'auteur.

Date

24 JULY 1980

Signature

NOTICE

The quality of this microfiche is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us a poor photocopy.

Previously copyrighted materials (journal articles, published tests, etc.) are not filmed.

Reproduction in full or in part of this film is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30. Please read the authorization forms which accompany this thesis.

THIS DISSERTATION
HAS BEEN MICROFILMED
EXACTLY AS RECEIVED

AVIS

La qualité de cette microfiche dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de mauvaise qualité.

Les documents qui font déjà l'objet d'un droit d'auteur (articles de revue, examens publiés, etc.) ne sont pas microfilmés.

La reproduction, même partielle, de ce microfilm est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30. Veuillez prendre connaissance des formules d'autorisation qui accompagnent cette thèse.

LA THÈSE A ÉTÉ
MICROFILMÉE TELLE QUE
NOUS L'AVONS REÇUE

THE UNIVERSITY OF ALBERTA

FATIGUE ALLEVIATION IN MAXIMAL ISOKINETIC SHORT DURATION MUSCULAR ACTIVITY

by

ROBERT J. PRICE

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH IN

PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

IN

PHYSICAL EDUCATION

DEPARTMENT OF PHYSICAL EDUCATION

EDMONTON, ALBERTA

FALL, 1980

THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled FATIGUE ALLEVIATION IN MAXIMAL ISOKINETIC SHORT DURATION MUSCULAR ACTIVITY submitted by ROBERT J. PRICE in partial fulfillment of the requirements for the degree of Master of Science in Physical Education.

Supervisor

Date July 21, 1980

ABSTRACT

Thirty-six volunteer male subjects were systematically assigned to four treatment groups randomly designated Control, Central, Peripheral I and Peripheral II.

The study determined the extent to which the alleviation of fatigue in a maximal, anaerobic repeated measures work setting could be attributed to central nervous system facilitation (CNSF) or lactic acid removal. These were described as central or peripheral mechanisms.

The hypotheses examined related to whether central or peripheral fatigue mechanisms would play a dominant role in fatigue alleviation in high intensity anaerobic repeated measures work. The site of fatigue was limited to the flexors of the lower arm at the elbow. Subjects were required to repeatedly contract the flexors of the lower arm concentrically and eccentrically while attempting to sustain maximal force. This output was described as torque. Torques were measured during an isokinetic movement in which the range and speed of the lever arm of an electrical dynamometer were predetermined at 80^{o} and 13.3^{o} per second, respectively. IEMG analysis revealed that declining levels of torque were not parallelled by declining levels of electrical activity within the fatiguing muscles. Treatments were initiated at a torque equivalent to 50 percent of each subject's MVCC for each work phase.

No significant differences which could be attributed to the treatments were found but a treatments by phases interaction was found in a number of dependent variables.

It was concluded:

(1) That the maximal anaerobic muscular activity prevented the effects of CNSF from influencing the torques generated in a repeated work measures setting.

(2) That active recoveries did not cause any significant difference in the torques generated between the groups.

(3) That intense anaerobic muscular activity taxed both central and peripheral fatigue mechanisms to the extent that neither was observed to be dominant in fatigue alleviation.

ACKNOWLEDGEMENTS

The writing of a thesis undoubtedly involves many more individuals than the principal researcher. I owe a debt of gratitude to many who freely proferred advice and assistance.

I would like to express my thanks to:

- my advisor, Dr. Mohan Singh, who provided considerable help and support throughout;

- my committee, Dr. David Sande, Dr. Art Quinney, and especially Dr. Ted Wall, for his frank and unique assistance which was truly appreciated;

- the subjects of this study for their time and effort;

- Shirley Hilger and Peter Poznansky, who provided invaluable assistance during the period of data collection;

- Barry Gibson, for his guidance in the statistical analysis;

- my employer, the Council of the State College of Victoria - Toorak (Australia) for the opportunity to study at The University of Alberta;

- my colleagues in the Department of H.P.E.R. at SCV - Toorak for their encouragement and support;

- Clara Gallagher, for her expert assistance in preparing and typing this dissertation.

Finally to my wife Carol, and our children Briony and Wesley, thankyou for your love, support and patience, without which I could not possibly have accomplished such an undertaking.

TABLE OF CONTENTS

Appendices

LIST OF TABLES

LIST OF FIGURES

LIST OF PLATES

Chapter 1

INTRODUCTION

Two theories have emerged in studies related to fatigue; fatigue of neuromuscular transmission (Sloan, 1965; Viitasalo and Komi, 1977; Asmussen and Mazin, 1978a, 1978b; Asmussen, 1979; Wood, 1979), and fatigue of the contractile element within the muscle cell (Merton, 1954; DeVries, 1968; Hermansen, 1971; Karlsson, Funderburk, Essen, and Lind, 1975; Wenger and Reed, 1976; Weltman, Stamford; and Fulco, 1979). Asmussen (1979) described these two models as being central and peripheral fatigue sites and attempted to ascribe to the central site a dominant role in diminution of performance so commonly associated with the fatigued state.

Fatigue is associated with a number of homeostatic disturbances. Lippold (1952), Bigland and Lippold (1959), DeVries (1968), and Komi and Buskirk (1972) demonstrated changes in the electrical activity in muscle tissue as a function of time during repeated contractions. Increased blood lactate levels have long been associated with impaired muscular activity (Jervell, 1928; Bang, 1936; Hermansen, 1971), while lactate removal or disappearance has been associated with active recoveries (Newman, 1937; Hermansen and Stensvold, 1972; Karlsson et al., 1978; Weltman et al., 1979). Recently, fatigue has been described as a factor limiting performance. Wenger and Reed (1976) presented evidence to implicate a number of biochemical reactions within the contractile

mechanism, the cytoplasm of the muscle cell and the organelles of the cell as potential contributors to fatigue. Wood (1979) demonstrated with a test to measure human visual reaction time that local muscular fatigue contributed to a reduced speed of reflex reaction time. He claimed that this was associated with impaired neural impulse transmission. Viitasalo and Komi (1977) stated that a further characteristic of muscular fatigue was a reduction in the conduction velocities of action potentials along used muscle fibres.

Central and Peripheral Fatigue

Asmussen (1979) reported observations by a Russian physiologist, Setchenov, who, in 1903, described central nervous system facilitation (CNSF) as being responsible for the alleviation of the fatigue associated with repeated bouts of sawing wood. Asmussen (1979) also reported an observation made in 1914 that exercise performed with a non-fatigued muscle group would increase blood flow through the fatigued muscle group and thus facilitate recovery.

Asmussen and Mazin (1978a, 1978b) conducted a series of experiments to determine whether CNSF during recovery periods could influence repeated bouts of muscular activity and if exercise performed by the non-fatigued muscle groups during recovery periods had any effect upon repeated work bouts. They hypothesized that there may be two sites where fatigue could develop; a peripheral site distal to the motor end-plate, and a central site associated with the wave of depolarization which accompanies neural transmission. The central site was said to be proximal to the motor end-plate.

The findings by Asmussen and Mazin (1978a, 1978b) provided

evidence to "support {the} 'nervous' explanation and disprove {the} 'circulatory' explanation" (Asmussen, 1979:319).

Astrand and Rodahl (1977), and Karlsson et al. (1975), suggested that the buildup of lactic acid in exercising muscle precipitated the onset of fatigue. Weltman et al. (1979) examined recovery from maximal effort exercise, lactate disappearance and subsequent performance. They found that active recovery below the anaerobic threshold of their subjects during the rest periods enhanced lactate disappearance, which in turn had a positive effect on their subjects' overall work performance.

Statement of the Problem

A number of factors are important in considering the issue of the dominant site of fatigue alleviation being central or peripheral.

Selection of the task. This study examined the alleviation of fatigue developed in a local muscular site. Other studies (Belcastro and Bonen, 1975; Bonen and Belcastro, 1975; Davies, Knibbs, and Musgrave, 1970; Weltman et al., 1979) examined fatigue alleviation in 'aerobic' activities such as pedalling on a stationary bicycle ergometer or running on a treadmill. The means of inducing muscular fatigue in the current investigation was via an electrical dynamometer (Singh and Karpovich, 1965).

In this study, subjects performed a high intensity, anaerobic isokinetic movement designed to induce a decline in the torque generated from maximum to 50 percent in approximately two minutes. In so doing, it was assumed that the subjects were within the first phase of fatigue (Stephens and Taylor, 1972).

The subjects were required to concentrically and eccentrically

contract the flexors of the forearm at the elbow from their initial maximal voluntary concentric contraction to 50 percent. This fatigue inducing protocol was used:

(a) To determine whether over a repeated measures-rest interval time course, mechanisms in fatigue alleviation could be detected in the so-called 'first phase' of fatigue.

(b) To minimize subject discomfort subsequent to testing. It was considered that the strenuous nature of four bouts of concentric-eccentric contractions from maximal torque to 50 percent of maximum would itself generate muscle soreness in an essentially sedentary population. This concern was later proven correct as subjects reported some discomfort following testing.

(c) To reach the termination point of each work phase of the test session in the time frame of one to two minutes which was within the first phase of fatigue as described by Stephens and Taylor (1972).

Selection of the rest treatment. Newman, Dill, Edwards, and Webster (1937) demonstrated that moderate activity caused lactic acid to disappear faster than the resting rate. Davies et al. (1970) claimed that the optimal rate of lactic acid disappearance was around 40 percent of the subject's maximal oxygen uptake. It was shown by Weltman et al. (1979) that active recovery during rest intervals, just below their subject's previously determined anaerobic threshold, provided the most rapid decline in lactic acid levels.

One group in this current study engaged in 'moderate' recovery exercise during the treatment or rest interval phases. This group described as Peripheral II (P II) pedalled on a Monark bicycle ergometer at a heart rate of 140-150 beats per minute (bpm). A second group

engaged in 'mild' recovery exercise during the treatment phases. This group, Peripheral I (P I), pedalled on a Monark bicycle ergometer at a heart rate of 110-120 bpm. For both groups, rest interval activities or treatments were of four minutes duration.

Asmussen and Mazin (1978a, 1978b) and Asmussen (1979) claimed that groups practising central nervous system facilitation (CNSF) produced elevated levels of performance in repeated fatigue bouts when compared to control groups. A group designated Central in this current study, engaged in four minutes of CNSF in their rest interval activities. This was accomplished by the subjects solving as many mathematical problems as possible for the duration of their rest periods (Appendix J).

The fourth group in this current study was a Control group which undertook no special treatment during the rest intervals. Subjects in the Control group rested while sitting in the electrical dynamometer chair for the four minute periods.

Monitoring rest treatments. Blood lactic acid levels were determined at intervals during the test to describe conditions of rest, peak and recovery. This was done to observe the disappearance or removal of lactic acid which may have been attributed to a treatment effect. Further, this assay enabled a determination as to the possible production of lactic acid in the group whose rest interval activity was to pedal at a heart rate of 140-150 bpm.

The groups engaged in pedalling on the bicycle ergometer had their heart rates monitored by a Quinton Model 650 Heart Rate Meter during all treatment phases.

The decline in torque was monitored by a strain gauge placed close to the pivot point of the lever arm of the electrical dynamometer.

This was linked via shielded cable to a Honeywell amplifier. The amplified signal was relayed to a Beckman recorder. See Plate 2.

Electromyographic variables were monitored by surface electrodes placed on the anterior aspect of the biceps brachii (Komi and Buskirk, 1970). This signal was fed to an Accudata 136 integrator equipped for sinusoidal wave rectification to allow the quantification of the electrical activity in the working muscles. See Plate 1.

Definition of Terms

Amplitude -- The extent of the deflection of the EMG recording above or below the base line.

Central Nervous System Facilitation -- During muscle fatigue, a feedback of nerve impulses from the fatigued muscles impinges on part of the reticular formation of the brain and causes an inhibition of the voluntary effort. Rest interval activities (diverting) produce an increased inflow of impulses from non-fatigued parts of the body to the facilitatory part of the reticular formation, thus shifting the balance between inhibition and facilitation in a facilitatory direction (Asmussen, 1979:319).

Central Fatigue -- Is caused by an inhibition elicited by nervous impulses from receptors in the fatigued muscles. The inhibition may act on the motor pathways anywhere from the voluntary centres in the brain to the spinal motor neurons. The inhibition is most likely to originate in the reticular formation (Asmussen, 1979).

Concentric Contraction -- A contraction in which the muscle shortens.

Eccentric Contraction -- A contraction in which the muscle lengthens due to an external force.

Plate 1

Honeywell Electronic Medical System and
Beckman Metabolic Measurement Cart Recorder

Plate 2

Electrical Dynamometer

Fatigue -- The transient decrease in performance capacity of muscles that have been active for a certain time. Usually evidenced by a failure to maintain or develop a certain force or power (Asmussen, 1979).

Integrated Electromyograph (IEMG) -- An electronic interpretation of the electrical activity in muscle tissue. The activity can be summated or averaged to give a more simplified yet quantifiable recording of the normal electromyograph.

Motor Unit -- The nerve cell body of the central nervous system, "plus the axon running down the motor nerve plus it's terminal branches and all the muscle fibres supplied by these branches," together constitute a motor unit (Basmajian, 1968:8).

Motor Unit Potential or Muscle Action Potential -- When a wave of excitation, or an impulse from the central nervous system reaches the myo-neural junction where the axonal branches terminate in muscle fibres, "a wave of contraction spreads over the fibre resulting in a brief twitch followed by rapid and complete relaxation" (Basmajian, 1968:12).

Peripheral Fatigue -- Is caused by an imbalance in the 'milieu interne' of the muscle, usually associated with an increase in anaerobic metabolites such as lactic acid (Wenger and Reed, 1976).

Torque -- The turning effect produced when a force is exerted on a body or lever which is pivoted about some fixed point.

Limitations

(i) The use of surface electrodes to record the electrical activity of the contracting biceps brachii. This may lead to problems in that deep muscle activity is difficult to record and obviously single

motor unit activity cannot be recorded.

(ii) Due to the large numbers of participants, differences and variations in the nature and depth of the tissue superficial to the muscle under consideration may cause alterations in the quality of the electrical impulses picked up at the surface level.

Delimitations

(i) The study was limited to two fatigue sites described as central or peripheral.

(ii) The angular velocity of the electrical dynamometer was limited to 13.3° per second.

(iii) Only the right arm of each subject was involved in the phases of flexion-extension, thus the only muscles involved in the study were the flexors of the lower arm at the elbow.

(iv) Pilot study evidence indicated that where concentric or eccentric contractions were to be examined separately, the limitations imposed by the return phase of the lever arm of the electrical dynamometer produced an uncontrolled recovery phase which may have distorted the results.

(v) The subjects were 36 male students engaged in graduate and undergraduate studies at The University of Alberta.

Hypotheses

The resulting research design was a treatment groups (4) by repeated measures (4) design, in which the following hypotheses were generated and tested for significance at the 0.05 level of probability. Based on these considerations, the specific hypotheses developed for this study were:

Hypothesis 1. There would be no significant difference in the torque generated between the four groups which could be attributed to the different treatment conditions.

Hypothesis 2. There would be no significant difference in the integrated electromyographic variables related to the fatiguing muscles which could be attributed to the different treatment conditions.

Hypothesis 3. There would be no significant difference in blood lactate levels between the four groups which could be attributed to the different treatment conditions.

Chapter 2

REVIEW OF THE LITERATURE

Many investigators have provided evidence as to the general electrical activity which can be detected in contracting muscle since the phenomenon was first observed by Galvani in 1786. It is a relatively recent observation, however, that electrical activity in muscle tissue increases as a function of time when the muscle works against constant force in an isometric contraction (Edwards and Lippold, 1956, Lippold, 1952) or isotonically (DeVries, 1968; Merton, 1954; Sloan, 1965). It appears that the sum of the electrical activity of a muscle bears a simple relation to the force developed. Bigland and Lippold (1954) found the relation to be essentially linear, which suggested that the increase in force developed was largely dependent on the increased number of motor units in action and to a lesser extent on the frequency of discharge. Bigland and Lippold (1954) further claimed that the linear relationship between force developed and the integrated electrical sum indicated that in the absence of fatigue the integrated EMG was proportional to the number of motor units involved in the muscular contraction, and to a lesser extent to the frequency of their discharges. More recent work by Komi (1973) has confirmed these earlier findings and extended our understanding of the EMG. Komi showed that the integrated EMG activity was the same in maximal contractions of different speeds. He pointed out that this was indicative of the same degree of

motor unit recruitment in maximal efforts; and further, that it did not matter whether the contraction was concentric, isometric or eccentric (Komi, 1973).

Grossman and Weiner (1966) cast a serious doubt over the validity and reliability of much surface electromyographic research data with their assertion that the electrical activity of the underlying muscle tissue may not be related to tension generated within the muscle, but may actually be in response to environmental interference, plus the muscle action potentials. Their doubts were largely dispelled by the work of Komi and Buskirk (1970) who outlined a strict code of preparation for repeated surface and inserted electromyographic recordings. Their conclusions were that the surface electrode produced more reliable recordings over the inserted electrode, when measures were made on successive days, and unless the inserted wire technique could be further standardized, it appeared inappropriate for long-term use (Komi and Buskirk, 1970).

Integrated Electromyogram and Fatigue

Lippold (1952) designed a rigid wooden frame dynamometer into which his experimental subjects fixed their leg in such a manner to enable the forces rotating the foot around the ankle joint to be measured. All movements were eliminated except the pressure exerted by the sole of the foot when the calf muscles contracted. Lippold's (1952) subjects made voluntary contractions of varying known strengths while simultaneous electromyograms were recorded and integrated. Lippold (1952) was able to conclude that there was a linear relation between the integrated electromyogram (IEMG) and the tension produced, while the

coefficient of correlation varied between 0.93 and 0.99. DeVries (1974: 285-301) provided a clear explanation of the function of integration when examining the wave-form produced by an EMG.

Lippold's findings may have stimulated interest in using the electromyogram in other research contexts. Merton (1954) made use of the electrical potential-activity relationship when examining strength and fatigue. He provided evidence which led to the conclusion that fatigue was a peripheral occurrence, first by demonstrating that when strength failed, electrical stimulation could not restore the power to the muscle. Second, by demonstrating that recovery from fatigue did not take place if the circulation to the muscle was arrested (Merton, 1954). While Merton devised a complicated apparatus to examine the isotonic contractions in the muscle adductor pollicis to fatigue, Bigland and Lippold (1954) used Lippold's simpler dynamometer to examine the action potentials in the muscle gastrocnemius in constant shortening, lengthening and isometric contractions. Their conclusion that tension, velocity and electrical activity were interdependent has been an important finding for the field of electromyography (Bigland and Lippold, 1954:223).

There was a number of researchers inquiring into different aspects of muscular fatigue during the 1950's. Lind (1959) examined muscle fatigue and recovery from fatigue induced by sustained contractions. Sherrer and Bourguignon (1959) reported upon the changes in the electromyogram produced by fatigue in man, with their findings essentially in support of Bigland and Lippold (1954). Sherrer and Bourguignon (1959) investigated the relationship between the integrated electromyogram (IEMG) and fatigue generated in the triceps muscle group during isometric

and isotonic contractions. They, too, showed that the electrical activity as quantified by the IEMG, was in linear relation to the force developed; that the increase in force depended more on the increased number of motor units than on the frequency of discharge, and that in the absence of fatigue, the IEMG was proportional to the number of motor units and to a lesser extent on the frequency of discharge (Sherrer and Bourguignon, 1959:157). In their account of the phenomena associated with fatigue, Sherrer and Bourguignon referred to 'neuromuscular blockage' and noted that further research was needed to determine the actual 'site' of fatigue. It is interesting to note that, in contrast, Lind (1959) claimed his evidence supported conclusively the idea of fatigue as 'peripheral' in nature rather than neural or central.

It would appear that Asmussen (1979) brought back to life a debate which first appeared near the commencement of this century and then almost 50 years later. It is not clear why the issue of fatigue sites should be so cyclic. The answer to this latter query perhaps can be found in the intrinsic complexity of fatigue.

In 1965, Sloan conducted a surface electromyographic study of fatigue in the rectus femoris muscle of ten healthy young men between the ages of 19 to 26 years. Fatigue was induced by the application of the Harvard step test. Like the earlier workers, Sloan also observed an increase in the amplitude of the IEMG and ascribed it to an increase in the number of motor units firing synchronously. He speculated upon the progressive failure of the contractile mechanism by noting that:

> . . . the fatigue pattern of the EMG may be due to the increased stimulation from the motor cortex required to maintain the same degree of contraction as the muscle responds less efficiently. The increased upper motor neurone activity results in larger groups of motor units discharging synchronously with the observed

> result of larger action potentials. This phenomenon may be seen before any other manifestations of fatigue are obvious (Sloan, 1965:397).

DeVries (1968) experimentally induced fatigue in the elbow flexor and three extensor muscle groups. He also constricted blood flow to the exercising muscle groups and noted that circulatory arrest brought about marked changes in the nature of the isometric fatigue curve produced by the elbow flexor group at 20 percent of their maximal voluntary contractions. "This finding lends further support to the hypothesis of Merton and Lippold et al., that rapidly induced fatigue is a peripheral effect" (DeVries, 1968:134). Despite Sloan's (1965) speculation that fatigue in muscular contractions may be characterized by central nervous system activity, DeVries' definite assertion, plus the earlier lactic acid research of Astrand and Rodahl (1963), Gisolfi, Robinson, and Turrell (1966), Newman et al. (1937), and Rowell, Kraning, Evans, Kennedy, and Kusumi (1966) seemed to indict the 'milieu-interne' as being the predominant site of fatigue.

It has long been recognized that lactic acid levels within circulating blood are a reflection of the metabolic activity of parts, or all of the muscular system of the organism being examined (Bang, 1936; Jervell, 1928). It has also been long accepted that active recovery can alleviate the symptoms of fatigue faster than passive recovery (Newman et al., 1937).

The severity or intensity of the exercise will determine the level of lactic acid formed (Hermansen, 1971). Recent research has also indicated that there is a selective type of recruitment from the muscular system's motor-unit pool (Wenger and Reed, 1976). During high intensity exercise of short duration, the fast glycolytic pool (FG)

is substantially recruited. The metabolic enzymatic pathway which supplies the majority of energy to these muscles is anaerobic glycolysis. Wenger and Reed (1976) claimed that the production of lactate by anaerobic glycolysis was a primary cause of the decrease in the glycolytic rate. This suggested that fatigue induced in one to two-minute work bouts of high intensity might be linked to the 'milieu-interne', or peripheral site rather than the central, and that the FG motor unit fibres were the dominant source of lactic acid during short-term intense exercise.

Davies et al. (1970) examined the rate of lactic acid removal in relation to different baselines of recovery exercise and reported the maximum rate at approximately 40 percent of the individual's maximal oxygen uptake. It should be noted that these authors used only four subjects and did not attempt to relate the rate of removal to fitness. Bloom (1976) and Bransford and Howley (1979) found lactic acid to be produced at 30 percent and 55 percent of maximal oxygen uptake respectively in untrained subjects.

Many other researchers have recently examined the limiting effects of the presence of high levels of lactic acid on performance and methods by which these might be achieved. Belcastro and Bonen (1976), Bonen and Belcastro (1975), Gisolfi et al. (1966), and Hermansen and Stensvold (1973), provided evidence which indicated that lactate removal was enhanced during recovery by moderate aerobic exercise as opposed to resting recovery. The suggestion was that the more oxygen made available to the muscle during recovery, the greater the rate of removal of lactic acid. These experiments have used maximal aerobic exercise as the criterion exercise. Weltman et al. (1977) reported on

the relationship between different recovery patterns (active versus passive, with and without oxygen inhalation) from high intensity short duration exercise and subsequent performance. Their criterion test was an all out pedalling task against a 5.5 Kp. resistance. They reported a low correlation between post-recovery exercise and blood lactate levels which suggested that factors other than lactate removal were critical for subsequent performance. While making note of various possibilities such as oxidation in the heart muscle, resynthesis in the liver and lactate being 'lifted up' to form glycogen, Astrand and Rodahl (1977) concluded that, ". . . the rate of lactic acid produced during heavy exercise is still an unsettled question" (1977:313). Essen (1978) provided data showing that there was less accumulation per unit time of lactic acid during intermittent, than continuous intense exercise. Tesch (1978) related the ratio of fast twitch fibres (FT) and slow twitch fibres (ST) to exhaustive exercise and lactic acid production. He noted that lactate concentrations were related to the percentage of FT fibres suggesting that the lactate concentration/ performance time ratio was higher in FT muscles. He concluded that subjects rich in FT fibres had higher anaerobic work capacities than those with higher percentages of ST fibres. Buchtal and Schmalbruch (1969) claimed that in the muscle biceps brachii, 25 percent of the fibres in the long head were rich in mitochondria and therefore could be termed FT fibres; they did not report on the fibre type of the short head of the muscle biceps brachii.

Weltman et al. (1979) continued to study recovery patterns from maximal effort exercise, lactate disappearance and subsequent performance using recovery thresholds above and below their subjects'

previously determined anaerobic threshold (AT). They concluded that elevated blood lactate had little effect on maximal effort duration and that exercise recovery below AT was more effective for lactate disappearance than exercise recovery above AT.

There appeared to be a shift in emphasis in the research of the 1970's away from the gross muscle studies of fatigue utilizing surface electrodes toward the use of inserted electrodes revealing various phenomena related to single motor units (Milner-Brown, Stein, and Yemm, 1973a, 1973b; Milner-Brown and Stein, 1975). A second area of considerable IEMG research in this period was the attempted diagnosis of neuro-muscular disorders (Goodgold and Eberstein, 1973; Smorto and Basmajian, 1977). In this latter context, Smorto and Basmajian (1977:13) claimed that exact interpretation of the recorded electrical activity of the skeletal muscle and peripheral nerves permitted the determination of the extent of nerve injury or the nature of intrinsic muscle disturbance and could help in planning medical therapy, surgical intervention and rehabilitation.

While there seems to be cyclic periods of activity, it is reasonable to assert that fatigue has been one of the most fruitful areas of research for electrophysiologists and exercise physiologists. Within the literature can be found a two model debate as to whether the site of muscular fatigue is located within the central nervous system or within the peripheral musculature.

Noting that the two models have their respective devotees, it was interesting to record that Stephens and Taylor (1972) proposed a

model of fatigue which implicated both central and peripheral sites. They reported that in sustained contractions there was an initial drop in the integrated EMG, while further decreases were accompanied by a lesser decline in IEMG. Stephens and Taylor (1972) suggested that the initial phase was indicative of transmission fatigue (fatigue of the neuromuscular junction), while the subsequent phase represented an inability of the contractile apparatus to produce tension.

Adding to the full restoration of the debate within the pertinent literature, Viitasalo and Komi (1977) described the signal characteristics of the EMG and fatigue. At the same time, they provided considerable evidence for the central model theorists. They suggested that along with recruitment of new motor units and increases in amplitude of the wave-form due to synchronous firing of motor units, that fatigue was characterized by a marked reduction in the conduction velocity of the action potential along the used muscle fibre.

The issue of fatigue which was seemingly resolved with the dominant site being peripheral, or at best being a two component phenomenon, was seriously challenged by this latter finding. Currently, there is renewed interest in the subject. In fact, Asmussen (1979) and Asmussen and Mazin (1978a, 1978b) claim to have demonstrated that the dominant site of fatigue is central. They received some support from Wood (1979) is his examination of the effects of fatigue upon visual reaction time. Wood (1979) reported exercise-induced changes in reaction time performance which appeared centrally mediated, involving a shift in motor unit recruitment patterns by the motor cortex of the brain.

Chapter 3

METHODS AND PROCEDURES

Subjects

Thirty-six male volunteers whose ages ranged from 19 to 35 years participated as subjects in the study. All were advised not to eat, drink, smoke or engage in any heavy physical exercise for a period of four hours prior to their test.

The subjects were scheduled for two visits to the laboratory. First, for the measurement of isometric strength of the flexors of the arm at the elbow to allow a systematic random assignment of subjects to treatment groups.

All signed an 'informed consent' document indicating their understanding of the procedures and willingness to participate in the study (Appendix I).

All subjects were required to come for their test wearing a gym strip of shorts, a loose shirt top and gym shoes. Upon entering the laboratory, each subject was familiarized with the test requirements and recording procedures.

Various anthropometric measures were taken with the subjects wearing only gym shorts and either socks or bare feet. The measures were: age in years and months; height in centimetres; weight in kilograms; and three arm measures. The subjects were asked to stand with their right arm flexed to 90° at the elbow with their lower arm

supinated. By palpating the acromion process of the scapular and the lateral epicondyle of the humerus and measuring the distance between these two anatomic landmarks, an upper arm length was derived (UAL). The lower arm length was measured as the distance between the lateral epicondyle of the humerus and the distal head of the radius (LAL). Both these measures were made in inches, converted to centimetres and added to give a measure of total arm length (TAL). All subjects were measured using these procedures with distances measured by a simple 'dressmakers' tape (Appendix A).

In order to facilitate the assignment of subjects to the four treatment groups, each was measured for maximal isometric arm flexor strength using a cable tensiometer. This procedure measured the isometric strength of the flexors of the arm at the elbow with the subjects sitting comfortably in a Hettinger chair (illustrated in Plate 3). All subjects placed their elbows by their sides, flexed to 90° as determined by a goniometer. The subjects supinated their lower arm and a tensiometric cuff, cable and chain was attached to a point vertically below their wrist. The cuff position, seating and flexed lower arm angle were standardized for all subjects by making individual adjustments to the apparatus. The subjects were instructed to maximally contract the flexors of the lower arm three times. The first for familiarization purposes, the second and third to determine the forces applied to the cable as measured by a cable tensiometer. The forces were recorded with the higher of the two recordings assigned as the subject's score. These scores were converted to pounds equivalents, utilizing a modified spring-scale calibration unit developed at The University of Alberta. The calibrated scores were then ranked in order of highest

Plate 3

Hettinger chair used to standardize the test of isometric strength of the flexors of the lower arm at the elbow.

Plate 4

Terrasyn Isolation Amplifier. Electrode jelly. Disposable surface electrode. Alligator clips.

to lowest in a descending scale of one to 36 to facilitate the systematic random assignment of subjects to treatment groups.

Equipment

Measurements were made of the electromyograph (EMG) and the integrated electromyograph (IEMG) using a Honeywell Electronic Medical system (Bigland and Lippold, 1954; DeVries, 1974; Lippold, 1952). This is illustrated in Plate 4. Surface electrodes transferred the muscle action potentials to an isolation amplifier (Terrasyn Inc. N IIIA EMG) via colour-coded alligator clips (AMI Co.) and shielded cables. Surface electrodes for the EMG recording were positioned according to the specification of Komi and Buskirk (1970). The electromyographic potentials were fed into an electrically operated integrator equipped for full wave rectification which produced an arbitrary quantitative figure. (For further detail of the operation of the Honeywell Electronic Medical system as used in this study, see Appendix D).

The torque developed by the subjects during the four phases of the test was recorded using an electrical dynamometer (see Plate 2). The subjects were instructed to sit with their backs straight and to apply force only through the concentric and eccentric contractions of the flexors of the lower arm at the elbow. In an attempt to eliminate extraneous movements, subjects were verbally prompted only to use the muscles of the upper arm most involved with the activity.

Circuit breaker cams in the electrical dynamometer were adjusted so that all subjects worked in the range of 65° of extension to 145° of flexion. The range of movement was set at 80°. The lever arm speed was set to cover the 80° in six seconds or at 13.3° per second. A

cycle of concentric-eccentric contractions took 14 seconds as the lever arm was manually reactivated at the extremity of each movement.

Close to the pivot point of the lever arm was a strain gauge. Electrical impulses from the strain gauge were fed into an amplifier within the Honeywell Electronic Medical system and then to a Beckman pen recorder. This enabled close monitoring of the decline in torque as fatigue levels increased. When a subject was unable to develop 50 percent of his initial maximal voluntary concentric contraction as indicated in the Beckman recorder, the phase of work was stopped. The subject then moved as quickly as possible into a treatment phase of the test.

Heart rates were monitored in two groups during their rest interval activity using a Quinton (Model 650) heart rate meter. The groups engaged in active recovery pedalled a stationary Monark (Varburg, Sweden) bicycle ergometer.

Procedure

Figure 1 provides a schematic representation of the research design used in this study. Thirty-six subjects were systematically assigned to groups. These groups were randomly assigned to the treatments.

The protocol of this study included a resting blood sample followed by the first concentric-eccentric work phase to 50 percent of the subjects' initial maximal voluntary concentric contraction (MVCC). The groups began the first treatment and after four minutes, they completed the second work phase to 50 percent of MVCC and commenced the second treatment. They then completed the third work phase to 50 percent

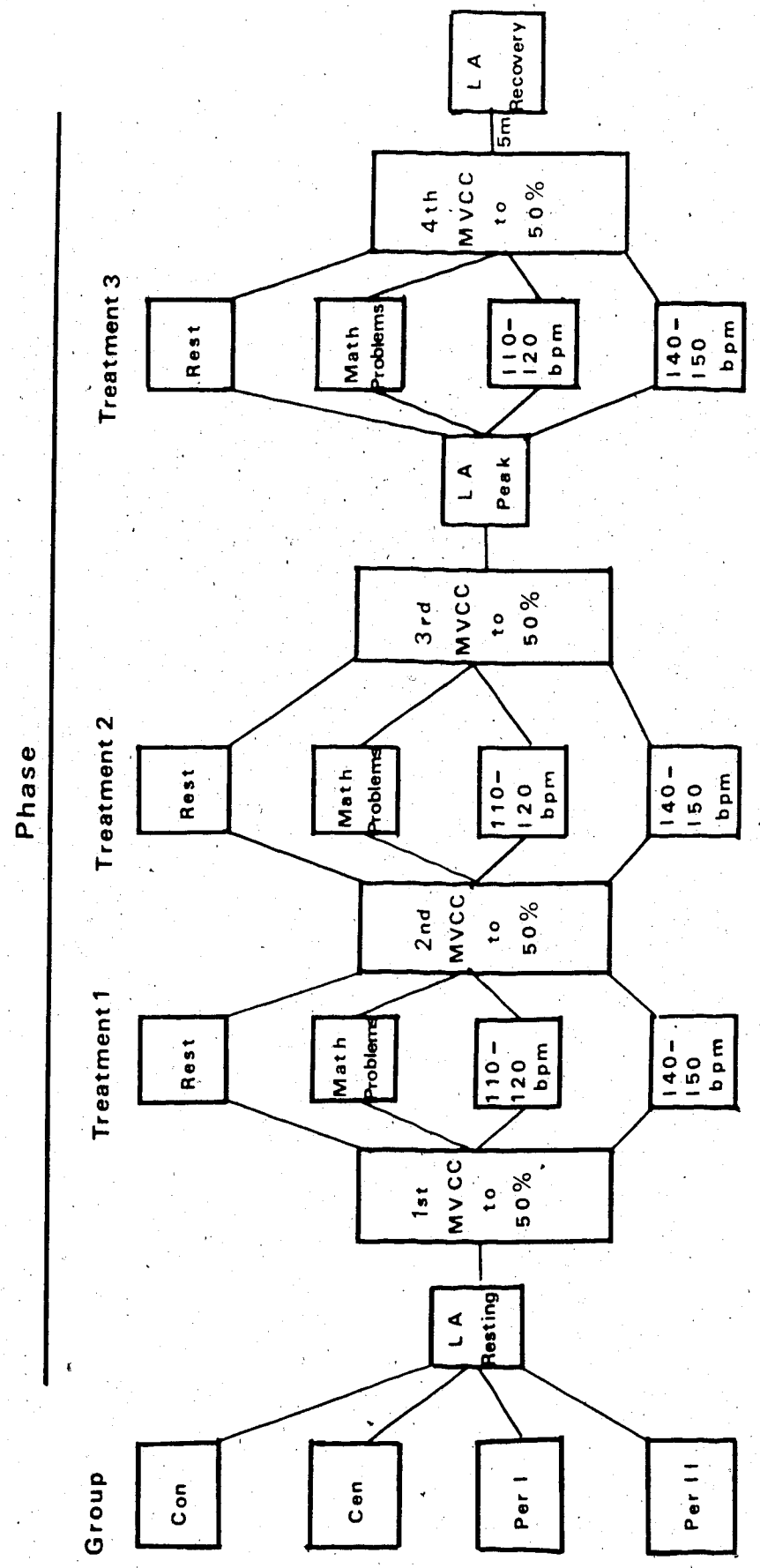

Figure 1. Schematic representation of the research design.

of MVCC which was followed by a second blood sample. This was used to indicate a peak lactic acid level. The subjects completed treatment three after the second blood withdrawal. The subjects completed the fourth phase of work to 50 percent of their MVCC which was followed by a period of five minutes where all subjects sat and rested in the electrical dynamometer chair. A third blood sample was then drawn to indicate a recovery value of lactic acid.

Group I Control (Con)

Subjects in the Control group performed no specific activity during their three rest intervals. They were instructed to sit quietly and relax for the four minutes.

Group [illegible] (Cen)

[illegible]s in the Central group remained seated in the electrical dynamometer chair. During their three rest intervals, they were given a clip board, pen and sheet of simple mathematical calculations. Included on the sheet were arithmetic problems related to addition, multiplication, subtraction, division and equations. For each rest interval, the subjects were asked to complete as many 'problems' as they could in the four-minute periods (see Appendix J).

Group III Peripheral Activity I
(P I)

Immediately upon the termination of each concentric-eccentric phase, the subjects were asked to pedal a stationary Monark bicycle ergometer. The subject's heart rate and the power output were monitored and the resistance altered as required to elicit a heart rate response of between [illegible]120 beats per minute (bpm). At the completion of the

four minutes, the subject stopped and moved back to the electrical dynamometer chair and commenced the next concentric-eccentric phase of the study. Details of heart rate monitoring and electrode placement are provided in Appendix F.

Group IV Peripheral Activity II
(P II)

Subjects assigned to this group were monitored as in Peripheral I, but utilized a target heart rate of 140-150 beats per minute.

Lactic Acid Levels

Blood was drawn from all subjects on three different occasions during the test session into heparinized 4 ml vacutainers.

(i) The first withdrawal was made from the antecubital vein of the non-exercising arm approximately one minute prior to test commencement. All subjects had been sitting in a quiet state for at least five minutes. This sample was taken to provide a base line value of lactic acid for later comparative purposes.

(ii) The second withdrawal was made from the antecubital vein of the exercising arm as soon as the subject had reached the termination point of the third concentric-eccentric phase. This sample was taken to provide a peak value of lactic acid.

(iii) The third withdrawal was made after the fourth work bout. All subjects reached the termination point of the fourth concentric-eccentric phase and were instructed to sit quietly for five minutes. Blood was drawn from the antecubital vein of the exercising arm, again utilizing a site approximately two to three mm below the first site. This sample provided evidence of the rate of removal or disappearance

of lactic acid from the fatigued muscle site.

Lactic Acid Assay

The lactic acid assay followed a similar procedure to that described by Mohme-Lundholme (1965) and was based upon the light absorbance properties of the enzyme NADH.

In the presence of LDH and NAD, lactate is converted into pyruvate. For each mole of lactate converted to pyruvate, one mole of NAD is converted into NADH. Therefore, the absorbance of NADH of ultra-violet light at 340 mn, reflects on a 1:1 basis the amount of lactate originally in the sample. The absorbance properties were determined in a Pye Unicam SP1800 UV spectrophotometer (see Appendix G).

Data Analysis

The major purpose of the study was to determine the differential effect of the four treatment conditions. The resulting experimental design was a treatment (4) by repeated measures (4) design. The statistical analysis of the following dependent variables was completed by a two-way ANOVA with repeated measures on the last factor.

Statistical Analysis

The data collected were subjected to a two-way analysis of variance with repeated measures on the last factor for each of the following dependent variables:

1) maximum concentric torque. Maximum torque developed during the initial concentric contraction of each phase of work.
2) minimum concentric torque. Torque developed during the final concentric contraction, usually 50 percent of the initial contraction.

3) mean concentric torque. Torque value derived by totalling the torque of each concentric contraction and dividing by the number of contractions.

4) maximum eccentric torque. Maximum torque developed during the initial eccentric contraction of each phase of work.

5) minimum eccentric torque. Torque developed during the final eccentric contraction.

6) mean eccentric torque. Torque value derived by totalling the torque of each eccentric contraction and dividing by the number of contractions.

7) sum of the torques in the first four concentric contractions.

8) sum of the torques in the first four eccentric contractions.

9) sum of the integrated value of the electromyographs for the first four concentric contractions in millivolts.

10) sum of the integrated value of the electromyographs for the first four eccentric contractions in millivolts.

Statistical computations were made using the Department of Educational Research programme ANOV 26.

A diagrammatic representation of the experimental design for this study was presented in Figure 1.

Chapter 4

RESULTS

Subject Characteristics

Thirty-six subjects participated in the study and were blocked into four groups which were randomly assigned to treatments. Individual characteristics are provided in Appendix A.

Table 1 includes the mean ($\bar{X}$), standard deviation (SD) and range (R) for the characteristics of the subjects in the four groups. Two data points are of interest. The mean age and the mean height for Peripheral II appear to be different to those values derived from the other groups. No explanation can be offered for these effects other than random experimental occurrences.

The results obtained from the current study refer to three main areas. Firstly, the torque developed by each subject during the four concentric-eccentric work phases. Torques were measured and obtained for each concentric and eccentric contraction. As the subjects applied force to the lever arm of the electrical dynamometer, a Beckman recorder was used to simultaneously record the torques. At 50 percent of the subject's initial maximal voluntary concentric contraction (MVCC), which took 1-1½ minutes, the work phase was terminated and a treatment commenced. It will be apparent that some of the dependent variables in this current study have been derived using the first four contraction cycles. This was necessary as in the latter work phases, the 50 percent decline in MVCC occurred

Table 1

Means ($\bar{X}$), Standard Deviation (SD), and Range (R) of Anthropometric Measures for All Subjects

Group		Age (Years & Months)	Height (Centimetres)	Weight (Kilograms)	Upper Arm Length	Lower Arm Length	Total Arm Length
Control	$\bar{X}$	23.4	172.7	73.2	32.9	26.7	59.6
	SD	3.40	8.80	9.10	1.93	1.81	3.58
	R	20.6 - 32.6	157.4 - 189.8	61.2 - 85.8	29.4 - 35.5	24.5 - 30.4	53.9 - 65.9
Central	$\bar{X}$	24.18	177.65	75.58	33.52	27.71	61.24
	SD	2.56	8.89	10.07	2.39	1.69	3.87
	R	21.0 - 27.7	168.2 - 188.6	62.1 - 93.2	30.0 - 37.5	26.0 - 30.4	56.3 - 67.9
Peripheral I	$\bar{X}$	23.5	175.3	75.5	33.4	27.5	61.0
	SD	2.77	4.9	6.5	1.5	1.19	2.29
	R	19.0 - 27.6	164.4 - 180.0	63.5 - 85.1	31.7 - 35.5	25.4 - 29.2	56.5 - 63.6
Peripheral II	$\bar{X}$	28.9	170.78	75.16	29.7	27.0	59.9
	SD	4.02	7.04	8.74	1.59	1.26	2.61
	R	23.2 - 35.3	156.0 - 180.6	60.8 - 83.1	31.1 - 36.8	26.0 - 28.1	58.4 - 66.0

in only four concentric-eccentric contraction cycles in some subjects. Secondly, data was obtained related to integrated electromyographic variables (IEMG) using an Accudata 136 integrater, a component of the Honeywell Medical system. These results are reported in millivolts of electrical activity and reflect the neuromuscular action potentials of the contracting muscles. The third area relates to the lactic acid levels of the subject's blood determined at three stages during the experimental protocol (see Figure 1) to establish a resting, peak and recovery value.

Torque Versus Treatments

The effects of rest interval activities upon the alleviation of fatigue are shown in Figure 2. This represents the torque in kilogram meters for the total of the first four concentric contractions after averaging. The first four concentric torque values were totalled and divided by four. Figure 2 clearly demonstrates the decline in torque over the four work phases for the four treatment groups. A two-way analysis of variance showed no significant treatment main effects or interactions on torque developed for the mean of the sum of the first four concentric contractions; therefore, no significant difference between groups could be attributed to the treatment conditions (see Appendix C).

Figure 3 shows the decline in torque in kilogram meters for the first four eccentric contractions when they were added together and an average value obtained. A two-way analysis of variance resulted in no significant treatment main effects on torque developed which indicated that again, there was no difference between the groups which could

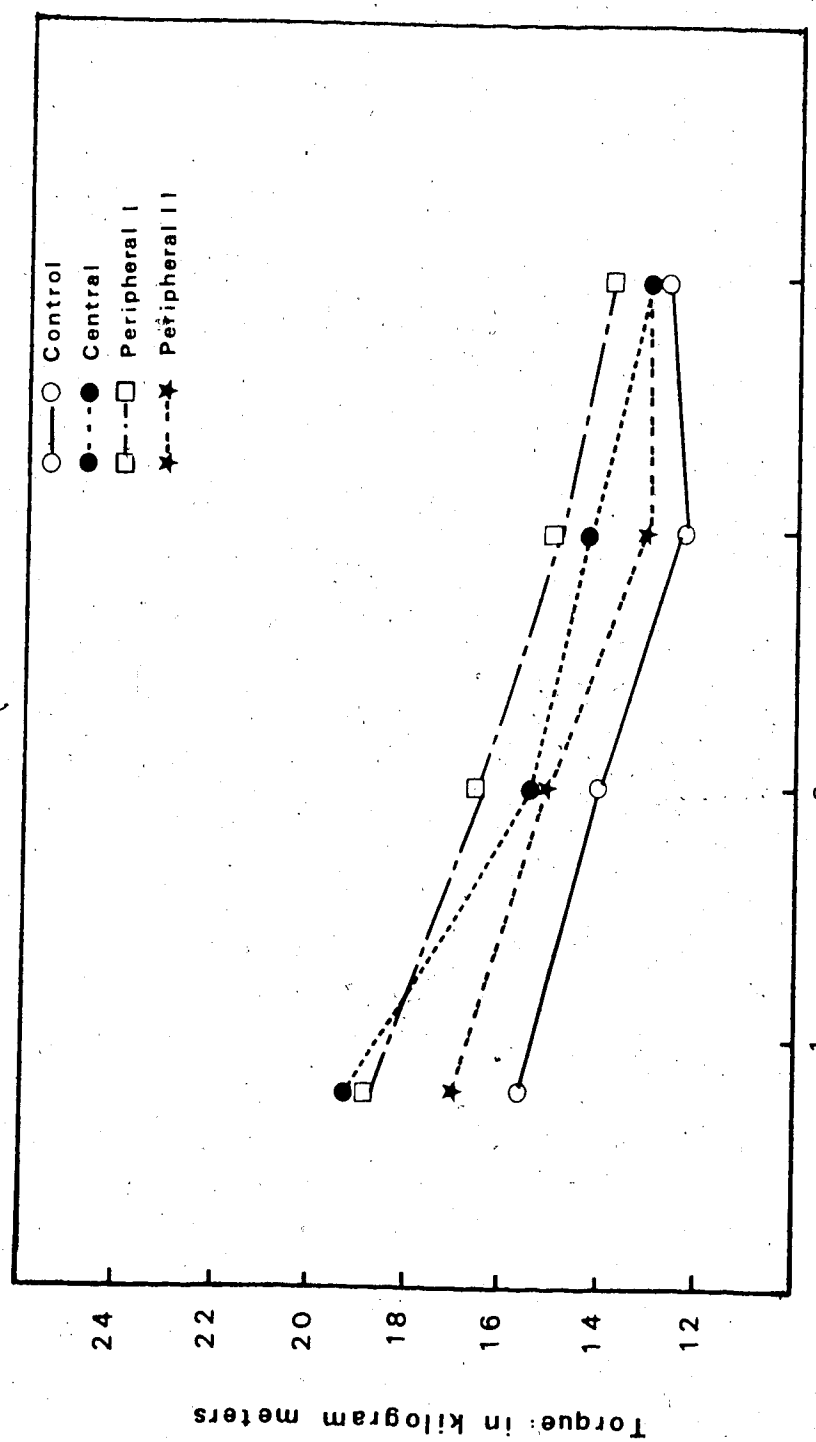

Figure 2. Sum of first four concentric contractions(averaged).

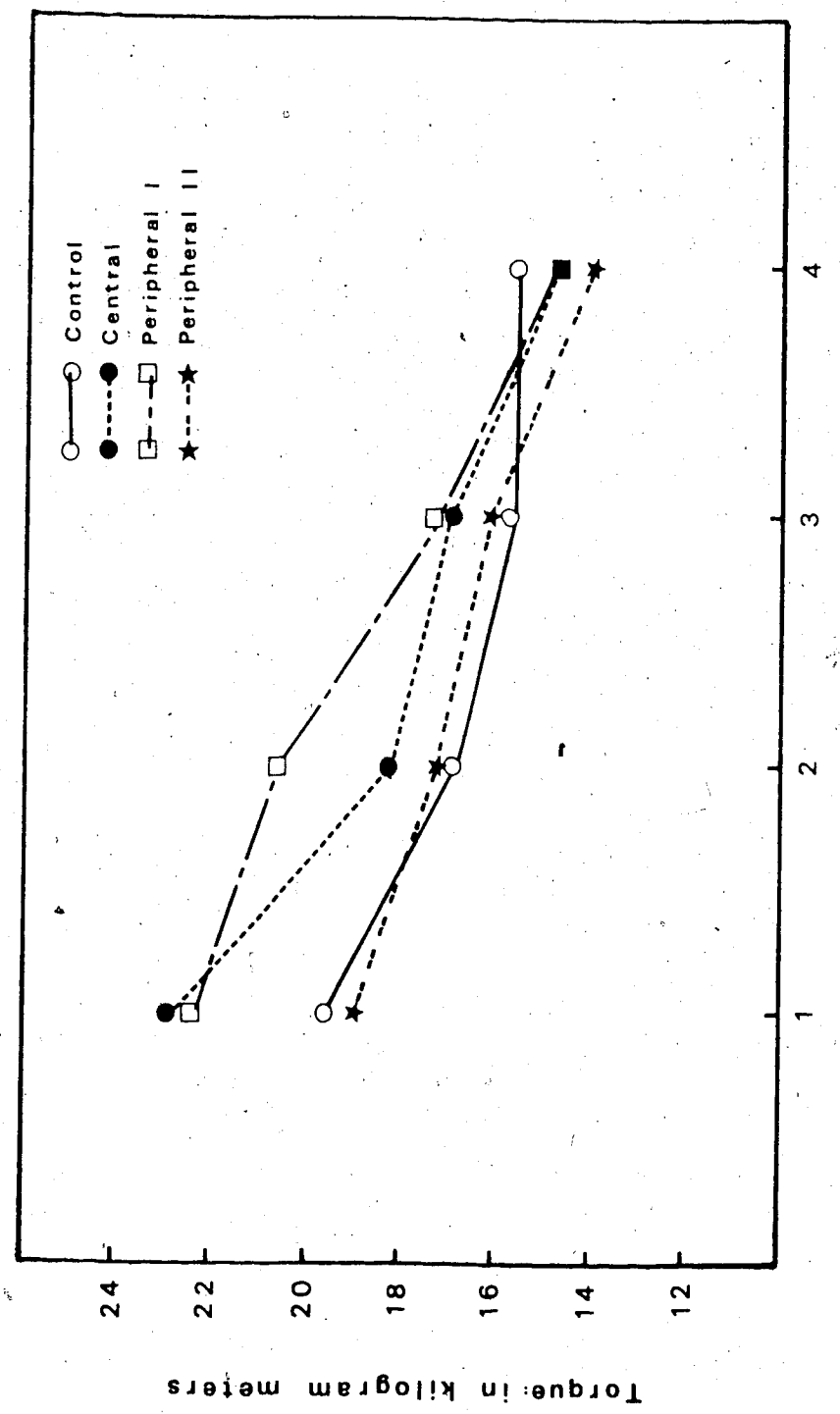

Figure 3. Sum of first four eccentric contractions.

be attributed to the treatment; however, a significant treatment by phases interaction was found. Simple effects tests indicated that at phase I, the Control and Peripheral I groups generated more torque than the Central and Peripheral II groups with the largest cell mean difference appearing between Central and Peripheral II $F(1,96) = 6.2$, $p < 0.05$. The difference between Peripheral I and Peripheral II was significant $F(1,96) = 5.6$, $p < 0.05$. Significance was also found for the difference between Central and Control $F(1,96) = 5.1$, $p < 0.05$. The only significant group difference between phase I and phase II involved the Central group $F(1,96) = 7.1$, $p < 0.05$. Therefore, the lower torque generated by the Central group in phase II in contrast to phase I must be due to errors in sampling as no other group demonstrated this effect.

Figure 4 illustrates the value derived when the mean torques for the concentric and eccentric contractions were added together. It should be noted that one subject took 12 cycles of concentric-eccentric contractions before he reached 50 percent of his MVCC while another subject took only four to reach his termination point (see Appendix H). The two-way analysis of variance revealed no significant main effects on torque developed. However, a significant treatment by phases interaction was found. Again, the results of the simple effects tests indicated that during phase I, Central and Peripheral I groups generated more torque than Control and Peripheral II groups. The largest mean cell difference was between Central and Control $F(1,96) = 6.59$, $p < 0.05$. Significant mean cell differences were found between Central and Peripheral II $F(1,96) = 6.2$, $p < 0.05$, also Peripheral I and Control $F(1,96) = 6.0$, $p < 0.05$, and Peripheral I and Peripheral II $F(1,96) =$

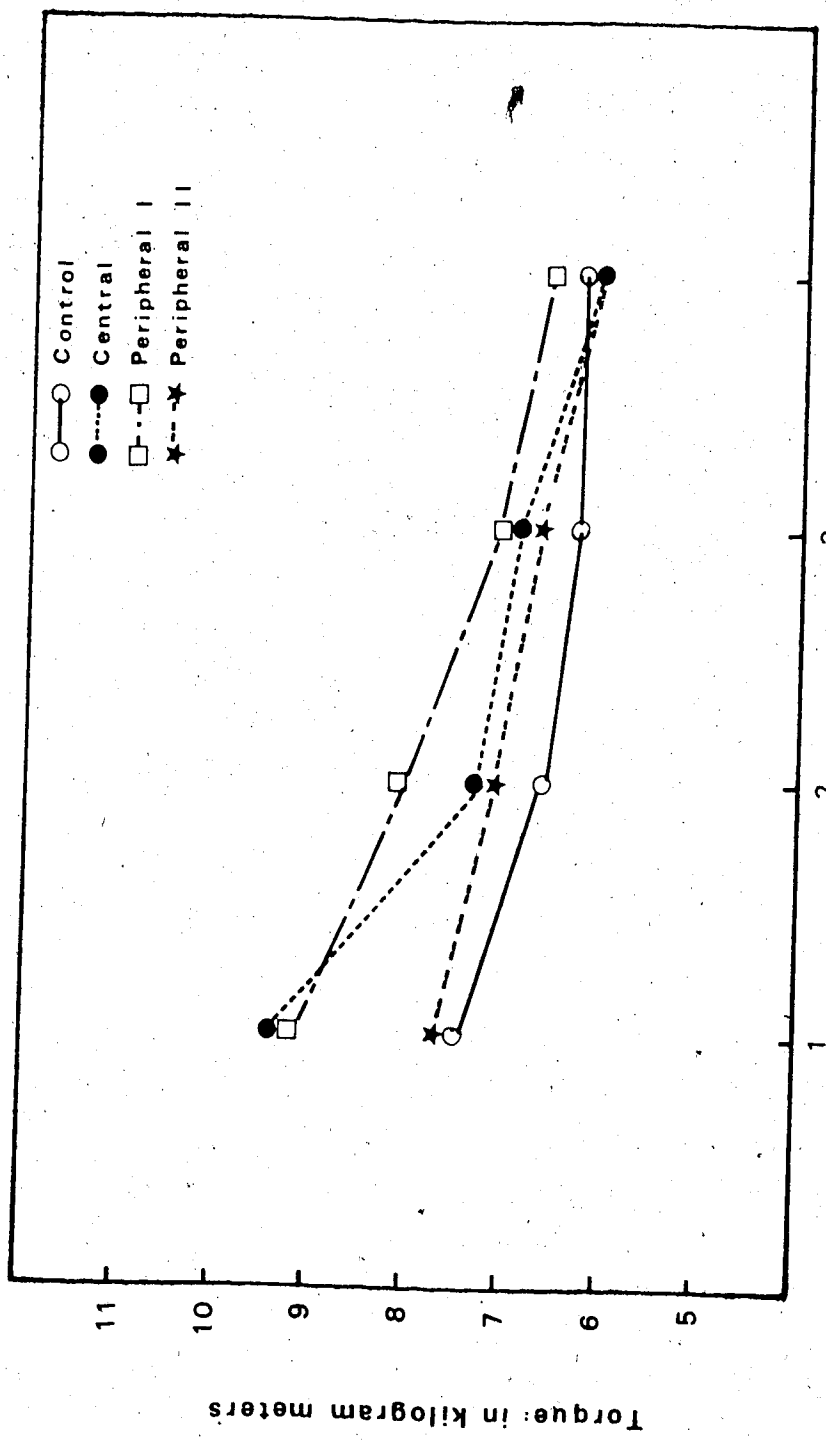

Figure 4. Sum of mean concentric, mean eccentric contractions.

5.6, $p < 0.05$. Figure 4 demonstrates that the only significant group difference between phase I and phase II involved the Central group $F(1,96) = 8.1$, $p < 0.05$. This must be attributed to the initial error in sampling.

Figure 5 illustrates the torque value derived during the eccentric contraction phase of the current study. This value represents a group mean in kilogram meters. It was derived by totalling an individual subject's torque for each phase and dividing that total by the number of contractions it took to reach 50 percent of his MVCC. Group means were then obtained by adding the individual means for the group and dividing by nine. The two-way analysis of variance revealed no significant treatment main effects on torque developed. A significant treatment by phases interaction was revealed. The results of the simple effects testing revealed the higher torque levels generated in phase I by the groups Central and Peripheral I. The largest mean cell difference was between Central and Peripheral II $F(1,96) = 6.7$, $p < 0.05$. Significant mean cell differences were also found between the Central and Control groups $F(1,96) = 5.85$, $p < 0.05$, also Peripheral I and Peripheral II $F(1,96) = 5.78$, $p < 0.05$, and Peripheral I and Control $F(1,96) = 4.83$, $p < 0.05$. Figure 5 continues to demonstrate that the only significant phase I - phase II difference involved the Central group $F(1,96) = 8.0$, $p < 0.05$. Once again, this can only be attributed to the initial sampling error.

Integrated Electromyographic Results

Figure 6 illustrates the integrated electromyographic results obtained when the integrated electrical activity was totalled for the

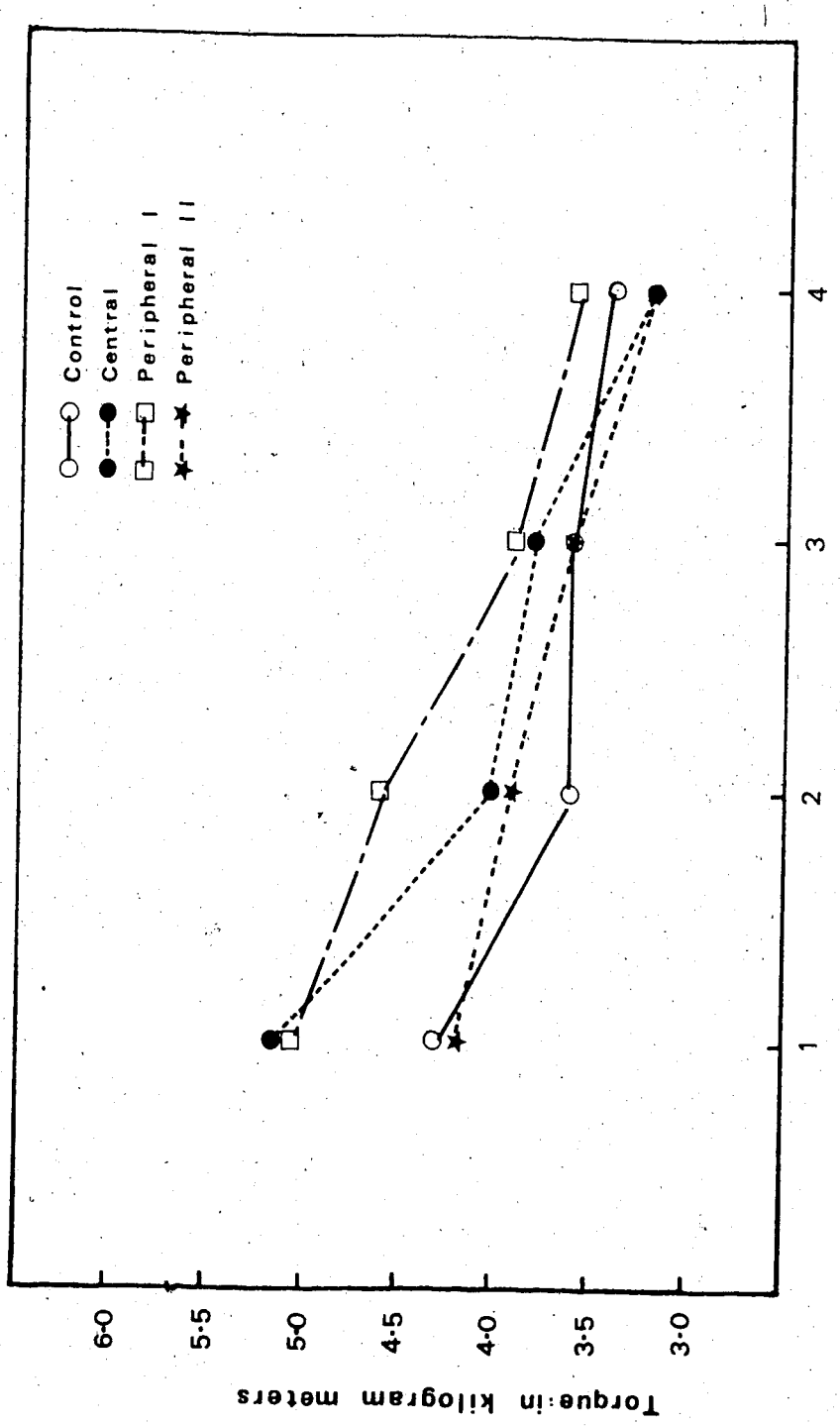

Figure 5. Mean eccentric contractions.

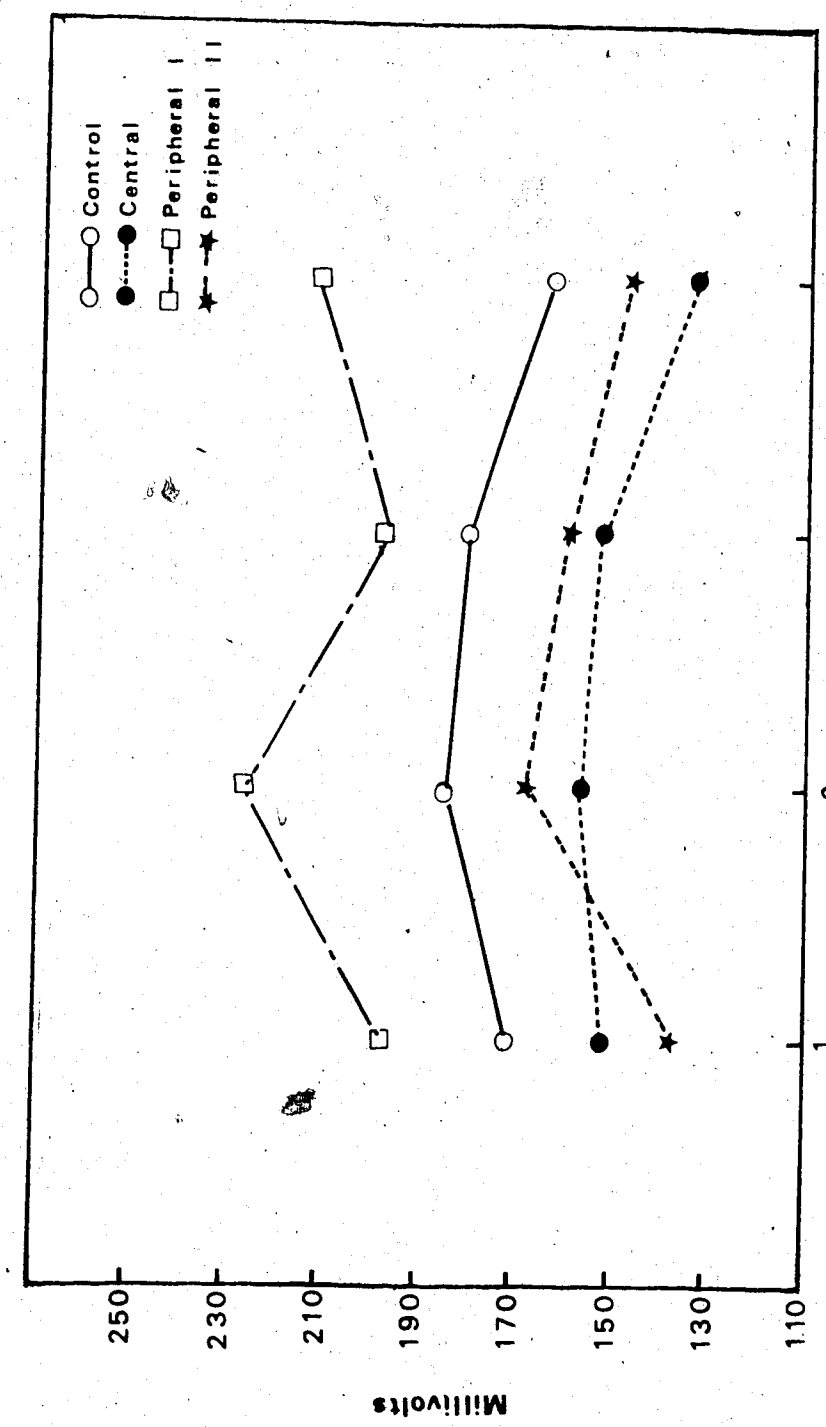

Figure 6. Integrated electromyographic data: Concentric contractions.

first four concentric contractions for each phase of work. Inspection of Figure 6 reveals a general increase in electrical activity from treatment 1 to treatment 4 in the groups Peripheral I (P I) and Peripheral II (P II). The Control and Central groups show a slight decline in electrical activity for the first four concentric contractions between treatment 1 and treatment 4. This increase in electrical activity in P I and P II is accompanied by the slight decline in Control and Central contrasts markedly with Figure 2 which showed the drop in torque developed during the four phases of work for the first four concentric contractions. A two-way analysis of variance revealed no significant treatment main effects on summated electrical potentials in millivolts during the first four concentric contractions.

Figure 7 illustrates the summed IMEG recording for the first four eccentric contractions. There was an increased IEMG recording of millivolts of electrical activity for the group P I between the first and fourth concentric-eccentric phase of work. A slight increase was present in the IEMG millivolt recorded between phases one and four for the groups P II and Control. This trend was reversed for the Central group. The electrical activity declined overall between treatments 1 and 4 for the Central group.

The increased electrical activity in the treatment groups P I and P II described for the concentric contractions was not so clearly marked for the eccentric contractions. The increased electrical activity for the Control group between the first and last work phases for eccentric contractions differed from the finding for this group during the concentric contractions. The trends for the Central group remained the same for the eccentric contractions as for the concentric.

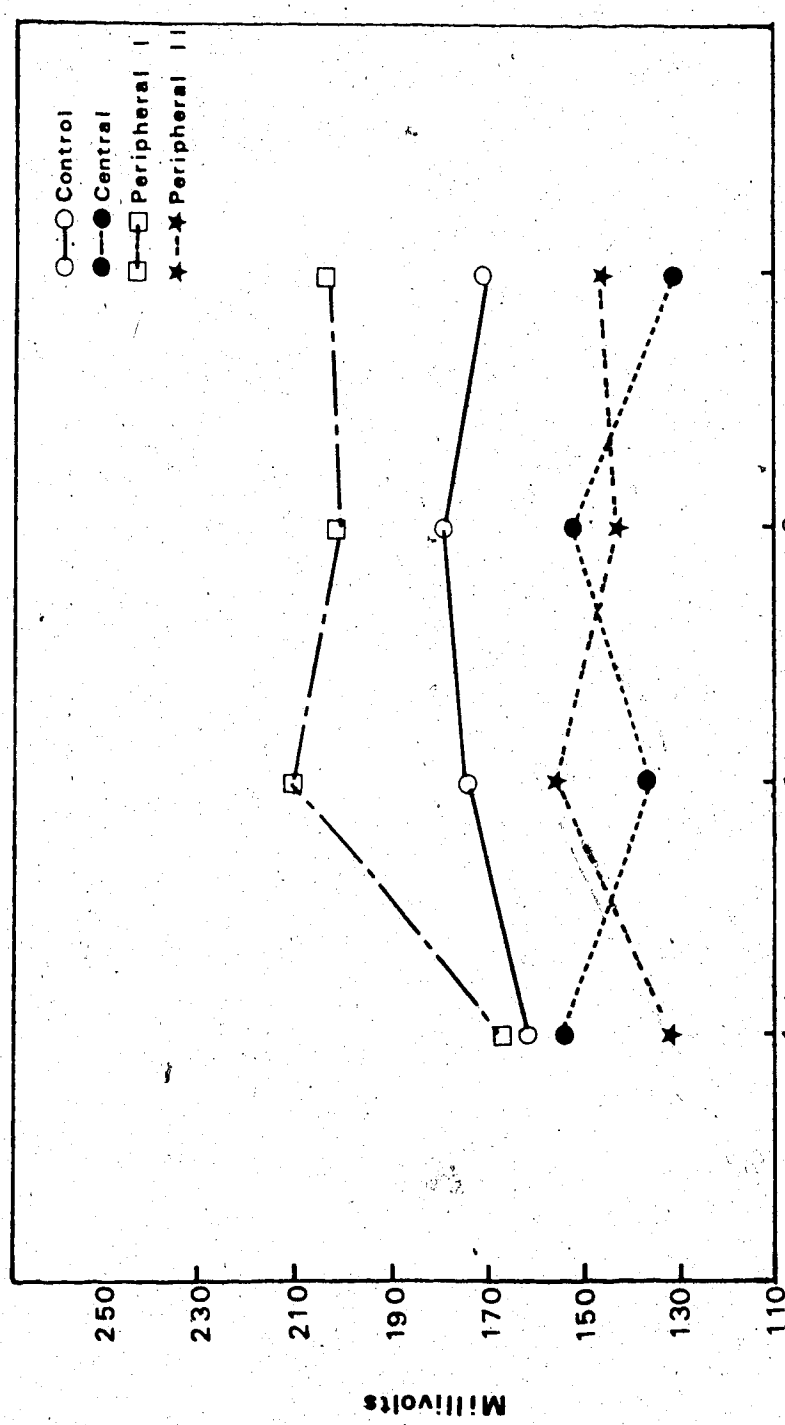

Figure 7. Integrated electromyographic data. Eccentric contractions.

Comparison between the IEMG values for the four work phases (Figures 6 and 7) and torque (Figures 2 to 5) provided tentative support for the findings of Lippold (1952) who stated that as tension within the muscle developed, there was an increase in the electrical activity. In this current study, it was shown that as fatigue set in as manifested by declining torque values, the electrical events within the muscle did not show a parallel drop. Interestingly, there was no uniform increase in electrical activity as perhaps may have been expected. While there was a relatively systematic drop in torque as a function of repeated maximal contractions, the accumulated action potentials responded much less systematically.

Lactic Acid Assays

Figure 8 illustrates the lactic acid values for the four groups expressed as means and standard deviations. The values illustrated are rest, peak and recovery, a post work value. The latter two values indicated the high intensity of the muscle group involved, with the recovery L.A. reflecting a total turn-overrate of lactate within the body.

Reference to Figure 1 indicated that in between the resting blood withdrawal and the second blood withdrawal to determine a peak lactic acid value, the subjects completed three phases of concentric-eccentric work to 50 percent of their initial maximal voluntary concentric contraction. They had also completed two treatment periods, each of four minutes' duration. The recovery lactic acid value was derived from blood withdrawn following a five-minute rest at the completion of the fourth work phase (see Appendix B).

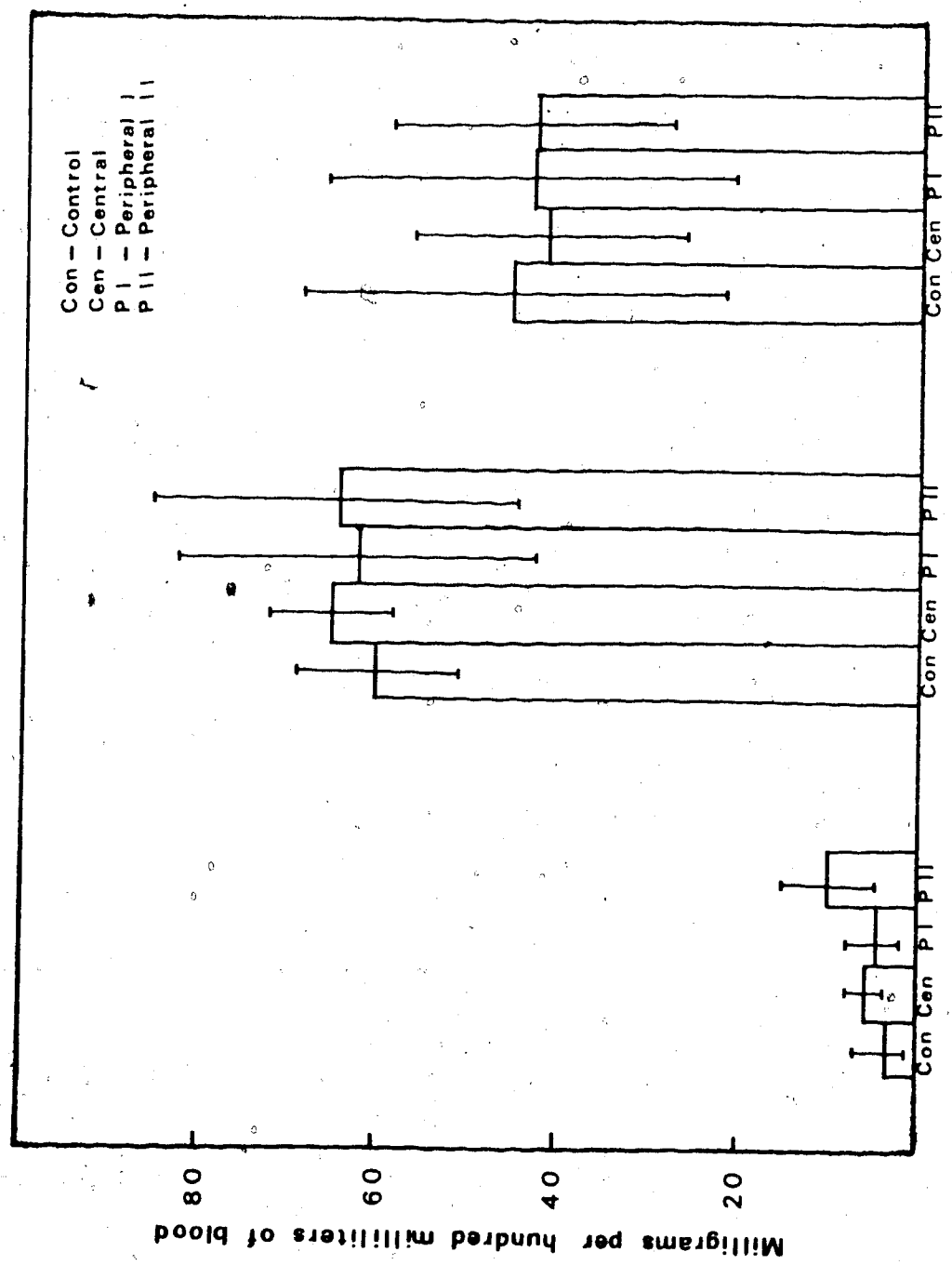

Figure 8. Mean, standard deviation for lactic acid.

It is thought that the resting mean of Peripheral II was large as one group member appeared not to have complied with the requirements of the study not to engage in any heavy physical activity prior to the test being carried out.

The peak lactic acid values must be considered high for short-term, small muscle group work. The blood sampling of the working arm immediately after exercise of maximal concentric and eccentric nature has no doubt contributed to these high values.

There was no significant difference for peak, or post work lactic acid values when the group means were examined; however, there appeared to be a large variation within each group which may relate to the differential utilization of lactic acid by individuals with each group (Belcastro and Bonen, 1975). This does not support Asmussen's (1979) claim that exercise in non-fatigued muscles will stimulate the blood flow through the fatigued muscle group and enhance repeated work measures.

Chapter 5

DISCUSSION

The protocol used in this study (illustrated in Figure 1) indicated that the work involved in the experimentally induced fatigue was intense, of short duration and isokinetic in nature.

The subjects attempted to maintain maximal torques in continuous concentric and eccentric contractions of the flexors of the lower arm at the elbow. Under these conditions, the decline in torque was rapid. This was also noted by other authors (Doss and Karpovich, 1965; Singh and Karpovich, 1966, 1967; Komi and Buskirk, 1972; Hermansen and Osnes, 1972; Hermansen and Stensvold, 1972). Wenger and Reed (1976), Tesch (1978) and Weltman et al. (1979) suggested that the rapid onset of fatigue due to high intensity, short duration muscular work was accomplished through the failure of anaerobic glycolytic metabolism to maintain the necessary levels of adenosine triphosphate and creatine phosphate (ATP-CP) within the muscle cell.

It is important to establish that the localized muscular activity unique to this study was of a high intensity, anaerobic type. It was an activity which produced 50 percent decline in the torque generated in the concentric contraction of flexors of the elbow in one to two minutes.

The results indicated (Figures 2 to 5) that there was no significant difference in the four phases of work between the four treatment

groups measured by maximal, minimal and mean torques. Hermansen and Stensvold (1972), Belcastro and Bonen (1975) and Weltman et al. (1979) indicated that the effects of fatigue could be alleviated by modifying their experimental subjects between work phase behaviour. This fatigue alleviation was associated with the removal of lactic acid in activity patterns which were largely aerobic in nature. It was an hypothesis of this current study that such might hold true for short-term, localized intense muscular activity. The mean torques generated during the decline to 50 percent between the treatment groups, however, showed no significant difference. It is suggested that the nature of the fatigue inducing work in this study was such to produce high lactate levels but the treatments did not differentially influence lactate removal, as evidenced by subsequent torque output.

Central nervous system facilitation (CNSF) was described as having positive effects on repeated phases of work (Asmussen and Mazin, 1978a and 1978b; Asmussen, 1979). Subjects in the Central group in this current study showed no significantly different torque values for either the concentric or eccentric torque components of each phase of work. Using a simple ergograph which involved the flexors of the forearm at the elbow in repeated contractions, Asmussen (1979) observed and recorded a torque decline. By inserting a series of between work phase mental tasks (or treatments), Asmussen (1979) claimed that his experimental subjects produced greater torques and increased numbers of contractions than did non-CNSF practising controls. Asmussen (1979) claimed further that these results confirmed his hypothesis that the dominant site of fatigue was central and not peripheral.

The experimental design of this current study included a group which employed CNSF as its between work phase treatment condition.

Other aspects of the test were held constant. The CNSF (Central) group demonstrated no significant difference in the number of contractions (see Appendix H) before they declined to 50 percent of their MVCC, nor did they show any significantly different torque values over a Control group and two other groups described as Peripheral I and Peripheral II. Like Asmussen's (1979) subjects, those in the current study fatigued the flexors of the lower arm at the elbow. The difference between the two studies was that subjects in the former employed repeated contractions of a submaximal nature. The contractions were performed until a submaximal weight could not be lifted. The rest interval period was two minutes between 20 bouts of exercise. In contrast, the current study used a modified version of this protocol. Fatigue was established more quickly as the subjects attempted to maintain maximal torques. It also employed a longer period for CNSF of four minutes. It is possible that the results obtained do not conform to the findings described by Asmussen (1979) for the following reasons. Central nervous system facilitation may lose its efficacy as a means of fatigue alleviation if the work bouts are of a high intensity and short duration or if the treatment period of CNSF is longer than two minutes.

The torques produced in the groups Peripheral I and Peripheral II showed no significant difference from those produced in the Control and Central groups. Several authors (Belcastro and Bonen, 1975; Bonen and Belcastro, 1975; Hermansen and Stensvold, 1972; Karlsson et al., 1975, Weltman et al., 1978, 1979) have shown that lactic acid disappearance is associated with improved work performance over repeated bouts of exercise. These workers have employed large muscle group activities such as running on an inclined or horizontal treadmill and pedalling on

a bicycle ergometer to induce elevated lactate levels. They have then employed a submaximal work level of the activity which induced the fatigue during the period of active recovery and noted the improvement in work in a repeated bout of exercise when their active recovery groups were compared with their resting controls. In contrast, one of the unique aspects of this current study was the anaerobic work confined to a localized small muscle group engaged in voluntary repeated maximal contractions. Reference to Figure 8 indicated that all subjects showed a marked increase in peak lactic acid over resting levels. Davies et al. (1968), Bloom et al. (1976) and Weltman et al. (1979) claimed that lactic acid removal was associated with submaximal aerobic activity. Further, Hermansen and Stensvold (1972) maintained that removal of lactic acid may have been associated with increased blood flow through the fatigued muscle. The groups in the current study employed a submaximal recovery work load described as mild - a heart rate of 110-120 beats per minute (bpm) and moderate - 140-150 bpm. The current study did not utilize an active recovery in which the working muscle groups were involved, but rather, the protocol described an active recovery which induced a pronounced effect on heart rate, stroke volume and subsequent cardiac output (Astrand and Rodahl, 1977; DeVries et al., 1974). These treatments had no significant effect upon the torque generated between groups for the four phases of work. It may have been that the active treatment groups pedalling on the bicycle ergometer experienced a shift in blood flow from the fatigued site of the biceps to the exercising muscles of the legs. It is possible that the increased heart

rate causing an increased cardiac output and blood flow did not cause an increase in blood flow to other parts of the body, including the fatigued biceps brachii. And thus there was no significantly reduced lactate level observable when compared to Central and Control groups (Astrand and Rodahl, 1977; DeVries, 1974).

The CNSF treatment condition adopted by the Central group did not have a significant effect upon the IEMG compared to the Control, Peripheral I and Peripheral II. Values derived were subjected to a two-way analysis of variance. No significant treatment effects were found. Asmussen (1979) suggested that if an experiment was conducted in which the mechanical output of contracting muscles was observed with a simultaneous electromyographic recording of the action potentials of the contracting muscles, then a parallel fall in mechanical output and IEMG should be expected. In the current study, a parallel fall in IEMG and torque was not found. Asmussen (1979) also stated that should CNSF induce higher levels of work to be performed in a repeated measures setting, there would be an increased IEMG value which parallelled the increased mechanical output. The current study did not produce evidence to substantiate such an assertion. It is likely that the high intensity anaerobic exercise produced high levels of afferent nervous system feedback to the CNS in the form of sensations of pain and fatigue. CNSF is alleged to cause an inhibition of afferent inflow which has the effect of maintaining a higher level of arousal established via the reticular activating system of the brain than would otherwise be expected in a non-CNSF practising subject. In turn, the subject practising CNSF could be expected to maintain higher levels of work in a repeated measures setting over a control subject. This hypothesis

was not supported by evidence from the current study. No significant differences were found in the Central group practising CNSF as their treatment condition over a Control group and two groups engaged in active recovery. Perhaps an explanation of the failure of the CNSF to influence the work could be found in the localized, high intensity work phases of the current study. Subjective impressions of pain and fatigue were common to all subjects as they attempted unsuccessfully to maintain maximal torques. It seems likely that these feelings either overrode or suppressed the effects which may be attributable to CNSF in a less demanding setting. It still seems possible that in a repeated measures submaximal test, CNSF could be shown to influence work output.

The current study provided evidence that in general, the level of electrical activity did not decline in magnitude proportionally to the decline in torque generated over a series of continuous concentric-eccentric contractions of the flexors of the forearm at the elbow. The non-parallel drop in IEMG recordings may provide tentative evidence that it was failure within the contractile mechanism which resulted in the observed decline in torque. It seems that the muscle cells responsible for the development of the torque applied to the lever arm of the electrical dynamometer were unable to respond to the action potential delivered to the contractile proteins via the sarcolemma. The non-parallel decline of torque and IEMG suggests that a cause may be located within the muscle cell's failure to maintain maximal contractions. What seems to be a less tenable explanation for the torque decline relates to the failure of the neuromuscular transmission mechanism. However, more research is needed to provide evidence to confirm or refute this speculation.

DeVries (1968, 1974), Hermansen (1972), Karlsson et al. (1975), Merton (1954), and Newman et al. (1937), suggested that a principal component in the development of muscular fatigue was a buildup of lactic acid within the muscle cell. It was thought that this by-product of the breakdown of glycogen during muscular activity inhibited the capacity of the contractile proteins to sustain high levels of externally measurable tension. On the other hand, Asmussen (1979) claimed that evidence had accumulated since 1903 which indicted diminished transmission of action potentials as being responsible for the failure observed in sustained muscular activity. This position received support from Scherrer and Bourguignon (1959), Sloan (1965), and more recently from Viitasalo and Komi (1977) and Wood (1979).

It can be noted that fatigue, and the possible mechanism of fatigue has been a question perplexing exercise physiologists for many years. Perhaps one reason for the emergence of the two model theory of fatigue as examined in this current study was the failure by the researchers involved to fully acknowledge the principal of the specificity of the type of muscular activity involved. The current study has not produced evidence which could support either the CNSF or the lactic acid removal theories of fatigue alleviation. Studies which show these theories to have influenced repeated measures work performance have utilized large muscle activities and recoveries in which the muscles previously fatigued were exercised in a submaximal manner. The specific exercise utilized currently was a local muscle group worked from MVCC to 50 percent of MVCC in a sequence of concentric-eccentric contractions in which the torque declined by half in one to two minutes. This was a specific, anaerobic and intense phase of work.

The lack of significant findings in this current study suggested that neither the Central (CNSF) nor the Peripheral (lactate removal) theories of fatigue alleviation contributed more than the between phase rest treatment of the Control group to the levels of torque generated by the four groups. As a result, neither the central nor the peripheral sites were dominant in inducing or alleviating fatigue. Fatigue was clearly observed and measured as a declining torque value and changed patterns of IEMG activity. This suggests that the most likely explanation for these observations was a combination of the reduced capacity for tension development in the contractile mechanism and a changed level of neuromuscular transmission. It again appears likely that the specific type of muscular activity was a contributing factor. In the past, it seems that certain sites of fatigue have been said to be dominant over others when perhaps the specific laboratory treatment undertaken by the experimental subjects was largely ignored, yet it perhaps played a large part in the outcomes of the experiment. It should be noted that in future research, care should be exercised in extrapolating the findings from one experimental setting or design to another if similar results are the desired or expected outcomes for the research on the latter occasion.

Just as Wenger and Reed (1976) described the limiting factors to performance as being numerous, complex and interrelated, so too Astrand and Rodahl (1977:115) wrote: "Fatigue is a very complex conception, especially since heavy exercise does load respiration and circulation as well as neuromuscular function." It therefore appears that the mutually exclusive, two model theory was not supported by evidence from this current study because many phenomena interacted to produce

the transient decrease in performance capacity of muscles. As more research is undertaken, it seems likely that the position of Stephens and Taylor (1972) who contended that both central and peripheral factors contributed to the decline in work output will receive wider support.

Chapter 6

CONCLUSIONS AND RECOMMENDATIONS

Conclusions

It was concluded:

(1) That the maximal anaerobic muscular activity prevented the effects of CNSF from influencing the torques generated in a repeated work measures setting.

(2) That active recoveries did not cause any significant difference in the torques generated between the groups.

(3) That intense anaerobic muscular activity taxed both central and peripheral fatigue mechanisms to the extent that neither was observed to be dominant in fatigue alleviation.

The nature of the work was described as high intensity anaerobic which was shown by Hermansen and Osnes (1972), Karlsson and Saltin (1970), Tesch (1978), Tesch, Sjodin and Karlsson (1978), and Wenger and Reed (1976), to be a contraction pattern dominated by fast twitch (FT) fibres. Little information is available on the selective recovery of FT as opposed to slow twitch (ST) fibres. It is not known if these two fibre types have the same recovery pattern. It would therefore appear important to know the fibre type composition of the muscle group and the r[redacted]tterns of the different fibre types in studies exa[redacted]om exhaustive exercise. Buchtal and Schmalbruch (19[redacted]ercent of the fibres of the long head of the

biceps brachii were rich in mitochondria and therefore classified as FT - their study did not include the short head of the biceps brachii. Later work (Tesch, 1978) indicated that FT fibres had a higher anaerobic capacity than the ST fibres. Tesch (1978) also claimed that within the same muscle, FT fibres formed more lactate compared to ST fibres.

It was possible that in the current study the individual fibre types contributed to the non-significant results related to lactic acid production rates. If the Control and Central groups included subjects with lower percentages of FT fibres than Peripheral I and Peripheral II, it would have been possible that their lactic acid values may have not shown as significantly different from those values attributed to Peripheral I and Peripheral II with a higher percentage of FT fibres. The effect of lactic acid production in Peripheral I and Peripheral II could therefore have been masked by lack of specific information to disclose the fibre types of the subjects in the current study.

There appears to be an unfesearched mechanism within the central nervous system which senses when a motor unit is reaching its limit or potential maximum capacity for work. Moreover, it appears that this mechanism can induce new motor units to contract which assists or even takes over from those motor units which have been fatigued. Viitasalo and Komi (1977) and Wood (1979) showed that as a motor unit fatigues and its tension developing capacity falls, so does the action potential innervating that motor unit. Their work suggests that as a motor unit reaches a certain low point of tension development, then it is spared from making further contributions to the work being performed and new

(or other) motor units are activated. The mechanism controlling this synchronization and recruitment is not known and appears to be a potentially productive area for future research.

The concentric-eccentric muscular activity employed in this current study showed a rapid torque decline as the subjects were unable to sustain maximal contractions. The integrated electromyograms (IEMG) illustrated in Figures 6 and 7 did not show a parallel decline with torque generated. Komi (1973) examined the relationship between muscle tension and velocity of contraction under concentric and eccentric work. His work demonstrated that no significant difference occurred in the IEMG between the two types of contraction when performed at maximum. All groups in the current study performed maximal concentric and eccentric contractions. To support the findings by Komi (1973), no clear difference was noted in the electrical activity of the four groups. It was demonstrated, however, that the Control and Central (CNSF) groups showed a slight difference in IEMG response in the concentric phases of work when compared to the treatment groups, Peripheral I (110-120 bpm) and Peripheral II (140-150 bpm). It can be noted in Figure 6 that there was a slight decline in IEMG values for Control and Central from the phase I value to phase IV. On the other hand, it can be noted that there is a slight incline from phases I to IV in the groups Peripheral I and Peripheral II. Although the two-way analysis of variance revealed that the differences described were not significant, the observed differences or trend may be indicative of a treatment effect being present should modifications to the protocol (Figure 1) be made. Further research may reveal that the active recovery treatment in a repeated measures experiment can influence

the IEMG recordings when submaximal torques are studied.

Recommendations

Three major recommendations emanated from the current study.

(1) Information is lacking on fibre typing of the flexors of the lower arm at the elbow. It is recommended that research efforts be mounted to:

(a) develop a needle biopsy technique which could reveal data as to the fibre composition of both heads of the biceps brachii, brachialis, brachioradialis and coracobrachialis muscles, and

(b) reveal whether the fast twitch (FT) and slow twitch (ST) characteristic lactic acid production described by Tesch (1978) holds true for the flexors of the lower arm at the elbow.

(2) The mechanism of motor unit recruitment and synchronization of firing patterns within motor units as exercising muscle fatigues is little understood. It is recommended that research be undertaken to determine the exact nature of this mechanism.

(3) The IEMG data developed in this current study suggested that neuromuscular transmission in fatigued muscles may be enhanced by active recovery treatments in a repeated measures experimental setting. It is recommended that research be undertaken to determine whether this phenomenon occurs in a repeated measures design submaximal work experimental setting.

BIBLIOGRAPHY

BIBLIOGRAPHY

Adrian, E.D., and D.W. Bronk. The discharge of impulses in motor nerve fibres. Part II. The frequency of discharge in reflex and voluntary contractions. Journal of Physiology, 1929, 67, 144-151.

Asmussen, E. Positive and negative muscular work. Acta physiologica Scandinavica, 1953, 28, 364-382.

_____ . Muscular fatigue. Medicine and Science in Sports, 1979, 11(4), 313-321.

_____, and B. Mazin. Recuperation after muscular fatigue by diverting activities. European Journal of Applied Physiology, 1978a, 38, 1-7.

_____, and B. Mazin. A central nervous component in local muscular fatigue. European Journal of Applied Physiology, 1978b, 38, 9-15.

Astrand, P-O., and K. Rodahl. Textbook of work physiology (2nd edition). New York: McGraw-Hill, 1977.

Bang, O. The lactate content of blood during and after muscular exercise in man. Skandinavian Archives of Physiology, 1936, Supplementum 10, 51-82.

Basmajian, J.V. Muscles alive: Their functions revealed by electromyography (2nd edition). Baltimore: Williams and Wilkins Co., 1968.

Bauer, A. Electromyographic fatigue curves. Unpublished Masters Dissertation, The University of Alberta, 1976.

Belcastro, A.N., and A. Bonen. Lactic acid removal rates during controlled and uncontrolled recovery exercise. Journal of Applied Physiology, 1975, 39(6), 932-936.

Bigland, B., and O.C.J. Lippold. The reaction between force, velocity and integrated electrical activity in human males. Journal of Physiology, 1954, 123, 214-224.

Bloom, S.R., R.H. Johnson, D.M. Park, M.J. Rennie, and W.R. Sulaiman. Differences in the metabolic and hormonal response to exercise between racing cyclists and untrained individuals. Journal of Physiology, 1976, 258, 1-18.

Bonen, A., and A. Belcastro. Comparison of self-selected recovery methods in lactic acid removal rates. Medicine and Science in Sports, 1975, 8(3), 176-178.

Bransford, D.R., and E.T. Howley. Effects of training on plasma FFA during exercise in women. European Journal of Applied Physiology, 1979, 41, 151-158.

Bruning, J.L., and B.L. Kintz. Computational handbook of statistics (2nd edition). Glenview, Illinois: Scott Foresman, 1977.

Buchtal, F., and H. Schmalbruch. Spectrum of contraction times of different fibre bundles in the brachial biceps and triceps muscles of man. Nature, 1969, 222, 89.

_____, and H. Schmalbruch. Contraction times and fibre types in intact human muscle. Acta physiologica Scandinavica, 1970, 435-452.

Cavanagh, P.R., and P.V. Komi. Electromechanical delay in human skeletal muscle under concentric and eccentric contractions. European Journal of Applied Physiology, 1979, 42, 159-163.

Clarke, D.H., and G.E. Stelmach. Muscular fatigue and recovery curve parameters at various temperatures. Research Quarterly of American Association of Health, Physical Education and Recreation, 1965, 79, 37(4), 468-479.

Davies, C.T.M., A.V. Knibbs, and J. Musgrave. The rate of lactic acid removal in relation to different baselines of recovery exercise. International Zeitschrift für Angewandte Physiologie einschlafen Arbeitsphysiologie, 1970, 28, 155-161.

DeVries, H.A. Method for evaluation of muscle fatigue and endurance from electromyographic fatigue curves. American Journal of Physical Medicine, 1968, 47(3), 125-135.

_____. Physiology of exercise for physical education and athletics (2nd edition). Dubuque, Iowa: Wm. C. Brown, 1974.

Doss, W.S., and P.V. Karpovich. A comparison of concentric, eccentric and isometric strength of elbow flexors. Journal of Applied Physiology, 1965, 20(2), 351-353.

Eason, R.G. Electromyographic study of local and generalized muscular impairment. Journal of Applied Physiology, 1960, 15(3), 479-482.

Edwards, R.G., and O.C.J. Lippold. The relation between force and integrated electrical activity in fatigued muscle. Journal of Physiology, 1956, 132, 677-681.

Ekstedt, J. Human single muscle fibre action potentials. Acta neurologica Scandinavica, 1964, 61, Supplementum 226.

Elftman, H. Biomechanics of muscle. Journal of Bone and Joint Surgery, 1966, 48-A(2), 363-377.

Essen, B. Studies on the regulation of metabolism in human skeletal muscle using intermittent exercise as an experimental model. Acta physiologica Scandinavica, 1978, Supplementum 454.

Floyd, W.F., and A.T. Welford (Eds.). Fatigue. London: The Ergonomics Research Society, H.K. Lewis and Co., 1953.

Fuglsang-Frederiksen, A., and A. Mansson. Analysis of electrical activity of normal muscle in man at different degrees of voluntary effort. Journal of Neurology, Neurosurgery and Psychiatry, 1975, 38, 683-694.

Gisolfi, C., S. Robinson, and E.S. Turrell. Effects of aerobic work performed during recovery from exhausting work. Journal of Applied Physiology, 1966, 21(6), 1767-1772.

Goodgold, J., and A. Eberstein. Electrodiagnosis of neuromuscular diseases (2nd edition). Baltimore: Williams and Wilkins Co., 1977.

Grossman, W.I., and H. Weiner. Some factors affecting the reliability of surface electromyography. Psychosomatic Medicine, 1966, 28, 78-83.

Guyton, A.C. Textbook of medical physiology (4th edition). Philadelphia: W.B. Saunders Co., 1971.

Hermansen, L. Lactate production during exercise. In B. Pernow and B. Saltin (Eds.), Muscle metabolism during exercise. New York: Plenum Press, 1971.

_____, and J.B. Osnes. Blood and muscle pH after maximal exercise in man. Journal of Applied Physiology, 1972, 32, 304-308.

_____, and I. Stensvold. Production and removal of lactate during exercise in man. Acta physiologica Scandinavica, 1972, 86, 191-201.

Hinson, M., and J. Rosentswieg. Comparative electromyographic values of isometric, isotonic and isokinetic contractions. Research Quarterly of American Association of Health, Physical Education and Recreation, 1965, 37(4), 468-479.

Jervell, O. Investigation of the concentration of lactic acid in blood and urine under physiological and pathological condition. Acta medica Scandinavica, 1928, 5, Supplementum 24.

Johnson, B.L., J.W. Adamczyk, K.O. Tennøe, and S.B. Strømme. A comparison of concentric and eccentric muscle training. Medicine and Science in Sports, 1976, 8(1), 35-38.

Karlsson, J., C. Funderburk, B. Essen, and A.R. Lind. Constituents of human muscle in isometric fatigue. Journal of Applied Physiology, 1975, 38(2), 208-211.

_____, B. Hulten, and B. Sjodin. Substrate activation and product inhibition of LDH activity in human skeletal muscle. Acta physiologica Scandinavica, 1974, 92, 21-26.

Karlsson, J., and B. Saltin. Lactate, ATP and CP in working muscles during exhaustive exercise in man. Journal of Applied Physiology, 1970, 29(5), 598-602.

Komi, P.V. Relationship between muscle tension, EMG, and velocity of contraction under concentric and eccentric work. In J.E. Desmedt (Ed.), New developments in electromyography and clinical neurophysiology (Volume I). Karger: Basel, 1973.

_____, and E.R. Buskirk. Reproductibility of electromyographic measurements with inserted wire electrodes and surface electrodes. Electromyography, 1970, 10(4), 357-367.

_____, and E.R. Buskirk. Effect of eccentric and concentric muscle conditioning on tension and electrical activity of human muscle. Ergonomics, 1972, 15(4), 417-434.

Lind, A.R. Muscle fatigue and recovery from fatigue induced by sustained contractions. Journal of Physiology, 1959, 127, 162-171.

Lippold, O.C.J. The relation between integrated action potentials in a human muscle and it's isometric tension. Journal of Physiology, 1952, 117, 492-499.

_____, J.W.T. Redfern, and J. Vuco. The electromyography of fatigue. Ergonomics, 1959, 3, 121-131.

Maton, B. Fast and slow motor units: Their recruitment for tonic and phasic contraction in normal man. European Journal of Applied Physiology, 1980, 43, 45-55.

_____, and S. Bouisset. The distribution of activity among the muscles of a single group during isometric contraction. European Journal of Applied Physiology, 1977, 37, 101-109.

Merton, P.A. Voluntary strength and fatigue. Journal of Physiology, 1954, 123, 553-564.

Milner-Brown, H.S., and R.B. Stein. The relation between the surface electromyogram and muscular force. Journal of Physiology, 1975, 246, 549-569.

_____, R.B. Stein, and R. Yemm. The contractile properties of human motor units during voluntary isometric contractions. Journal of Physiology, 1973a, 228, 285-306.

_____, R.B. Stein, and R. Yemm. The orderly recruitment of human motor units during voluntary isometric contractions. Journal of Physiology, 1973b, 230, 359-370.

Nelson, A.J., M. Moffroid, and R. Whipple. The relationship of integrated electromyographic discharge to isokinetic contractions. In J.E. Desmedt (Ed.), New developments in electromyography and clinical neurophysiology (Volume I). Karger: Basel, 1973.

Newman, E.V., D.B. Dill, H.T. Edwards, and F.A. Webster. The rate of lactic acid removal in exercise. American Journal of Physiology, 1937, 118, 457-461.

Roundy, E.S., and L.D. Cooney. Effectiveness of rest, abdominal cold packs and cold showers in relieving fatigue. Research Quarterly of American Association of Health, Physical Education and Recreation, 1967, 39(3), 690-695.

Rowell, L.B., K.K. Kraning II, T.O. Evans, J.W. Kennedy, and F. Kusumi. Splanchnic removal of lactate and pyruvate during prolonged exercise in man. Journal of Applied Physiology, 1966, 21(6), 1773-1783.

Sherrer, J., and A. Bourguignon. Changes in the electromyogram produced by fatigue in man. American Journal of Physical Medicine, 1959, 38, 148-157.

Singh, M., and P.V. Karpovich. Isotonic and isometric forces of forearm flexors and extensors. Journal of Applied Physiology, 1966, 21(4), 1435-1437.

_____, and P.V. Karpovich. Effect of eccentric training of agonists on antagonistic muscles. Journal of Applied Physiology, 1967, 23, 742-745.

Sloan, W.A. Electromyography during fatigue of the healthy rectus femoris. South African Medical Journal, 1965 (May), 395-396.

Smorto, M.P., and J.P. Basmajian. Electrodiagnosis - a handbook for neurologists. New York: Harper and Row, 1977.

Stephens, J.A., and A. Taylor. Fatigue of maintained voluntary muscle contraction in man. Journal of Physiology, 1972, 220, 1-18.

Taylor, A. The significance of grouping motor unit activity. Journal of Physiology, 1962, 162, 259-269.

Tesch, P. Local lactate and exhaustion. Acta physiologica Scandinavica, 1978, 104, 373-374.

_____, B. Sjodin and J. Karlsson. Relationship between lactate accumulation, LDH activity, LDH isozyme and fibre type distribution in human skeletal muscle. Acta physiologica Scandinavica, 1978, 103, 40-46.

Viitasalo, J.H.T., and P.V. Komi. Signal characteristics of EMG during fatigue. European Journal of Applied Physiology, 1977, 37, 111-121.

Weibe, P.N. Patterns of muscular fatigue during repeated maximal concentric and eccentric contractions. Unpublished Masters Dissertation, The University of Alberta, 1976.

Weltman, A., B.A. Stamford, and C. Fulco. Recovery from maximal effort exercise, lactate disappearance and subsequent performance. Journal of Applied Physiology, 1979, 47(4), 677-682.

_____, A., B.A. Stamford, R.J. Moffatt, and V.L. Katch. Exercise recovery, lactate removal and subsequent high intensity exercise performance. Research Quarterly of American Association of Health, Physical Education and Recreation, 1978, 48(4), 786-796.

Wenger, H.A., and A.T. Reed. Metabolic factors associated with muscular fatigue during aerobic and anaerobic work. Canadian Journal of Applied Sports Science, 1976, 1, 43-47.

Winer, B.J. Statistical principles in experimental design (2nd edition). New York: McGraw-Hill, 1971.

Wood, G.A. Electromyographical correlates of local muscular fatigue effects upon human visual reaction time. European Journal of Applied Physiology, 1979, 41, 247-257.

APPENDICES

APPENDIX A

Anthropometric Measures of Experimental Subjects in Control, Central, Peripheral I and Peripheral II Groups

Anthropometric Measures of Experimental Subjects in Control, Central, Peripheral I and Peripheral II Groups

Subject	Age Years	Height Centimetres	Weight Kilograms	Upper Arm Length[1] Centimetres	Lower Arm Length[2] Centimetres	Total Arm Lenghth[3] Centimetres
CONTROL GROUP						
1	24.7	170.2	72.2	33.0	26.9	59.9
2	23.9	167.2	61.2	30.6	24.5	55.1
3	22.6	169.6	85.8	33.0	25.7	58.1
4	20.6	170.9	73.0	34.2	27.3	61.5
5	23.4	189.8	84.3	35.5	30.4	65.9
6	23.2	173.9	70.2	34.3	26.9	61.2
7	21.7	157.4	63.1	29.4	24.5	53.9
8	22.5	178.4	66.5	32.6	27.9	60.4
9	32.6	177.0	82.5	34.3	26.3	60.6
	$\bar{X}$ = 23.4 SD = 3.4 R = 20.6 - 32.6	$\bar{X}$ = 172.7 SD = 8.8 R = 157.4 - 189.8	$\bar{X}$ = 73.2 SD = 9.14 R = 61.2 - 85.8	$\bar{X}$ = 32.9 SD = 1.93 R = 29.4 - 35.5	$\bar{X}$ = 26.7 SD = 1.81 R = 24.5 - 30.4	$\bar{X}$ = 59.6 SD = 3.58 R = 53.9 - 65.9
CENTRAL GROUP						
10	21.0	193.0	93.2	37.5	30.4	67.9
11	22.5	168.9	64.9	31.5	26.0	57.5
12	27.7	173.9	74.0	32.3	27.9	60.2
13	23.3	177.0	78.6	34.9	28.7	63.6
14	22.9	176.5	70.6	32.3	26.0	58.3
15	21.1	168.2	62.1	32.3	26.0	58.3

Subject	Age Years	Height Centimetres	Weight Kilograms	Upper Arm Length[1] Centimetres	Lower Arm Length[2] Centimetres	Total Arm Length[3] Centimetres
16	25.9	183.1	88.0	35.5	29.4	64.9
17	27.1	188.7	71.9	35.5	28.7	64.2
19	26.2	169.6	77.0	30.0	26.3	56.3
	$\bar{X}$ = 24.18 SD = 2.56 R = 21.0 - 27.7	$\bar{X}$ = 177.65 SD = 8.89 R = 168.2 - 188.6	$\bar{X}$ = 75.58 SD = 10.07 R = 62.1 - 93.2	$\bar{X}$ = 33.52 SD = 2.39 R = 30.0 - 37.46	$\bar{X}$ = 27.71 SD = 1.69 R = 26.0 - 30.4	$\bar{X}$ = 61.24 SD = 3.87 R = 56.3 - 67.86
			PERIPHERAL I GROUP			
20	25.4	179.0	73.7	34.2	27.9	62.1
21	19.0	172.1	84.4	33.0	26.9	59.9
22	23.6	173.9	63.5	33.0	26.9	59.9
23	27.6	179.7	74.5	34.9	29.2	64.1
24	26.6	178.4	85.1	31.7	29.2	60.9
25	24.9	164.4	73.6	31.1	25.4	56.5
26	21.8	175.2	72.0	34.9	27.3	62.2
27	20.9	180.0	75.4	35.5	28.1	63.6
28	23.3	175.2	77.5	33.0	27.3	60.3
	$\bar{X}$ = 23.5 SD = 2.77 R = 19.0 - 27.6	$\bar{X}$ = 175.3 SD = 4.9 R = 164.4 - 180.0	$\bar{X}$ = 75.4 SD = 6.5 R = 63.5 - 85.1	$\bar{X}$ = 33.4 SD = 1.5 R = 31.1 - 35.5	$\bar{X}$ = 29.5 SD = 1.19 R = 25.4 - 29.2	$\bar{X}$ = 61.0 SD = 2.29 R = 56.5 - 63.6

Subject	Age Years	Height Centimetres	Weight Kilograms	Upper Arm Length[1] Centimetres	Lower Arm Length[2] Centimetres	Total Arm Length[3] Centimetres
			PERIPHERAL II GROUP			
29	26.7	170.8	83.1	36.8	29.2	66.0
30	30.1	169.0	73.0	31.8	26.9	58.7
31	23.9	180.6	90.8	33.0	28.1	61.1
32	35.3	156.8	71.5	33.0	25.7	58.7
33	27.3	163.5	60.8	33.0	25.7	58.7
34	30.0	175.2	68.6	31.1	26.0	57.1
35	23.2	175.2	79.7	32.3	27.3	59.6
36	31.0	172.7	77.5	33.0	28.1	61.1
38	33.0	173.3	71.5	32.4	26.0	58.4
	$\bar{X}$ = 28.9	$\bar{X}$ = 170.78	$\bar{X}$ = 75.16	$\bar{X}$ = 29.7	$\bar{X}$ = 27.0	$\bar{X}$ = 59.9
	SD = 4.02	SD = 7.04	SD = 8.74	SD = 1.59	SD = 1.26	SD = 2.61
	R = 23.2 - 35.3	R = 156.0 - 180.6	R = 60.8 - 83.1	R = 31.1 - 36.8	R = 26.0 - 28.1	R = 58.4 - 66.0

[1] Found by palpating acromion process of scapular and measuring to lateral epicondyle of humerus.
[2] Found by palpating lateral epicondyle of humerus and measuring to distal head of radius.
[3] Determined by adding upper and lower arm lengths.

APPENDIX B

Lactic Acid Values (Milligrams/100 Millilitres of Blood) for Control, Central, Peripheral I and Peripheral II Groups

Lactic Acid Values (Milligrams/100 Millilitres of Blood) for Control, Central, Peripheral I and Peripheral II Groups

$\bar{X}$ = Mean Score SD = Standard Deviation R = Range

Subject	Rest	Peak	Recovery
	CONTROL GROUP		
1	3.5	91.7	51.8
2	1.3	84.0	80.5
3	---	35.0	23.1
4	4.3	64.4	35.7
5	4.2	35.5	33.6
6	---	31.6	12.3
7	2.1	103.6	80.5
8	2.8	51.1	37.8
9	9.8	52.8	54.6
	$\bar{X}$ = 3.99 SD = 2.7 R = 1.26 - 9.8	$\bar{X}$ = 60.61 SD = 8.8 R = 31.5 - 103.6	$\bar{X}$ = 45.5 SD = 23.65 R = 12.25 - 80.5
	CENTRAL GROUP		
10	7.0	84.0	50.4
11	---	101.5	49.0
12	2.1	43.8	17.5
13	5.6	63.0	36.4
14	5.3	57.8	44.8
15	---	47.6	26.6
16	---	101.5	59.5
17	6.9	43.4	26.6
18	8.4	49.0	63.0
	$\bar{X}$ = 5.92 SD = 2.1 R = 2.1 - 8.4	$\bar{X}$ = 65.72 SD = 7.5 R = 43.75 - 101.5	$\bar{X}$ = 41.5 SD = 15.7 R = 17.5 - 63.0

(continued)

Subject	Rest	Peak	Recovery
	PERIPHERAL I GROUP		
19	1.4	94.5	57.4
20	9.8	80.5	52.5
21	9.8	58.1	10.5
22	1.8	38.5	31.5
23	5.6	37.8	51.1
24	2.1	82.6	88.2
25	---	52.5	9.1
26	2.2	66.5	65.8
27	6.2	52.5	26.6
	$\bar{X}$ = 4.86 SD = 3.52 R = 1.4 - 9.8	$\bar{X}$ = 62.6 SD = 19.9 R = 37.8 - 94.5	$\bar{X}$ = 43.62 SD = 26.3 R = 9.1 - 88.2
	PERIPHERAL II GROUP		
28	21.0	76.4	70.0
29	24.6	85.4	38.5
30	3.5	36.4	39.9
31	3.4	59.5	41.3
32	17.5	52.5	40.6
33	7.0	58.1	---
34	9.1	40.6	24.5
35	1.8	73.5	54.6
36	19.6	100.8	82.6
	$\bar{X}$ = 9.75 SD = 5.9 R = 1.75 - 24.5	$\bar{X}$ = 64.81 SD = 21.1 R = 36.4 - 100.8	$\bar{X}$ = 43.5 SD = 17.76 R = 24.5 - 82.6

APPENDIX C

Summaries of Analyses of Variance

Summaries of Analyses of Variance

Source of Variation	SS	DF	MS	F	p
FIRST FOUR ECCENTRIC CONTRACTIONS					
Between Subjects	2920.883	35			
Group main effects	145.477	3	48.492	0.559	0.64588
Subjects within groups	2775.414	32	86.732		
Within Subjects	1119.227	108			
Treatment main effects	706.466	3	235.489	67.668	0.00000
Group*Treatment interaction	78.609	9	8.734	2.510	0.01263
Treatment x Subjects within groups	334.086	96	3.480		
MEAN CONCENTRIC PLUS MEAN ECCENTRIC CONTRACTIONS					
Between Subjects	557.090	35			
Group main effects	23.262	3	7.754	0.465	0.70884
Subjects within groups	533.823	32	16.682		
Within Subjects	157.840	108			
Treatment main effects	96.519	3	32.173	61.782	0.00000
Group*Treatment interaction	11.344	9	1.260	2.421	0.01599
Treatment x Subjects within groups	49.992	96	0.521		

(continued)

Source of Variation	SS	DF	MS	F	p
MEAN ECCENTRIC TORQUE					
Between Subjects	146.446	35			
Group main effects	7.755	3	2.585	0.596	0.62198
Subjects within groups	138.689	32	4.334		
Within Subjects	55.553	108			
Treatment main effects	33.575	3	11.192	60.787	0.00000
Group*Treatment interaction	4.304	9	0.478	2.597	0.01002
IEMG CONCENTRIC: FIRST FOUR CONTRACTIONS SUMMED					
Between Subjects	652076.000	35			
Group main effects	80465.188	3	26821.727	1.502	0.23290
Subjects within groups	571613.000	32	17862.906		
Within Subjects	66505.000	108			
Treatment main effects	9158.074	3	3052.691	5.603	0.00139
Group*Treatment interaction	5040.570	9	560.063	1.028	0.42369
Treatment x Subjects within groups	52307.000	96	544.865		

APPENDIX D

Honeywell Medical System Recording Unit

Honeywell Medical System Recording Unit

An electromyographic recording was made during the four concentric-eccentric phases of the test for all subjects.

The surface electrodes monitored the muscle action potentials generated during each movement of the concentric-eccentric phases. In doing so, data was compiled related to both the concentric and eccentric muscular contractions performed by all subjects.

The surface electrodes were connected via colour-coded alligator clips (AMI Co.) and shielded cables, to an isolation amplifier (Terrasyn Inc. Model NIIIA EMG). Next, the electromyographic potentials were fed into an electrically operated integrator, equipped for full wave rectification.

The system used to record results was the Honeywell Electronic Medical System. Four channels were used in this study.

Channel 1. An EMG channel provided a clinically interpretable record of electrical activity in the muscle.

Channel 2. An integrator channel electronically collected the EMG output and integrated the electrical muscle potential. This output was transformed to a visual reading on a multichannel oscilloscope and a paper reading from an oscillograph. The integrator also worked as an area summator for the total electromyographical activity in the EMG channel and produced an output proportional to the area of the EMG wave form. The integrator quantified the muscular electrical potentials of the fatiguing muscles, which enabled an assessment of work performed to be made.

Channel 3. A strain gauge channel simultaneously measured and

recorded the muscle tension developed. This channel was linked via the appropriate wiring, to a Beckman Recorder. The strain gauge was located in the electrical dynamometer lever arm close to the pivotal point which was also the location of the subject's elbow. An elbow pad at this point was provided to assist in the comfort of each subject and to standardize the position of force application. Each subject was instructed to "feel" the point of his elbow to coincide with the back of the elbow pad and to keep the elbow in this position throughout the four concentric-eccentric phases. The lever arm had a softly padded moveable wrist support which enabled an individual adjustment of the lever arm for all subjects.

Channel 4. An integrator of the strain gauge. The recording indicated the summated and integrated electrical impulses from the strain gauge, which recognized the variable angles of the activity and the variable muscular tension developed at those angles. The total force generated during the successive concentric and eccentric contractions was recorded on the fourth channel.

The four channels were simultaneously fed into a Model 1912 Visicorder Oscillograph. The oscillograph uses a high intensity mercury vapour lamp to provide an ultra violet light source which recorded on ultra violet light sensitive paper (Kodak Direct Print Extra Thin {type 2022} Linagraph paper).

APPENDIX E

Calibration of Electrical Dynamometer and EMG

Calibration of Electrical Dynamometer and EMG

Electrical Dynamometer

The lever arm was calibrated according to the technique of Singh and Karpovich (1965). Known weights (Weider Barbell Co.), which had been checked for accuracy by weighing on a lever balance scale (Health-o-Meter, Continental Scale Corp., Ill., USA) were placed on the lever arm 12 inches from the axis of rotation, and a strain gauge recording was made on the Beckman pen recorder. Figures derived in this manner were considered to indicate forces in foot/pound units. Conversion to kilograms/meters was later made.

EMG

The Terrasyn isolation amplifier emits a calibration impulse of five millivolts. Prior to each test, the EMG amplifier was set to ensure that the gain control caused a consistent ultra violet light deflection in the Visicorder in response to the calibration impulse.

APPENDIX F

EMG and Quinton Model 650 Electrode Placement

EMG and Quinton Model 650 Electrode Placement

EMG Electrode Placement

To ensure the highest quality signal possible, it was necessary to remove any dead skin and surface oils by rubbing the appropriate site with an Electrodyne Co. disposable skin cleaner swab. This preparation provided both low skin resistance and low skin potential.

The sites for the three EMG electrodes were as follows:

(1) The search electrode was placed on the mid-line of the anterior aspect of the muscle belly of the muscle biceps brachii with the subject standing in the anatomic position.

(2) The reference electrode was placed five centimetres distally and in line with the search electrode.

(3) The third electrode was placed on the acromion process of the scapular, to act as an earth electrode.

The surface electrodes used were Electrodyne Co. disposable silver-silver chloride of 5 mm diameter. Although not recommended for use on more than one occasion, a pilot study revealed identical EMG traces when the same electrodes were used on a second occasion. The electrodes therefore were used more than once. On successive uses, the pre-gelled pad of foam rubber of approximately 1 mm thickness was re-gelled, using Harco electro-conductive gel (HAR 154-B). This was done to permit maximum conductivity between the skin and electrode.

The most suitable form of adhering the reused disposable electrodes was to remove the surrounding adhesive pad of the electrode and then cut a small hole in a rectangle of Johnson and Johnson Elastic adhesive tape of approximately 5 cm x 3 cm. The back stub of the electrode was then placed through the hole and both were firmly pressed onto the prepared skin site. When connected to the AMI alligator clips

and shielded wiring which transmitted the muscle action potentials to the amplification equipment, the results were entirely satisfactory. In this manner, 12 sets of disposable electrodes were used three times.

Quinton Model 650 Electrode Placement

Two groups, Peripheral I and Peripheral II, had their heart rates monitored at 110-120 beats per minute (bpm) and 140-150 bpm respectively during their treatment periods. The monitoring device was a Quinton Model 650 Heart Rate Meter, which gave a digital heart rate reading every ten seconds of the four minutes' pedalling. The three electrodes were Quinton silver-silver chloride discs of approximately ten millimetres diameter, designed for use exclusively with this heart rate meter. These were placed in the appropriate positions on the left, sternal and right anterior chest wall in a horizontal line approximately ten centimetres below the horizontal line of the nipples.

APPENDIX G

Lactic Acid Assay Technique

Lactic Acid Assay Technique

The samples were prepared in the following way. After withdrawal, 0.1 ml of blood was added to 0.4 ml of ice cold perchloric acid and stirred on a vortex mixer and centrifuged at 3,000 revolutions per minute on an International Centrifuge. This caused the hemolysed red blood cells and white blood cells to form a pellet in the bottom of the test tube leaving the supernatant ready for withdrawal. The supernatant was pipetted into a clean test tube and frozen at -60°C. According to Mohme-Lundholme (1965), it was possible to find reliable lactic acid results up to one month following initial freezing of the supernatant. In this experiment, all samples were assayed between six and 18 days following blood withdrawal.

In preparation for the spectrophotometric reading, the test tubes containing the supernatant were removed from the freezer and allowed to come to room temperature while free standing in racks.

The supernatant was added to the following mixture. Test tubes had been prepared by the addition of 2.0 ml glycine buffer (Sigma Drug Co., No. 826.3), 0.4 ml LDH (Sigma Drug Co., No. 826.6), 0.2 ml NAD (Sigma Drug Co., No. N-7004).

The mixture was incubated for one hour (60 minutes) at 25°C and then read in the Pye Spectrophotometer at 340 nanometer wave length. This step required the following procedures:

(a) The spectrophotometer was zeroed with an air reading.

(b) Two equal cuvettes, the mixture containers, were selected. This was necessary so that any difference between readings could be attributed to the contents and not the cuvettes.

(c) The cuvette containing the mixture of supernatant, buffer and enzymes had to be read 'against' a cuvette containing buffer and enzymes. This second cuvette was known as a blank. Reading against a blank enabled the concentration of LDH to be determined and by its causal relationship with lactic acid (LA), the lactic acid level to be established.

To ensure that the levels of NADH revealed by the spectrophotometer were true reflections of levels of the lactic acid in the subject's blood at the time of withdrawal, each sample for each subject was assayed simultaneously three times. One sample of three was referred to as a triplicate.

A reading of 0.002 on the spectrophotometer corresponded to approximately one milligram of lactic acid for every 100 millilitres of blood when placed on a standard curve and converted to lactic acid concentration. This value was established as the criterion for acceptance or rejection of the particular triplicate. In other words, if two of the three values in the triplicate sample did not fall within this range, it was rejected and the sample was re-assayed in triplicate. Such a rigorous acceptance-rejection criterion led to a rejection of 26 first-run assays.

After the second-run assays were done, the data was then compared to a standard curve to reveal the $\mathring{A}$ reading of light absorbance or lactic acid presence. This value was then multiplied by 70 to correct for the factor of dilution and to reveal lactic acid values in milligram per hundred millilitres of blood (Mg %).

APPENDIX H

Concentric-Eccentric Contractions to 50 Percent MVCC

The following table illustrates the individual and group values related to the number of contractions taken before the subject reached the termination point for each phase of work which was 50 percent of his maximal voluntary concentric contraction.

CONTROL GROUP

Subject	Phase 1	Phase 2	Phase 3	Phase 4
1	9	6	6	7
2	11	7	6	6
3	9	6	7	6
4	8	8	8	10
5	12	11	6	7
6	10	8	7	8
7	7	6	5	4
8	6	6	6	9
9	7	7	4	6
Mean	8.7	7.2	6.1	7.0

CENTRAL GROUP

10	8	5	6	5
11	7	10	7	6
12	9	7	6	6
13	6	10	8	7
14	6	7	6	7
15	8	9	9	11
16	8	8	8	8
17	5	7	5	9
18	5	4	6	7
Mean	6.8	6.8	6.7	7.5

PERIPHERAL I

19	7	5	6	7
20	6	7	6	7
21	8	10	10	11
22	9	6	7	6
23	6	7	7	7
24	7	7	5	6

PERIPHERAL I (continued)

Subject	Phase 1	Phase 2	Phase 3	Phase 4
25	10	5	7	4
26	7	9	8	10
27	5	12	10	11
Mean	7.2	7.5	7.3	7.6

PERIPHERAL II

28	6	6	5	5
29	8	9	7	7
30	8	7	6	5
31	6	4	5	5
32	9	8	8	8
33	12	10	9	10
34	10	8	8	9
35	10	10	11	10
36	12	8	8	8
Mean	9.6	7.7	7.4	7.4

APPENDIX I

"Informed Consent" Form

CONSENT TO UNDERGO TESTING

I, _____, hereby agree to volunteer in a study to determine the influence of rest interval activities on fatigue patterns of the flexors of the forearm. I understand that I will,

a) Perform maximal flexions and extensions of the forearm until a point of 50% of my MVC is reached. This will be done four (4) times with rest intervals of four minutes between each work bout.

b) Be required to undergo blood sampling three times:

i) at the commencement of the test,

ii) after the third bout of flexion-extension to 50% of MVC;

iii) five minutes after the completion of the fourth bout of flexion-extension.

I understand that with any type of exercise there are potential risks and have been fully informed of the nature of the tests involved. If at any time during the test I experience any severe or unusual discomfort, I will ask to discontinue the test.

In agreeing to such an examination, I waive any legal recourse against The University of Alberta from any and all claims resulting from this test.

Date _____ Subject _____

Witness _____ (Signature) _____

APPENDIX J

Central Group Mathematics Problems

"CENTRAL" REST INTERVAL ACTIVITY GROUP

Mathematic Calculations - Sheet 1

Please complete as many of the following problems as you can.

ADDITIONS

(i)	764	(ii)	1862	(iii)	5921	(iv)	7186	(v)	99874
	820		9374		7435		5909		21721
	374		8107		8162		8874		19536

SUBTRACTIONS

(i)	68214	(ii)	7482	(iii)	99411	(iv)	8627	(v)	94461
	41926		6997		26322		5737		94461

EQUATIONS

(i) $649 + 32 - (84 \times 3) =$ (ii) $1870 - (136 \times 4 + 515) =$

(iii) $(178 \times 13 + 16 \times 9) - 87 =$ (iv) $791 + 18 + 233 - (15 \times 8 + 99) =$

MULTIPLICATIONS

(i)	447	(ii)	764	(iii)	8671	(iv)	7996	(v)	84117
	59		72		93		82		77

DIVISIONS

(i) $8642 \div 29$ (ii) $714 \div 86$ (iii) $7448 \div 11$ (iv) $9861 \div 27$ (v) $8862 \div 18$

"CENTRAL" REST INTERVAL ACTIVITY GROUP

Mathematic Calculations - Sheet 2

Please complete as many of the following problems as you can.

SUBTRACTIONS

(i)	(ii)	(iii)	(iv)	(v)
9286	6641	7948	99616	37625
7162	5887	5512	27787	28647
____	____	____	____	____

MULTIPLICATIONS

(i)	(ii)	(iii)	(iv)	(v)
2616	35515	17728	54623	86747
14	83	17	245	321
____	____	____	____	____

DIVISIONS

(i) 8416 ÷ 26 (ii) 1338 ÷ 13 (iii) 22645 ÷ 21 (iv) 82961 ÷ 85
(v) 34521 ÷ 23

ADDITIONS

(i)	(ii)	(iii)	(iv)	(v)
41686	93827	78662	634461	74468
63345	74136	93715	792885	59213
____	____	____	____	____

EQUATIONS

(i) 326 + 291 - (42 x 7) = (ii) 1001 - (25 x 14 + 250) =
(iii) (36 x 24 + 26 x 12) - 57 = (iv) 251 + 365 + 254 - (26 x 6 + 17) =

"CENTRAL" REST INTERVAL ACTIVITY GROUP

Mathematic Calculations - Sheet 3

Please complete as many of the following problems as you can.

MULTIPLICATIONS

(i)	916	(ii)	8744	(iii)	7316	(iv)	86927	(v)	71131
	12		19		52		174		297

DIVISIONS

(i) 79829 ÷ 13 (ii) 41486 ÷ 26 (iii) 84936 ÷ 15 (iv) 792114 ÷ 28
(v) 13426 ÷ 33

ADDITIONS

(i)	545621	(ii)	312268	(iii)	918397	(iv)	129854	(v)	619772
	133747		828473		21[illegible]35		613653		619779

EQUATIONS

(i) 289 + 324 - (78 x 4) = (ii) (34 x 46 + 54 x 13) - 67 =
(iii) 875 - (356 x 2) = (iv) (262 x 5 ÷ 351 x 2) - 256 =

SUBTRACTIONS

(i)	7866216	(ii)	824275	(iii)	956714	(iv)	890013	(v)	474861
	4277413		655372		472481		426304		395643