EFFECTS OF DOWNHILL RUNNING ON MOTONEURON POOL EXCITABILITY AS MEASURED BY THE HOFFMANN REFLEX

by

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Dedicated to my Mother and Father,
without whose unconditional love and support
this milepost could not have been reached.

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CHAPTER ONE

INTRODUCTION

Central nervous system responses to exercise have recieved increased attention in the literature. Alterations in neuromuscular states have important implications in both pathophysiology (Pivik, Bylsma and Margittai, 1986; Gandau, Weaver and Hornbein, 1960; Angel and Hoffmann, 1963) and tranquilizing drug replacement (deVries and Adams, 1972). Alterations in neuromuscular state have been shown at both the central (deVries, 1981) and peripheral (deVries and Adams, 1972) level following exercise, utilizing the Hoffmann reflex and electromyography (EMG), respectively. Concentric exercise of both low and high intensity reduces motoneuron pool excitability utilizing the H/M ratio as a dependent measure (Bulbulian and Darabos, 1986). Low intensity exercise (40% VO2 maximum) and high intensity exercise (75% VO2 maximum) reduce the mean H/M ratio by 12.8% and 21.5%, respectively. Patellar reflex threshold and intensity also decline post-exercise (Sato and Takemoto,1972). To date, central nervous system (CNS) responses have been examined only after conventional exercise in which the active muscles mainly function concentrically. Whether eccentric exercise, at similar metabolic demand to concentric exercise, has a different CNS response has yet to be determined. The possible benefits to understanding the central nervous system response to

eccentrically-biased exercise may include exercise prescription for neuromuscular relaxation and insight into the pathophysiology of delayed-onset muscle soreness (DOMS).

Purpose of the Study

The purpose of this study was to examine central nervous system responses to eccentrically-biased exercise as measured by motoneuron pool excitability. Specifically, the H/M ratio was recorded before and after treadmill exercise bouts running at 0% grade and -10% grade (downhill) at a similar metabolic demand.

Understanding the exercise-induced alterations in motoneuron pool excitability may have important pathophysiological and theraputic consequences, especially with respect to DOMS. By expanding our exercise parameters to include eccentric work, we can better understand the factors effecting motoneuron excitability during exercise. Despite differences in mechanical demands (Buczek and Cavanagh, 1987), when relative intensities are matched, metabolic responses are similar between concentric and eccentric work (Knuttgen, 1971).

Mechanical disruption of tissues associated with eccentric work may also produce afferent feedback to the CNS, thus altering motoneuron excitability, and possibly producing a neuromuscular state which potentiates the injury (Karpati, 1974).

Furthermore, neuromuscular suppression may affect the

overall tranquilizing reponse of the individual to exercise since exercise has long been prescribed for stress management by physicians and psychiatrists (Byrd, 1963).

This study attempted to discern the effects of eccentric-biased exercise on motoneuron pool excitability, specifically with respect to eccentric work producing delayed-onset muscle soreness.

Hypotheses

This study proposes the following hypotheses:

1. Non-biased exercise will reduce the H/M ratio.
2. Eccentric-biased exercise will attenuate the reduction in the H/M ratio.
3. The H/M ratio will return to control levels 24 hours post non-biased exercise, but will be elevated with respect to control levels following eccentric-biased work.
4. Sitting quietly will produce no change in the H/M ratio.

CHAPTER TWO

LITERATURE REVIEW

Literature reviewing neuromuscular relaxation and exercise, motoneuron pool excitability and exercise, and physiological mechanisms of motoneuron excitabilty will be discussed. Also, literature concerning eccentric work and delayed-onset muscle soreness will be reviewed.

Neuromuscular Relaxation and Exercise

Physiological measures of exercise-induced neuromuscular relaxation include alterations in resting muscle action potentials (deVries, 1968; deVries, 1972) and spinal motoneuron excitability measured by the H-reflex technique (deVries, 1981; Bulbulian and Darabos, 1986).

Utilizing resting electromyography as the measure of neuromuscular relaxation, deVries (1968) measured right forearm flexor muscle action potentials (MAP's) before and after 5 minutes of bench stepping in 12 males and 17 females. Post-exercise electrical activity decreased 58% while controls showed a non-significant 1.5% decrease. Training effects were shown by exercising 18 males aged 26 to 53 for approximately 6 weeks using heavy resistance exercise combined with a run-walk program. Training resulted in an increased maximum mean VO2 of 4.46 ml/kg/min. Post-training EMG activity decreased an average of 38%. Decreased EMG activity was attributed to changes in proprioceptive feedback patterns.

Comparing exercise and a tranquilizing drug, meprobamate, deVries (1972) again found decreased resting bicep EMG activity following exercise. Muscle electrical activity following 15 minutes of walking at an intensity of 100 beats per minute decreased 20%, 23% and 20% at 30,60 and 90 minutes post-exercise. Neither exercise at 120 beats per minute nor meprobamate showed a significant change.

Not all studies utilizing resting MAP as the dependent measure have found beneficial effects associated with exercise. Recording MAP from the frontalis muscle, Farmer (1978) found no exercise effect in either type A or B young males. This contradictory evidence may be due to the use of the frontalis muscle which is not a muscle whose resting tension is strongly associated with relaxation at rest (Nidever, 1959). The biceps brachii was found to have the highest common tension factor at rest.

The literature would suggest that exercise, both acute and chronic, can reduce the alpha motoneuron discharge to the resting musculature resulting in a decreased resting MAP levels. To more precisely examine neuromuscular changes post-exercise, investigators have turned to examining reflex parameter alterations.

Increased motoneuron discharge may be due to an increased resting potential or excitability. Quantifying the H-reflex has been suggested as a measure of the motoneuron pool excitability (Angel and Hofmann, 1963). First described

by Hoffmann (1922) and later modified by Hugon (1973), the H-reflex results from transcutaneous electrical stimulation of the tibial nerve as it passes through the popliteal fossa. Electrical stimulation results in two distinct MAP's recorded at the soleus via surface electrodes. The first, the M-wave, results from direct depolarization of the alpha motoneurons leading to the soleus. The second, delayed approximately 30 milliseconds, is the H-wave. The H-wave results from electrical stimulation of tibial Ia afferents propagating to the spinal cord and back to the soleus via reflex arc apparatus. H- wave magnitude results from the number of motor units depolarized by the electrically stimulated reflex arc and reflects the algabraic sum of both inhibitory and excitatory influences converging on the motoneuron at that instant (Angel and Hofmann, 1963). The latter is termed motoneuron pool excitability.

Though not purely monosynaptic (Burke, Candevia and McKeon, 1983) and highly variable inter-individually (Crayton and King, 1981), the H-reflex technique can be rendered very stable and useful by expressing its value as a ratio of the maximum H-wave to the maximum M-wave, the H/M ratio. By expressing the H-wave relative to the M-wave, any change in conductivity, electrode shift, nerve movement, or subject position, all of which would effect H-wave amptitude (Angel & Hofmann, 1963; Crayton & King 1981), would also effect the M-Wave similarly, leaving the H/M wave ratio unaltered. Using the H/M ratio as the dependent

measure coupled with it's high intra-individual reproducability (Crayton & King 1981) make it very useful as a tool to investigate changes in motoneuron exitability.

When utilized in conjunction with exercise, the H/M ratio is consistently reduced post-exercise (de Vries, 1981; Bubulian and Darabos, 1986). Testing 10 subjects, two aged 80 and 66 and 8 aged 20-34, de Vries et. al. (1981) showed decreased motoneuron pool excitability following 20 minutes of bicycle ergometer exercise at 60% heart rate range intensity. Post-exercise H/M ratios dropped 18.2% (range 6-44%) which was highly significant ($p < .001$). However, subjects were screened and selected on the basis of high resting EMG (> 2 microvolts) and symptoms of nervous tension; i.e. unremitting worry, irritability, insomnia, restlessness, persistent feelings of tension or feeling of panic in everyday life. As it has been shown that subjects with the highest initial neuromuscular arousal benefit most from exercise (de Vries, 1968) this population may have a more dramatic response than a more normal one.

Utilizing the H/M ratio as a dependent measure, Bubulian and Darabos (1986) report that exercise depression of spinal cord excitability is intensity dependent. Testing ten male and female subjects (ages 20-45 years) before and after 20 minutes of low intensity (40% VO2 max) and high intensity (75% VO2 max) treadmill running, the authors showed a decrease in H/M ratio of 12.8 % and 21.5%,

respectively. H/M ratios differed significantly between intensities and from control (+2.1%) thus, both low and high intensity exercise have been shown to reduce motoneuron pool excitability as determined by the H/M ratio measure in nervous and normal subjects.

Crayton and King (1981) found no relationship between amount of regular excercise and H-reflex parameters. Other investigators (Rochcongar, Dassonville and LeBarn, 1979) have found athletes average values for H max / M max to be not different from non-athletes, (53 %). However, significant differences have been reported between anaerobic athletes (37 %) and aerobic athletes (55-75%). Because they are cross sectional in nature, these studies shed little light on the effect of training on motoneuron excitability. Differences shown may be due to natural selection.

Other reflex response parameters are equivocal. Reports have shown decreased (Macarez and Henane 1970), little change (Henane and Macarez, 1972) or enhanced achilles tendon reflex following exercise (Cheah and Tan, 1970, Mayes, 1975; Johnson, Jokl and Jokl, 1963). At rest, weight trained athletes were found to have shorter pattellar reflex latency time than long distance trained athletes, though achilles reflex time was similar (Kamen,Kroll and Zigon, 1981). Following 3 isometric bouts to fatigue, achilles tendon reflex parameters remained unchanged. Patellar reflex total time and reflex motor time increased for the weight trained athletes, while endurance athletes showed an

increase following the first bout followed by decreases following the second and third bouts. Similar results were found by Sato and Takemoto (1972) for isometric exercise. Isotonic fatigue, however, decreased patellar reflex threshold and intensity. Fractionating reflex time into central and peripheral factors, Kroll (1974) found total reflex time increases post-isotonic exercise due to increases in peripheral motor time and not central factors. Variation in reflex response has been suggested to be due to competing factors of (1) facilitation due to acceleration of muscle spindle activity and (2) suppression due to fatigue (Sato and Takemoto, 1972). Examining total reflex response involves every segment of the reflex loop; stretch receptor, Ia afferent, spinal cord intergration, efferent motoneuron and muscle response. This large number of variables combined with variation in tendon tap force, position and inter-individual variability may account for the discrepancies in the literature.

Motoneuron pool excitability after strength training has been investigated using H-reflex potentiation during maximal voluntary contraction (V1 measurement)(Sale, MacDougall, Upton and McComas, 1983). The V1 MAP is a supramaximal H-wave that appears due to the antidromic stimulus being negated by voluntary efferent activity in the motoneuron allowing the usually suppressed H-wave response to appear (Sale, McComas, and MacDougall, 1982). Strength training

potentiated the V1 response 49.7% for the pooled muscle responses of the extensor digitorum brevis, soleus, brachioradialis and hypothenar muscles. No significant increases occurred in individual muscles and no acute responses were observed (Sale, MacDougall, Upton and McComas, 1983). Increased V1 response was attributed to increased volition drive post-training.

Physiological Mechanisms of Altered Motoneuron Excitability

Physiological mechanisms possibly responsible for exercise-induced changes in motoneuron pool excitability can be categorized as thermal, neural and chemical in origin. Neuromuscular relaxation may result from 1) decreased muscle spindle activity due to increased hypothalamic and total body temperature (Von Euler, 1956) 2) exercise-induced "random and intermittent, constantly changing proprioceptive stimuli allowing normal cortical activity leading to a relaxed state" (Haugen, 1960) and 3) exercise-induced CNS depression due to endogenous opiate release (Morgan and Horstman, 1976).

Measuring electroencephalogram (EEG) activity from motor, temporal, and occipital areas of the cat and rabbit cortex simultaneously with muscle spindle afferents, show an inverse relationship with hypothalamic and total body temperature (Von Euler, 1956). Increased body temperature decreases muscle spindle activity and synchronizes cortical activity. deVries (1968) postulated this as a mechanism for

exercise-induced relaxation effects that stem from two results. Decreased cortical activity reduces alpha-motoneuron stimulation to skeletal muscles and decreased gamma motoneuron bias on the muscle spindle intrafusal fibers decreases reflex muscular activity. The primary motor center responsible for shivering thermogenesis is known to have descending excitatory effects on motoneuron pools in the lateral horns (Hensel, 1973). Increased muscular tone results from a feedback oscillation of muscle spindle stretch reflex mechanisms. Therefore, increases in body temperature via exercise could decrease motoneuron pool excitatory impingment and thereby its excitation level. De-efferentated muscle spindles show increased acceleration sensitivity as temperature decreases (Poppele and Bowman, (1970).Thus, decreasing temperature decreases the Ia afferent stimulation of alpha-motoneurons. Conversely, increased body temperature with exercise may reduce motoneuron excitability.

Neural influences on motoneuron dendritic zones may be divided into excitatory, inhibitory, descending efferent, and peripheral afferent. Peripheral afferent activity upon the motoneuron pool may be either excitatory or inhibitory. Group Ia and II afferent stimulation results in EPSP's in motoneuron dendrites via monosynaptic, myotatic stretch reflex activity (Valbo, 1981). Activity in group Ia muscle spindle afferents from a particular muscle cause

monosynaptic excitation of motoneurons to homonymous and close synergist muscles as well as disynaptic inhibition of antagonist motoneurons (Eccles and Lundberg, 1958). Group Ia inhibition of alpha-motoneurons occurs through presynaptic inhibition. Thus, the Ia EPSP could be decreased by increasing presynaptic inhibition or increased by decreasing the tonic level of presynaptic inhibition (McCrea, 1986). Primary afferent depolarization (PAD) has been shown not to be solely under the influence of other primary afferent interneurons. Other non-specific mechanisms such as increased extracellular potassium are contributors (Jimenez, Slodkin and Rudomin, 1985).

Information about muscle tension is relayed centrally via group Ib afferents arising from Golgi tendon organs. Both excitatory and inhibitory effects on motoneurons have been shown utilizing Ib stimulation (Eccles and Lundberg, 1958), though homonymous inhibition seems the major effect. The motor servo theory of Houk and coworkers (Houk, 1979; Houk, Crago and Rymer, 1981) integrates the effects of Ia and Ib afferent activity on motoneuron excitability into a system for regulating muscle "stiffness". Defined as the ratio of force change to length change, stiffness is postulated as being regulated by the balance of Ib to Ia afferent input sensitivity. Increasing the length feedback gain via spindle bias increases muscle stiffness.

Motoneuron pools also have numerous and complex interneuronal interactions mediating peripheral and

decending influences which are beyond the scope of this review (see McCrea, 1986). Motoneurons are known to recieve both excitatory and inhibitory effects from decending pathways involving the reticulospinal (Peterson, 1979), corticospinal (Jankowska and Tanaka, 1974), rubrospinal (Burke, et. al.,1970) and vestibulospinal (Grillner, Hongo and Lund, 1966) systems sharing Ia inhibitory interneurons.

In summary, motoneuron excitability is the integrated result of all impinging excitatory and inhibitory inputs on the motoneuron dendritic zone.When dealing with reflex pathways, excitabilty depends on 1) the gamma mediated stretch sensitivity of muscle spindles 2) ability of spindle primary endings to transmit afferent input to alpha-motoneurons, which is controlled by spinal interneuronal presynaptic inhibition and 3) the excitability of the alpha-motoneurons determining reflex amplitude. Since the alpha motoneuron is the common element of all factors, motoneuron excitability is the main source of variation in reflex amplitude (Bonnet, Requin and Semjen,1981).

Alterations in the extracellular chemical mileu effect motoneuron excitability. Increased extracellular potassium levels hypopolarize motoneurons and increase excitability (Jimenez, Slodkin and Rudomin, 1985).

Endogenous opiods (endorphins) effect spinal cord activity. Morphine increases supraspinal descending inhibitory influences on afferent sensory impulses in cats

(Satoh and Takagi, 1971). Methionine-enkephalin, leucine-enkephalin and beta-endorphin also inhibit action potential propagation in myenteric neurons sterospecifically similar to central nervous system neural membranes (Williams and North, 1979). Endorphins act to hypopolarize presynaptic fibers decreasing neurotransmitter release (Cherubini and Morita, 1985). In a series of experiments on volume-induced spontaneous bladder contraction reflexes in the rat, Dray and coworkers (Dray and Metsch, 1984; Dray,Nunan and Wire, 1985; Dray, Nunan and Wire, 1986; Dray,Nunan and Wire, 1987) showed this reflex to be inhibited by enkephalin-like analogs, morphine and beta-endorphin. Furthermore, beta-endorphin also inhibited enkephalin antagonists and potentiated submaximal levels of agonists, indicating a possible regulatory role of beta-endorphin in spinal reflex activity. Rapid onset of this endorphin mediated reflex inhibition following intracerebral ventricular injection suggests supraspinal origins.

To summarize, exercise could induce altered motoneuron excitability by any of the above mentioned theories, alone or in combination; increased total body and hypothalamic temperature, peripheral to central feedback or increased central and peripheral endorphin levels.

Eccentric Exercise and Motoneuron Excitability

All of the above cited studies relating exercise-induced changes in neuromuscular arousal utilized concentric-biased exercise, i.e. exercise whose main active

component is concentric in nature. Despite biomechanical differences between concentric and eccentric work, the metabolic and physiologic responses are similar (Abbott, Bigland and Ritchie, 1952). Eccentric exercise produces metabolic reponses (i.e. heartrate, VO2) similar to concentric exercise, though the magnitude is reduced at similar external workloads. Decreased metabolic demand during eccentric work for similar external workload is due primarily to lessened motor unit recruitment (Asmussen, 1956). Both concentric and eccentric work require increased oxygen consumption with increased intensity, although the slope is steeper for concentric work (Knuttgen, 1971). Heartrate responds similarly.

No differences in local skin temperature were noted comparing concentric and eccentric work in the triceps surae (Bobbert, 1986). Body temperature response to eccentric work has yet to be measured. As metabolic heat production is proportional to oxygen consumption, one would assume that for a similar VO2, both concentric and eccentric exercise would respond with similar increases in total body temperature.

To date, no studies have examined endorphin reponse to eccentric work. Both types of exercise represent a stress and endorphin release increases consistently under the stress of exercise (Farrell, 1985). Endogenous opiod release in response to the stress of exercise may regulate

proprioceptive feedback reflex arcs in the same manner as in rat parasympathetic reflex arcs (Dray, Nunan and Wire, 1985). Increased central levels of endorphins may inhibit reflex resonses to group I and group II afferent input. Whether eccentric and concentric exercise at the same metabolic load represent equal stresses is unknown. Differences may exist in endorphin response.

Any of the previously postulated mechanisms for alterations in motoneuron pool excitability associated with concentric exercise could also exert influences during eccentric work. Differences in proprioceptive feedback, motor unit recruitment or percieved stress could alter central nervous system responses to eccentric work compared to concentric work.

Delayed-Onset Muscle Soreness and Motoneuron Excitability

Despite similar physiologic changes to concentric exercise, eccentric exercise often results in delayed-onset muscle soreness (DOMS) (Armstrong, 1984).Three major theories have been postulated; the mechanical trauma theory (Armstrong, 1977), local ischemia theory (Karpati, 1974; Makitie, 1977) and muscle spasm theory (deVries, 1966). Many aspects of these theories overlap and may, alone or in combination, be mediated or potentiated by increased motoneuron excitability.

Fewer motor units are recruited at the same workload in eccentric work as compared to concentric work, resulting in

higher mechanical forces per unit area in eccentric work (Asmussen, 1956). Mechanical trauma due to the resulting high mechanical forces encountered results in physical disruption of cell and tissue integrity (Armstrong, 1977). Comparing downhill treadmill running to level treadmill running reveals that despite similar support phase times, vertical impact force is 14% greater in the former. Braking impulse is 200% greater than the propulsive force during downhill running while level running produces similar values (Dick and Cavanagh, 1987). Power absorption required in downhill running is 1585 watts compared to 954 on the level (Buczek and Cavanagh, 1987). High mechanical forces associated with a small number of motor units (Bigland-Ritchie and Woods, 1976) results in necrosis, torn and swollen myofibers and widening of the intracellular space (Stauder and Fritz, 1987). Armstrong associated DOMS to sarcolemic disruption allowing diffusion of monocyte attracting products, e.g. collagen breakdown products, into the interstitium. Subsequent accumulation of Group IV free nerve ending stimulants such as histamine, kinins and potasium result in pain sensation. Group IV feedback via spinal interneuronal linkages or descending pain associated influences from higher brain centers could alter motoneuron excitability.

Tissue ischemia may also result in sarcolemmal damage and predispose the above condition (Karpati, 1974). Increased reflex activity due to ischemia was postulated to

result in a localized, tonic spasm (deVries, 1966). Biceps EMG activity increased 93% post exercise-induced DOMS and was related to the extent of subjective soreness. Spasm indicated by increased MAP's would result in further ischemia due to vessel occlusion, thus catalyzing a viscious cycle. This spasm theory resulted from investigations where elevated EMG activity was measured during muscle soreness (deVries, 1966). Other investigators have not been able to substantiate increased EMG with exercise-induced muscle soreness (Abraham, 1977; Bobbert, Hollander and Huijing, 1986). deVries suggests inconcurent findings due to the lack of equipment sensitivity of the follow-up investigators. McGlynn and coworkers (McGlynn, Laughlin and Rowe, 1979) found increased EMG post-eccentric exercise, yet it was not found to be related to soreness.

Despite the equivical evidence of EMG activity, microcontracture not sufficient for EMG detection could exert localized effects on muscle spindle output. Mechanical damage to frog sartorii muscles increases oxygen consumption due to increased contractile activity (Barnes and Williams, 1987). If localized near spindle receptors, this injury induced microcontracture may effect Ia afferent activity resulting in altered motoneuron pool excitability.

It is obvious that either mechanical trauma, ischemia or spasm could affect proprioceptive input to motoneuron pools. Structural damage evidenced in myofibers and

connective tissue could as easily occur within muscle spindles themselves. Substance P elicited in ischemic states stimulates group IV free nerve endings (Rodbard and Farbstein, 1972) which may direct alterations in supraspinal and spinal interneuronal influences on motoneuron resting potential. Sensations of pain are also a stimulus for endorphin release.

Increased osmotic pressure often associtated with injury (Reneman, 1978) may also alter Ia output.

Altered motoneuron pool excitability consequent to eccentric exercise may have a causitive and /or potentiating effect on delayed-onset muscle soreness. Increased excitability resulting from structural damage may result in muscular spasm leading to prolonged damage, local ischemia and decreased waste and nutrient transfer.

Summary

To date, the literature reviewed reveals that motoneuron pool excitability is reduced following conventional exercise in a direct, intensity dependent manner (Bulbulian and Darabos, 1986). Mechanisms for this reduction have been postulated without adequate experimental study. No literature has dealt with eccentric exercise and its effects on motoneuron pool excitability.

The chemical, thermal and neural factors thought associated with decreased motoneuron excitability following conventional concentric exercise could respond similarly or

dissimilarly in eccentric exercise. Despite similar metabolic responses at equal intensities, eccentric work requires greater biomechanical stress, such as increased power absorption and braking impulse. This often results in delayed-onset muscle soreness. Understanding how the motoneuron pool excitability level responds following eccentric exercise and during subsequent DOMS may allow insight into the physiological mechanisms of exercise-induced neuromuscular relaxation and delayed-onset muscle soreness.

CHAPTER THREE

METHODS

The subjects, experimental design, maximal oxygen consumption and H-reflex measurement procedure, and the statistical analysis procedures will be described in this chapter.

Subjects

Subjects were 6 males aged 18-35 recruited from Kansas State University and surrounding community. All subjets were recreational or competetive runners, free of neuromuscular disorder and medication. Each subject was fully informed both orally and in writing of the experimental procedures and subsequently signed an informed consent (Appendix A).

Experimental Design

The experimental design consisted of two exercise and one control treatments. The two exercise treatments consisited of 20 minutes of treadmill running at 0% grade (non-biased) and -10% grade (eccentric-biased).

Exercise intensities were matched at 50% VO2 maximum for both exercise trials. Determination of running speed at 0% grade was undertaken in the protocol for the maximal oxygen consumption test by forming a regression line of running speed with oxygen consumption. Treadmill speed at 0% grade eliciting 50% VO2 maximum was then determined from the regression equation. To determine a treadmill speed that

would demand 50% VO2 maximum at -10% grade, data from a previous experiment in our lab (Miller, personal communication) allowed formulation of the following relationship:

treadmill speed demanding = speed at 0% demanding
50% max at -10% (50% VO2 max / 0.65)

Oxygen consumption values were measured at 9 and 10 and also 19 and 20 minutes of each exercise bout to determine actual relative intensities. Control sessions consisited of sitting quietly for 20 minutes. After each condition subject heartrates were allowed to return to within 5 beats per minute of resting values before further testing. Treatments were given at the same time of day with at least three days separating the control and 0% grade run from subsequent trials. Downhill running (-10% grade) was separated at least 10 days from subsequent testing to allow recovery. Treatments were randomly distributed within trials and subjects randomly assigned to trials.

Immediately pre-, post-, and 24 hours post-treatment, H- and M- waves were measured to calculate H/M ratios. Subjects performed all three conditions, thus acting as their own controls.

Muscle Soreness Assesment

Immediately prior to each reflex test, subjects filled out a subjective evaluation of muscular soreness obtained from Byrnes et.al. (1985) on five lower body anatomical areas: front of thigh, back of thigh, buttocks, front of

lower leg and back of lower leg (Appendix B). Percieved muscle soreness was judged on a continuous scale of 1=normal, 4=uncomfortable, 5=sore, 8=very sore and 10=very, very sore.

Measurement of Running Economy and Maximal Oxygen Consumption

Running economy was determined immediately prior to each maximal oxygen consumption test. Protocol consisted of three- two minute stages running at 0% grade on a Quinton treadmill at progressively higher speeds. Actual speeds tested were determined individually and ranged from 5-9 miles per hour. Subjects were fitted with a Daniels breathing valve attached to a 4.0-L mixing chamber through a 45 cm flexible hose. Ventilation volumes recorded from an Alpha Technologies VMM-2 ventilation meter. A 500 ml/min gas sample was drawn from the mixing chamber through anhydrous CaSO4 in a 25-ml drying tube for gas analysis. During the second minute of each stage, expired air was analyzed for carbon dioxide and oxygen content using Beckman LB-2 and OM-11 analyzers, respectively. Data collected were used to calculate minute oxygen consumption (VO2 in L/min).

Maximal oxygen consumption was determined by maintaining constant treadmill speed and increasing treadmill grade 2.5% per minute until volitional exhaustion. Expired air volume, carbon dioxide and oxygen content measures were obtained for each 30 second period of each

stage.

H-Reflex Measurement

The H-reflex measure was obtained utilizing a modified method of deVries et.al. (1981). The tibial nerve was stimulated via a stimulating electrode located in the popliteal fossa. Exact stimulating electrode position was determined using a probe electrode to find the location that produced the largest obtainable H-wave at a set intensity. An 8mm Ag/AgCl electrode was then secured with adhesive backing over the site. The stimulator indifferent electrode consisted of an EKG plate electrode located approximately 15 cm proximal to the popliteal fossa on the posterior ipsilateral thigh.

Single square wave impulses of 0.5 msec duration were then delivered at 10 second intervals via a Grass S44 stimulator and Grass SIU5 stimulus isolation unit. Stimulation voltage began at 5 volts and was increased in 5 volt increments until the H-wave disappeared. Stimulus voltage increments were then increased to 10 volts until the plateau M-wave value was obtained. The criterion for M-maximum plateau was set at three consecutive M-wave values showing no increase despite increased stimulus voltage. Peak H-wave value was determined initially by measuring two H-wave values at each stimulus voltage. H-wave amplitude was more accurately determined by re-examining the 10-15 volt stimulus range around the peak H-wave in 1.0 volt increments

to insure the peak H-wave was not missed when using the wider 5 volt ascending increment.

Two H-wave values were recorded at each 1.0 volt increment to determine the stimulus intensity for H-maximum. Eight more H-wave amplitudes were recorded at H-maximum stimulus. Eight of the ten total measurements for H-maximum were then averaged, after discarding the highest and lowest measured H-maximum amplitude, and the resulting mean taken as H-maximum.

Muscle action potentials were measured using two 8mm Ag/AgCl electrodes in a bipolar arrangement. The active electrode was placed approximately 3 cm distal to the insertion of the gastrocnemius along the midline. The indifferent electrode was placed on the lateral maleolus. An EKG plate was placed on the medial maleolus of the ipsilateral leg serving as the ground. Electrode sites were abraded to produce interelectrode resistances less than 5000 ohms. Action potentials were amplified (Colbourn Hi Gain Bioamplifier/Coupler S75-01) at 100 gain for M-maximum determination and 1000 gain for H-maximum determination. Frequency band pass was set at 1.0-250 Hz measured at the 3dB point. Amplified signals were then passed in series through a Tektronix T912 storage oscilloscope and displayed for measurement. Oscilloscope sensitivity was manipulated to produce the highest resolution possible. Amplitude was measured to the nearest 0.25 division, giving M-wave

measures to the nearest 0.5 millivolt and H-wave measures to the nearest 0.25 to 0.5 millivolt value. Only the positive deflection was measured to allow increased oscilloscope sensitivity. Hugon (1973) has found the negative and positive amplitudes of the H-wave proportional.

Each electrode site was marked for removal of EKG plate electrodes during exercise and to allow accurate replacement of adhesive electrodes if they dislodged during exercise.

All reflex testing was done in a copper paneled Faraday cage to eliminate 60 Hz interference. The subject was in a prone position, tested leg flexed at the knee to approximately 60 degrees and the foot secured in neutral to slight plantar flexion (90-105 degrees) on a foot plate. Subjects were asked to assume a comfortable position. Arm and head position was noted and repeated in subsequent trials. Subjects were encouraged to remain still, relaxed and alert during testing to avoid changes in arousal state.

Statistical Analysis

Data was analyzed using a one-way analysis of variance with a block design. Significant main effects were analyzed post hoc using least squares means tests to determine differences among treatments. Significance level was set at $p>0.05$.

CHAPTER 4

RESULTS AND DISCUSSION

Physical characteristics of subjects, typical H- and M-wave response and comparisons between H- and M- wave associated variables following non-biased and eccentric-biased exercise will be presented. A discussion of the results follows.

Physical Characteristics of Subjects

Physical characteristics of the subjects are presented in Table 1. All subjects were males. Subjects' average height, weight and age were 178.64 cm, 71.52 kg and 27 years, respectively. Mean maximal oxygen consumption was 66.28 ml/kg/min for subjects tested.

Table 1.

Physical characteristics of subjects

Subject #	Height (cm)	Weight (kg)	Age (yrs.)	VO2 maximum (ml/kg/min)
1	177.8	73.18	28	70.65
2	180.34	75.00	25	63.20
3	182.88	75.00	25	66.52
4	180.34	69.10	25	68.80
5	175.26	66.36	34	58.04
6	175.26	70.45	27	66.28
$\bar{X}$	178.64	71.52	27	66.28
$\pm$SD	3.076	3.481	3.633	4.905

Typical H- and M-wave Responses

Subject 1 represents a typical H- and M-wave response pattern illustrated in Figure 1. The H-wave increases with increasing stimulus intensity (usually subthreshold for the M-wave), plateaus, and then decreases before the maximum M-wave amplitude is attained. M-wave amplitude increases to a plateau value at which no further increase in M-wave amplitude occurs with increasing stimulus voltage. H/M ratio is then defined as the maximum H-wave divided by the maximum M-wave (in millivolts). H-maximum stimulus values ranged from 13 to 62 volts. M-maximum stimulus values ranged from 50 to 90 volts.

Raw Data

Table 2 presents the raw data for all six subjects for the three treatments; control (no exercise), non-biased (0% grade), and eccentric-biased (-10% grade) treadmill running. Maximum H-wave and M-wave values are given in millivolts for each subject and treatment along with treatment means and standard deviations. H/M ratio data are also presented as means $\pm$ S.D.

Oxygen Consumption during Exercise Trials

Oxygen consumption was measured during the 9th and 10th and also the 19th and 20th minutes of each exercise trial. Average oxygen consumption for the 9th and 10th minutes for the level and downhill trials were 32.4 and 29.45 ml/kg/min, respectively. Corresponding relative intensities were 49.09% and 44.6% of VO2 maximum. Intensities were not significantly

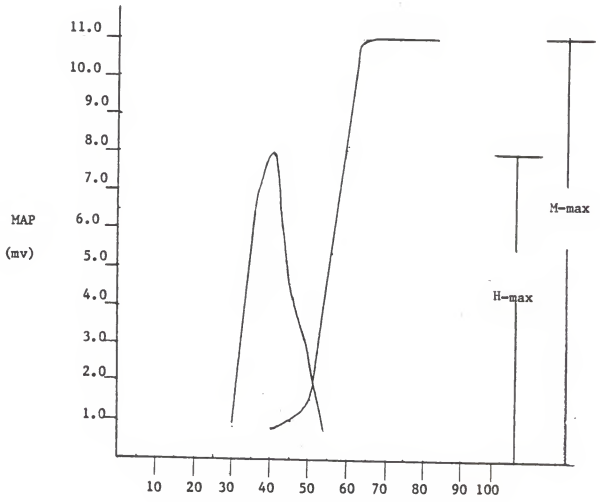

Figure 1 - Plot of typical Stimulus-Response for H- and M-waves. Stimulus in volts; response in millivolts. Illistration of H-wave and M-wave maximums provided.

TABLE 2

H-maximum, M-maximum, and H/M ratio values for subjects and means for pre-, post- and 24 hours post-treatment. Values presented in millivolts except ratios which are expressed as percent.

CONTROL

	PRE			POST			24HR POST		
SUB	H	M	H/M	H	M	H/M	H	M	H/M
1	10.00	11.00	90.90	9.90	11.20	88.39	7.28	11.20	65.00
2	1.93	8.30	23.25	2.39	8.40	28.45	1.85	8.40	22.02
3	9.02	8.40	107.38	8.98	8.40	106.91	8.72	8.40	103.81
4	5.76	10.80	53.33	7.04	10.40	67.69	5.09	9.80	51.94
5	4.88	11.60	42.07	5.76	12.00	48.00	4.50	11.00	40.91
6	3.18	11.00	28.91	2.94	8.80	33.41	4.01	9.70	41.34
$\bar{X}$	5.80	10.18	57.64	6.17	9.87	62.14	5.24	9.75	54.17
±SE	1.3	0.59	13.96	1.26	0.63	12.76	0.99	0.49	11.49

NON-BIASED

	PRE			POST			24HR POST		
SUB	H	M	H/M	H	M	H/M	H	M	H/M
1	8.00	11.72	71.43	7.70	11.00	70.00	8.74	11.20	78.04
2	2.73	8.60	31.74	2.59	8.40	30.83	2.58	7.60	33.95
3	7.88	9.60	82.08	6.33	8.80	71.93	8.43	8.40	100.36
4	5.31	9.80	54.18	4.53	9.80	46.22	5.76	10.80	53.33
5	4.86	11.00	44.18	4.57	11.00	41.55	4.18	11.00	38.00
6	6.40	10.00	64.00	4.98	9.50	52.42	3.48	10.10	34.46
$\bar{X}$	5.86	10.03	57.94	5.12	9.75	52.16	5.53	9.85	56.35
±SE	0.82	0.39	7.51	0.71	0.44	6.61	1.06	0.61	11.16

ECCENTRIC-BIASED

	PRE			POST			24HR POST		
SUB	H	M	H/M	H	M	H/M	H	M	H/M
1	9.08	11.00	82.55	7.28	10.60	68.68	7.90	10.40	75.96
2	3.13	8.90	35.17	2.13	8.80	24.20	2.87	8.80	32.61
3	8.43	8.40	100.36	6.04	8.00	75.50	8.98	8.40	106.90
4	5.88	10.60	55.47	2.91	10.20	28.53	4.57	10.80	42.31
5	5.29	10.00	52.90	4.46	10.20	43.72	5.04	9.80	51.43
6	3.58	10.80	33.10	3.28	10.90	30.09	4.75	11.20	42.41
$\bar{X}$	5.90	9.95	59.93	4.35	9.78	45.12	5.76	9.73	60.12
±SE	1.0	0.44	10.87	0.81	0.46	8.98	0.92	0.42	11.04

different between trials (p = 0.6106). Oxygen consumption for the 19th and 20th minutes rose slightly during both trials to 33.69 and 31.92 ml/kg/min for the level and downhill runs, respectively. Relative intensities rose to 51.03% VO2 maximum for the level run and 48.31% VO2 maximum for the downhill trial. Relative intensities were not significantly different between trials (p = 0.0898). Combining all oxygen consumption data for trials produces relative intensities of 50.06% at 0% grade and 46.46% at -10% grade, a statistically significant difference of 3.6% VO2 maximum (p = 0.0054). In general, relative intensities between 0% grade and -10% grade treadmill runs were not appreciably different.

H/M Ratio Responses to Exercise

Mean pre-test H/M ratio values were 57.6% (±14.0%) for control, 57.9% (±7.5%) for 0% grade and 59.9% (±10.9%) for the downhill runs (see Table 2). Analysis of variance showed significant differences between subjects (p=0.0003), but not between treatments (p=0.9357) indicating the H/M ratio is highly variable inter-individually, yet stable intra-individually.

TABLE 3

Analysis of Variance for the Pre-test H/M ratio between treatments.

Source	df	Sum of Squares	Mean Square	F value	p>F
Model	7	0.97145	0.13877	9.98	0.0008
Error	10	0.13907	0.01391		
Total	17	1.11052	Root MSE 0.117928		

Source	df	Type I SS	F value	p>F
Subject	5	0.969590	13.94	0.0003
Treatment	2	0.001862	0.07	0.9357

Following a 20 minute no exercise control period, the mean H/M ratio value rose from 57.6% to 62.1% (±12.8%). Level treadmill exercise reduced the mean H/M ratio from 57.9% to 52.2% (±6.6%) and eccentrically-biased downhill running decreased the mean ratio further, from 59.9% to 45.1% (±9.0%).Analysis of variance tables for H/M ratios on pre-, post- and 24 hour post-treatment conditions are presented in Tables 3 to 5. Analysis of variance showed highly significant differences between subjects for post-exercise H/M ratios as expected. Comparison of treatment differences were not quite significant (p=0.0653), yet showed a least squares means trend toward differences between control and downhill treatments (p=0.0231).

TABLE 4

Analysis of Variance for Post-test H/M ratio comparing treatments.

Source	df	Sum of Squares	Mean Square	F value	p>F
Model	7	0.828281	0.118325	9.78	0.0009
Error	10	0.120956	0.012095		
Total	17	0.949237	Root MSE 0.10998		

Source	df	Type I SS	F value	p>F
Subject	5	0.74051	12.24	0.0005
Treatment	2	0.08777	3.53	0.0653

Twenty-four hours post-treatment, H/M ratios returned to levels not significantly different between treatments ($p=0.1492$). Mean 24-hour post-treatment H/M ratios were 54.2% ($\pm$11.5%), 56.4% ($\pm$11.2%) and 60.1% ($\pm$11.0%) for control, level and downhill run treatments, respectively. Again, significant subject differences were found ($p=0.0001$).

TABLE 5

Analysis of Variance for 24 hour Post-test H/M ratios comparing treatments.

Source	df	Sum of Squares	Mean Square	F value	p>F
Model	7	1.12289881	0.16041412	68.23	0.0001
Error	10	0.02351115	0.00235112		
Total	17	1.14640996	Root MSE	0.04848830	

Source	df	Type I SS	F value	p>F
Subject	5	1.11202207	94.59	0.0001
Treatment	2	0.01088774	2.32	0.1492

Percent Change in H/M Ratio

Individual and mean percent change in H/M ratio following control, non-biased and eccentric-biased exercise are presented in Table 6. Analysis of variance tables for percent change comparisons between treatments are presented in Tables 7 to 9.

Percent changes in H/M ratio between pre-test and post-test values following control treatment resulted in a 12.6% (±4.9%) increase. Responses ranged from a 2.8% decrease to a 26.9% increase (see Figure 2). Four of six subjects increased H/M ratio from pre-test values following a 20 minute no exercise control period, ranging from 14.1% to 26.9%. Two other subjects showed slight decreases. Conversely, all subjects decreased H/M ratio values following exercise of either type. Treadmill running at 0%

TABLE 6

Percent Change between Pre-, Post- and 24 hour Post-test H/M ratios for treatments.

subject	Pre-Post(%)	Post-24 hr. Post(%)	Pre-24 hr. post(%)
Control			
1	-2.76	-26.46	-28.49
2	22.37	-22.60	-5.29
3	-0.78	-2.90	-3.66
4	26.92	-23.27	-2.61
5	15.60	23.74	43.05
6	-1.31	0.66	-0.66
$\bar{X}$	12.62	-11.04	0.09
± SE	4.89	7.77	9.51
Concentric-biased			
1	-2.0	11.49	9.25
2	-2.87	10.12	6.96
3	-12.38	39.52	22.26
4	-14.69	15.38	-15.69
5	-5.9	-8.52	-13.91
6	-18.10	-34.26	-46.16
$\bar{X}$	-9.33	5.61	-3.88
±SE	2.72	10.15	9.78
Eccentric-biased			
1	-16.80	10.60	-7.98
2	-31.39	34.75	-7.28
3	-24.77	41.59	6.51
4	-48.57	48.30	-23.72
5	-17.35	17.63	-2.78
6	-8.19	40.94	28.13
$\bar{X}$	-24.65	37.63	1.53
±SE	5.68	9.97	5.73

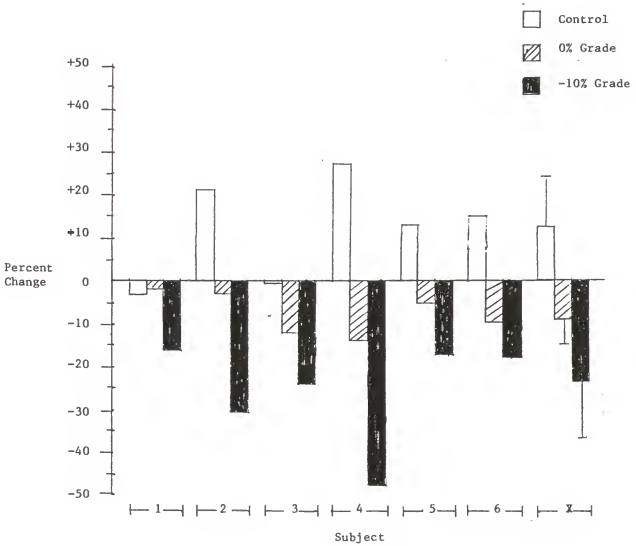

Figure 2 - Percent change pre-test to post-test for subjects across treatments. Mean values given with standard deviations.

grade reduced the H/M ratio a mean of 9.3% (±2.7%). All subjects showed a decrease, responses ranging from -2.0% to -18.1%. Eccentric-biased exercise produced a greater mean H/M reduction. Mean percent change following downhill running was -24.7% (±5.7%).

TABLE 7

Analysis of Variance Table for Percent Change in H/M ratio Comparing Pre-test and Post-test data.

Source	df	Sum of Squares	Mean Square	F value	p>F
Model	7	4484.5791	640.6541	3.92	0.0256
Error	10	1633.4351	163.3435		
Total	17	6118.0143	Root MSE 12.7806		

Source	df	Type I SS	F value	p>F
Subject	5	273.6621	0.34	0.8804
Treatment	2	4210.9170	12.89	0.0017

Analysis of variance showed significant differences between treatments (p=0.0017), but not subjects (p=0.8804). Least squares means analysis (see Table 10) showed control percent change significantly different from downhill running (p=0.0005) and level running (p=0.0139). Differences between exercise trials approached significance (p=0.0647). Post hoc t-test analysis found the percent change after level running not significantly different from zero (p=0.1040).

H/M ratio changes during the 24-hour period following treatment reversed changes observed immediately post-

treatment. Percent change from post-test to 24-hour post-test for control decreased 11.1% (±7.8%). Corresponding changes for level and downhill running were increases of 5.6% (±10.2%) and 37.6% (±10.0%), respectively. Analysis showed significant differences in recovery between treatments (p=0.0158) with eccentric-biased exercise resulting in a significantly greater percent change. No differences were shown between non-biased exercise and control recovery.

TABLE 8

Analysis of Variance table for Percent Change in H/M ratio comparing Post-test and 24 hr. Post-test data.

Source	df	Sum of Squares	Mean Square	F value	p>F
Model	7	9544.7781	1363.5398	2.40	0.1015
Error	10	5682.4778	568.2477		
Total	17	15227.2567	Root MSE	23.8379	

Source	df	Type I SS	F value	p>F
Subject	5	2202.5023	0.78	0.5891
Treatment	2	7342.2764	6.46	0.0158

Trends seen in post-testing were reversed during the 24 hour recovery period resulting in a net percent change pre-test to 24-hour post not significantly different between treatments (p=0.9219) nor zero for control (p=0.9931), non-biased (p=0.6698) and eccentric-biased (p=0.8790) exercise.

TABLE 9

Analysis of Variance table for Percent Change in H/M ratio comparing Pre-test and 24 Post-test data.

Source	df	Sum of Squares	Mean Square	F value	p>F
Model	7	933.1041	133.300	0.23	0.9673
Error	10	5730.6487	573.6049		
Total	17	6663.7529	Root MSE 23.9387		

Source	df	Type I SS	F value	p>F
Subject	5	839.0994	0.29	0.9062
Treatment	2	94.0048	0.08	0.9219

Acute and Delayed-Onset Muscle Soreness

Subjective evaluation of muscle soreness for all subjects and treatments is presented in Table 11. Analysis of variance for muscle soreness ratings is presented in Table 12.

TABLE 10

Least squares means table for percent change in H/M ratio between treatments.

---------- Percent change pre- to post-treatment.----------

Treatment	Mean	S.E.	p>t Ho: Mean=0
Control	12.6226	5.2176	0.0361
0% grade	-9.3308	5.2176	0.1040
-10% grade	-24.6459	5.2176	0.0008

------Percent change post- to 24 hour post-treatment.-------

Treatment	Mean	S.E.	p>t Ho: Mean=0
Control	-11.0433	9.7318	0.2829
0% grade	5.6115	9.7318	0.5769
-10% grade	37.6368	9.7318	0.0031

-------Percent change pre- to 24 hour post-treatment-------

Treatment	Mean	S.E.	p>t Ho: Mean=0
Control	0.0861	9.7729	0.9931
0% grade	-3.8786	9.7729	0.6998
-10% grade	1.5260	9.7729	0.8790

Probability > (t) Ho: Treatment means equal. Pre-treatment = A; Post-treatment = B; 24 hour Post-treatment = C.

	A to B	B to C	A to C
Control vs. 0%	0.0139	0.2541	0.7861
Control vs. -10%	0.0005	0.0054	0.9191
0% vs. -10%	0.0647	0.0423	0.7040

TABLE 11

Sums of ratings of soreness in 5 lower body sites; front of thigh, back of thigh, buttocks, front of lower leg and back of lower leg.

	Control			0% grade			-10% grade		
Sub	Pre	Post	24Post	Pre	Post	24Post	Pre	Post	24Post
1	5	5	5	5	5	5	5	5	7
2	5	5	5	5	5	5	5	6	9
3	5	5	5	5	5	5	5	7	13
4	6	6	5	6	6	5	6	7	12
5	5	5	5	5	5	5	5	9	14
6	5	5	5	5	5	5	5	8	12
$\bar{X}$	5.167	5.167	5.00	5.167	5.167	5.00	5.167	7.00	11.167
+SD	0.40	0.40	0.40	0.40	0.40	0.40	0.40	0.40	0.40

Ratings of subjective soreness for back of lower leg only.

	Control			0% Grade			-10% Grade		
Sub	Pre	Post	24Post	Pre	Post	24Post	Pre	Post	24Post
1	1	1	1	1	1	1	1	1	1
2	1	1	1	1	1	1	1	1	1
3	1	1	1	1	1	1	1	2	6
4	1	1	1	1	1	1	2	2	2
5	1	1	1	1	1	1	1	1	1
6	1	1	1	1	1	1	1	3	5
$\bar{X}$	1.0	1.0	1.0	1.0	1.0	1.0	1.17	1.67	2.67
+SD	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33

TABLE 12

Analysis of Variance for the sum of rated soreness in 5 lower body sites; front of thigh, back of thigh, buttocks, front of lower leg and back of lower leg.

Source	df	Sum of Squares	Mean Square	F value	p>F
Model	8	298.1904	37.2738	5.42	0.0047
Error	12	82.4762	6.8730		
Total	20	380.6667		Root MSE 2.6216	

Source	df	Type I SS	F value	p>F
Sub	6	126.6667	3.07	0.0464
Time	2	171.5238	12.48	0.0012

Neither control nor non-biased exercise resulted in any significant changes in muscle soreness at any time. Eccentric-biased exercise resulted in significant increases in muscle soreness ratings both between subjects ($p=0.0464$) and time of rating (pre-, post- and 24-hour post; $p=0.0012$). Soreness was significantly elevated over control and non-biased exercise both immediately post- ($p=0.0024$ and $p=0.0177$, respectively) and 24-hours post-exercise ($p=0.0001$). Mean soreness ratings were 7.0 $\pm$0.39 post- and 11.2 $\pm$0.39 24-hours post. Soreness ratings 24-hours post were significantly different from immediately post ($p=0.0001$). Soreness ratings increased 36.1% immediately post-exercise and an additional 56.4% in the subsequent 24 hour recovery, resulting in a total increase of 116.7% pre-

exercise compared to 24-hour post-exercise.

Calf Muscle Soreness

Soreness ratings isolated for back of lower leg responded in a similar pattern to that of the overall lower body, except subjective soreness was not significantly elevated immediately post-eccentric-biased exercise ($p=0.2867$). Subjective calf soreness was significantly different from all other times at 24 hours post-downhill running.

Discussion

The purpose of this study was to compare motoneuron pool excitability alterations following eccentric-biased exercise to conventional, non-biased exercise. A major objective of the experiment was to equate metabolic workload at a relative intensity of 50% VO2 maximum in order to eliminate differences in the non-biased and eccentric-biased treatments attributable to exercise intensity. The results show that the relative intensities studied were 46.5% and 50.1% VO2 maximum for downhill and level treadmill exercise bouts, respectively. The difference between relative intensities of the exercise bouts was 3.6% VO2 maximum, a statistically significant difference. Differences were not statistically different at minutes 9 and 10 nor 19 and 20.

Differences seen seem to have resulted from a larger upward drift in oxygen comsumption (UDO) for the downhill run. This UDO agrees with previous literature values reporting non-steady state conditions for eccentrically-biased exercise (Dick and Cavanagh, 1987), in which treadmill running at -10% grade elicited a 10% UDO between minutes 10 and 40 of exercise. The present study demonstrated an 8.4% UDO between minutes 10 and 20. This UDO may have contributed to the significant difference between combined relative intensities of the exercise bouts. Failure to match relative intensities based on the derived equation (see Methods) may have been due to a familiarization effect of the subjects to downhill treadmill running as many subjects participated in previous downhill running trials. However, considering the non-significant differences in relative intensities between trials at minutes 9 and 10 and also between minutes 19 and 20, exercise trials can be considered matched for relative intensities.

H/M Ratio Responses to Exercise

H/M ratio values ranged from 22.0% to 107.4% for subjects tested. Previous literature reports mean H/M ratio values of 41.5% $\pm$ 18.5% (Crayton and King, 1981); 18.8% $\pm$1.75% (deVries, Wiswell, Bulbulian and Moritani, 1981); 17.0% and 48% for normal and spastic patients, respectively (Angel and Hoffman, 1963). No previous study has reported H/M ratio values over 100%. Methodology utilized in the present experiment can account for such discrepancies.

Recording electrodes were placed in a bipolar arrangement, with the active electrode over the soleus and the indifferent electrode over the lateral maleolus, thus increasing the effective pickup field to include not only the triceps surae, but peroneals as well. Direct stimulation of tibial alpha-motoneurons results in a M-wave in the triceps surae only. Group I afferent stimulation results in depolarization of alpha-motoneurons of not only homonymous, but synergist muscle groups as well. Resulting H-wave MAP's are the result of MAP's of the triceps surae and peroneal muscle groups combined, allowing the H-wave to exceed the M-wave in one subject.

Pre-test H/M ratios were highly variable inter-individually, yet stable intra-individually. This supports previous studies showing the H/M ratio stable and reproducible within subjects (Crayton and King, 1981).

Comparison of post-test H/M ratios between treatments showed neither type of exercise nor control produced significant alterations in motoneuron excitability ($p=0.0657$). Mean H/M ratios rose to 62.1% following control treatment, decreased to 52.2% following non-biased exercise and decreased further to 45.1% following eccentric-biased exercise. Lack of significant reduction following low-intensity non-biased exercise is in disagreement with previous studies (deVries, Wiswell, Bulbulian, and Moritani, 1981; Bulbulian and Darabos, 1986). deVries et. al. (1981)

reported decreases in mean H/M ratios 18.2% following 20 minutes exercise at 40% heartrate range. Treadmill running at 4% grade for 20 minutes at 40% VO2 maximum reduced H/M ratio in 9 subjects (Bulbulian and Darabos, 1986). Differences in the present study may have been hindered by the small subject pool (n=6) and high inter-individual variability. Though not statistically valid, one can discern trends for differences between control and downhill run trials utilizing post hoc t-tests (p=0.0231).

No differences were noted in H/M ratios between treatments by 24 hours post-treatment (p=0.1492) indicating that changes seen immediately post-treatment were abolished during the subsequent 24 hour period.

Percent Change in H/M Ratio

High inter-individual variations in H/M ratio values can be overcome by examining the percent change in H/M ratio between testing periods.

Comparing percent change between pre-treatment and post-treatment H/M ratios reveals significant differences between treatments (p=0.0017). Control treatment increased mean H/M ratio values 12.6% $\pm$4.9%, non-biased exercise decreased the mean H/M ratio 9.3% $\pm$2.7% and eccentric-biased exercise decreased the H/M ratio mean 24.7% $\pm$5.7%. Inter-individual variation was controlled, as evidenced by insignificant subject differences between treatments (p=0.34).

In subjects tested, 20 minutes of inactivity aroused

the motoneuron pool as evidenced by the 12.6% increase in H/M ratio. Bulbulian and Darabos (1986) also showed an increase following control periods of inactivity (+2.11%), though non-significant. Apparently, inactivity tends to arouse many subjects (four of the six in the present study). Lack of proprioceptive input, anxiousness or lack of distraction may increase descending excitatory or decrease inhibitory influences on the motoneuron pool during inactivity. This exemplifies the necessity of utilizing a control treatment in studies of exercise-induced alterations in motoneuron excitability.

Low-intensity non-biased exercise reversed the post-control increase, resulting in a mean decrease of 9.3% in the H/M ratio. All six subjects showed a decrease. This reduction was significantly different from control values, but not from zero, indicating low-intensity non-biased exercise may decrease motoneruon pool excitability compared to inactivity, but may not be significantly different from a period of normal activity. This finding is inconsistent with those of deVries et.al. (1981) and Bulbulian and Darabos (1986). Lack of significance in the present study was probably attributable to the small sample size. However, one previous study (deVries, et.al. 1981) utilized a subject pool screened for high state and trait anxiety. As these subjects would benefit most from exercise (deVries, 1981) greater reductions would be expected in this population than

in the present studied.

Exercise-bias differences may also account for discrepancies between the present study and those previously cited. Both deVries et.al. (1981) and Bulbulian and Darabos (1986) utilized concentric-biased exercise protocols. Bicycle ergometer exercise was used in the former, while the latter utilized 4% grade treadmill running. Level treadmill running (0% grade), equally concentric and eccentric, differs from uphill and downhill running in muscle enzyme responses and motor unit recruitment patterns (Armstrong, 1983). Both uphill and downhill running increase glucose-6-phosphate dehydrogenase levels in slow twitch muscle of rats, indicative of fiber damage. Level running does not alter glucose-6-phosphate dehydrogenase levels.

Large percent change reductions were noted in all subjects' H/M ratio following 20 minutes of low-intensity eccentricly-biased exercise. Mean H/M ratio reduction of 24.7% was significantly different from zero ($p=0.0008$) and control ($p=0.0005$). Comparing eccentricly-biased to non-biased exercise resulted in a non-significant trend toward a larger reduction in the former ($p=0.0647$).

Comparisons between eccentricly-biased and non-biased exercise trials may be confounded by the statistically significant difference in relative intensities. Post-exercise H/M ratio reductions are reported intensity dependent (Bulbuian and Darabos, 1986), with increased intensity producing greater H/M ratio reductions. In the

present study, the eccentricly-biased trial was conducted at 46.5% VO2 maximum and the non-biased at 50.1% . As the former was conducted at a lower relative intensity, H/M ratio reductions may have been attenuated. Actual H/M ratio reductions following eccentricly-biased exercise may be greater. Reducing the non-biased trial intensity would have presumably lessened the recorded 9.3% decrease, thus increasing the magnitude of the difference between exercise types. Greater H/M ratio reductions found for the lower intensity exercise in the present study disagree with the previous study (Bulbulian and Darabos, 1986) and suggest metabolic intensity may not be the underlying factor in H/M ratio reduction following exercise.

Contrary to the hypothesized results, large reductions in motoneuron pool excitability occured following eccentricly-biased exercise. Possible mechanisms for this decrease are many.

Body temperature does not appear to play a major role in exercise-induced alterations in motoneuron pool excitability. As metabolic demands were matched between non-biased and eccentric-biased exercise trials, body temperature reponses are assumed to be similar (Stalin and Hermansen,1966). Temperature-induced neuromuscular relaxation (Von Euler, 1956) may exert effects in a non-exercise situation, but apppears to have limited effects in the exercise-induced reduction of motoneuron excitability.

It is interesting to note that the 24.65% reduction in H/M ratio of the present study following eccentric-biased treadmill running at 50% VO2 maximum corresponds to a 21.5% reduction elicited at 75% VO2 maximum in concentric-biased treadmill running found previously (Bulbulian and Darabos, 1986). Apparently, motoneuron pool excitability attenuations post-exercise do not correspond strictly to metabolic intensity, but to some other factor. Relative motoneuron stimulation may provide a more insightful measure of intensity.

Fewer motor units are recruited per unit force in eccentric work (Asmussen, 1956) resulting in a lower integrated EMG/tension ratio (Komi and Vitasalo, 1977). However, integrated EMG (iEMG) increases during downhill (-10%) running while no change occurs during level running (Dick and Cavanagh, 1987). Increased motor unit recruitment during eccentrically-biased exercise may result from fatigue of predominantly slow twitch fibers as they are disproportionately damaged in eccentric exercise (Armstrong, 1983). Eccentric exercise produces greater low frequency fatigue than concentric exercise (Edwards, Mills and Newham, 1981). In order to maintain force output, more motor units must be recruited via Group Ia feedback (Houk, 1979), yet muscle stiffness, relative sensitivity to Ia/Ib input, must remain constant. Peripheral afferent depolarization has been postulated as a modulator of afferent sensitivity (McCrea, 1986), and thus may act to regulate the relative

input sensitivity to Ia and Ib input, allowing a higher absolute stimulation while maintaining the Ia/Ib input ratio. Increased PAD would then reduce the H/M ratio in direct proportion to the level of motor unit recruitment.

Central neuromodulators, such as endogenous opiods and enkephalins, probably effect H-reflex parameters subsequent to exercise. Stress, such as exercise (Bortz, 1981; Colt, 1981; Farrel, 1982) and pain (Oliveras, Besson, Gulbaud and Liebeskind, 1974) increases endorphin levels in humans. Beta-endorphin regulates neuromodulator activity in parasympathetic reflexes of the rat (Dray, Nunan and Wire, 1986). Increased central endorphin levels may inhibit alpha-motoneuron excitability directly, via interneuronal influences or by altering perpheral afferent depolarization and thus sensitivity to proprioceptive feedback of Group I and II afferents. Similar motoneuron depression after 50% VO2 maximum downhill running and 75% VO2 maximum level running suggests that downhill running represents a stress similar to that of a higher metabolic intensity. The unaccustomed activity of downhill running or discomfort associated with eccentric work may influence endorphin responses dissociated from metabolic intensity.

Morphological changes in the motoneuron itself, associtated with activity, alter sensitivity to Group Ia EPSP input. Motoneuron somas enlarge in response to exhaustive exercise (Gerchman, 1963). Greater motor unit

recruitment necessary to overcome slow twitch fatigue during eccentric or high intensity conventional exercise may result in increased motoneuron cell body size, thus decreasing input resistance. Decereased input resistance lowers motoneuron sensitivity to Ia EPSP's (Burke, 1973). Attenuated H/M ratios post-exercise may result. Morphological changes regress to control levels as adaptation occurs (Gerchman, 1963).

Alterations in gamma-bias on muscle spindles postulated as a neuromuscular relaxation mechanism probably do not exert effects on the H/M ratio reduction following exercise. Fusimotor activity is considered absent or negligible at rest (Vallbo, 1981). All H/M ratio measures in this and previous studies were taken at rest.

In accordance with the relaxation model of Haugen (1960), "random and intermittent, constantly changing proprioceptive stimuli allowing normal cortical activity" leads to a relaxed state. Downhill running at metabolic demand similar to level running requires a greater running speed , possibly creating proprioceptive stimuli more conducive to relaxation than level running.

One must also determine if the H/M ratio is a measure of alpha-motoneuron pool excitability alone, or if it also measures the extent of peripheral afferent depolarization (PAD). As the H-reflex stimulus is peripheral, PAD regulating mechanisms also affect the Group Ia artificial stimuli reaching the motoneuron pool. Exercise

may leave the motoneuron pool unaltered, alterations in the H/M ratio may result from changes in PAD associated with exercise. Downhill running, with its associtated increased knee flexion and power absorption (Buczek and Cavanaugh, 1987) increase Ia and Ib afferent input, respectively. Increased Group I input increases spinal PAD (McCrea, 1986), and may decrease H/M ratio values post-downhill running independent of alterations in motoneuron pool excitability.

H/M Ratios 24 Hours Post-Treatment

Contrary to the results hypothesized, exercise-induced alterations in H/M ratios were abolished by 24 hours post-treatment for all treatment conditions including eccentricly-biased exercise. Percent changes not significantly different from zero were measured at 24 hours post-treatment compared to pre-test values. In light of increased EMG activity 48 hours post-eccentric work previously reported (deVries, 1966), it was postulated that the motoneuron pool would be subjected to increased excitatory afferent input 24 hours post-downhill running. Increased EMG following eccentric work, if present, can thus be assumed localized in the periphery, with no central excitatory or inhibitory effects on motoneuron pool excitability. Whatever the model for decreased H/M ratios post-exercise, its effects are short-lived.

Acute and Delayed-Onset Muscle Soreness

Acute and delayed-onset muscle soreness ratings were elevated only after eccentric-biased exercise. Ratings of soreness increased immediately post-downhill running 36.11% ($p=0.0024$) for total lower body and 43.1% ($p=0.2867$) for the back of lower legs. Twenty-four hours post-downhill running, total soreness ratings were elevated 116.67% above pre-test values ($p=0.0.0025$). Calf muscle soreness scores were elevated 128.8% ($p=0.0025$). Back of lower leg scores responded similarly to total lower body scores. Muscle soreness responses in the present study were consistent with those reported elsewhere (Abraham, 1977; Armstrong, 1984).

Changes in motoneuron excitability do not seem related to degree of reported soreness. Both non-biased and eccentric-biased exercise produce reductions in H/M ratios, yet soreness was reported only after eccentric-biased exercise. Soreness ratings were highest 24 hours post-test while H/M ratio levels had returned to pre-test values. No apparent central mechanism, as evidenced by H/M ratio measurement, plays a role in acute or delayed-onset muscle soreness. Whether peripheral factors exert influences on the CNS in DOMS is uncertain. Soreness may be associated with increased muscle spindle sensitivity resulting from structural damage, local microcontracture and/or edema. As the H-reflex bypasses the spindle apparatus, these changes would not be apparent utilizing this method. Measuring the tendon-tap reflex, T-reflex, may be a more appropriate tool,

as it measures the myotatic reflex apparatus intact. Comparing differences between the T-reflex and H-reflex parameters reveals peripheral factors which may exert influences centrally not apparent using the H-reflex alone, i.e. gamma-bias changes associated with DOMS.

CHAPTER 5

Summary, Conclusions and Recommendations for Future Research

Summary

Neuromuscular adaptation to exercise has recieved scant attention in the literarture. Alterations in neuromuscular state have been shown both at the central (de Vries, 1981) and peripheral (deVries and Adams, 1982) level following exercise, utilizing the Hoffmann reflex and electromyography, respectively. Understanding the neuromuscular responses and adaptations to exercise may allow better prescription of exercise in relaxation, tranquilizer replacement therapy and neuromuscular disorder.

Motoneuron pool excitability can be measured utilizing the Hoffmann reflex, or H-reflex. H-reflex methodology determines the ratio of two distinct MAP's, the M- and H-waves, respectively, to an electrical stimulus on the tibial nerve. Direct alpha-motoneuron depolarization results in the M-wave, while Ia afferent stimulation produces the reflex mediated H-wave. Measuring the largest obtainable H- and M-waves and converting to a ratio, the H/M ratio, gives the dependent measure of motoneuron pool excitability. Changes in the H/M ratio are taken as a change in motoneuron excitability.

Both low-intensity (deVries, et.al., 1981) and low- and high-intensity exercise (Bulbulian and Darabos, 1986) have reduced H/M ratios. The purpose of this study was to

compare the effects of eccentric-biased exercise (-10% grade treadmill running) with non-biased exercise (0% grade treadmill running) at similar metabolic demand (50% VO2 maximum). Six competitive or recreational, male runners, aged 25 to 34, were tested for alterations in H/M ratio pre- post- and 24 hours post-treatment. Treatments consisted of 20 minutes of either 1) control-no exercise, 2) -10% grade treadmill running and 3) 0% grade treadmill running. Exercise intensties were adjusted to 50% VO2 maximum. Subjective muscle soreness ratings were obtained simultaneously with H-reflex measures.

Data was analyzed using a SAS statistical package. Treatment differences were analyzed using a one-way analysis of variance in a general linear model procedure.

Both non-biased and eccentric-biased exercise resulted in decreased H/M ratios in all six subjects. Non-biased exercise reduced mean the H/M ratio 9.3%, while eccentric-biased exercise resulted in a 24.7% decrease, both of which were significantly different from control values (+ 12.6%), but not each other. Differences between exercise treatments approached significance (p=0.0653). H/M ratios returned to control levels 24 hours post-treatment for all treatment conditions.

Ratings of muscle soreness increased immediately post- (+36.1%) and 24 hours post-downhill running (+116.7%). Neither control nor non-biased exercise altered soreness ratings.

Both non-biased and eccentric-biased exercise decrease motoneuron pool excitability as measured by the H-reflex technique. Eccentric-biased exercise shows a larger reduction, though not statistically significant in the present study. Muscle soreness, both acute and delayed-onset, do not appear related to changes in motoneuron excitability.

Conclusions

The following conclusions are based on the assumption that the H/M ratio measures motoneuron pool excitability. This assumption may not be entirely valid, as peripheral input to the motoneuron via Group I afferents is known to be under regulation of presynaptic peripheral afferent depolarization (PAD).

Based on the results of this study, decreased motoneuron pool excitability is greater following eccentrically-biased exercise than non-biased exercise, when metabolic relative intensities are equal. Mechanisms of this response are unknown, and may involve neuromuscular fatigue, inhibition via exercise-induced endorphin release or alterations in descending influences on both alpha-motoneurons and/or peripheral afferents.

Recommendations for Future Research

Future research in the area of neuromuscular response to exercise must include:

1) First establishing the validity of measures such as the H-reflex. Neurological examination of motoneuron excitability under differing stimuli should be carried out with direct, intracelluar measurement of transmembrane potentials.

2) A repeat of the present study, increasing the subject pool.

3) Information regarding the stimuli altering H/M ratio responses post-exercise need to be examined individually. Such research may include examining H/M ratio alterations following exercise while under endorphin-block.

4) Research investigating the duration of H/M ratio reduction following various intensities and types of exercise.

5) Increase psychological testing to determine psychological correlates with H/M ratio changes.

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APPENDIX A

INFORMED CONSENT

I, the undersigned, have read and fully understand the following:

1. The purpose of this study is to determine how exercise that produces muscle soreness effects spinal cord exitability and relaxation.

2. Relaxation will be judged by measurement of an electrically stimulated reflex in the calf. A small, noticable, but not painful, electric shock will be applied to the back of the knee and reflex response recorded. This measurement will require approximately 15 minutes before and after the exercise bout. The electrical stimulus will range from 10 to maximal voltage to elicit the maximal reflex (generally about 110 volts). However, the actual current flow across the skin will be very small (less than 15 mA). The subjects senstation will be that of an uncontrolled muscle twitch lasting a very small fraction of a second (0.5 msec). Most subjects become drowsy or fall asleep in this proceedure.

3. The study will require seven laboratory visits. The first requires approximately 30 minutes and the subsequent visits requiring approximately 60 minutes. In the first session the subject will be orientated to the lab and experiment. Also during the first session the subject will perform a graded exercise test to determine maximal oxygen consumption. This test measures cardiorespiratory fitness. Test protocol consists of running on a motorized treadmill beginning at 5 mph at 0% grade and increasing the speed 1.0 mph at two minute intervals until 9 mph. The next stage will lower speed to 6 mph and increase grade to 5%. Subsequent stages will maintain 6mph and increase grade 5% in two minute intervals until maximal effort is attained. This test will produce short term discomfort similar to that of a one-half mile race. Total test time averages about 15 minutes with only the last two stages being difficult. During this test breathing will be monitored through a mouthpiece and heart rate by electrocardiogram (ECG).

The following visits will consist of a pre- and post- exercise reflex test as outlined in #2

above. Between the pre- and post- test you will either run on a treadmill on a downhill grade of 10% at a low intensity for 20 minutes,run on a level treadmill at a low intensity for twenty minutes or spend the twenty minutes sitting quietly (control session).

Post-testing will occur at 5 minutes after exercise or control session.

A reflex test as outlined in #2 above will also be done 24 hours after both the exercise and control sessions.

Exercise and control sessions will be determined at random after arriving for testing.

TESTING SUMMARY

Session 1 - Orientation, GXT
Session 2 - Downhill run, 20 minutes,reflex tests
Session 3 - Level run, 20 minutes, reflex tests
Session 4 - Sitting quietly, 20 minutes,reflex tests
Sessions 4-7 are reflex tests 24 hours after sessions 2-4.

4. Benefits to you include;

(1) Fitness test results showing cardiovascular fitness level and cardiovascular disease screening.

(2) Final results of the study will be made available to you indicating the effectiveness of exercise in tension control.

5. Risks to the subject include;

(1) Possible injury during treadmill exercise.

(2) Possible coronary event during maximal testing. Risks run 1 in approximately 100,000, much less for young healthy adults.

Risks are minimized by C.P.R. certification of tester and ready access to phone and emergency numbers. Risk of coronary event is all but eliminated by coronary risk screening, including resting EKG monitoring for abnormalities prior to testing.

Downhill running will result in some minor muscle soreness in the legs. Muscular discomfort

should disappear within 3 days.

6. The results of the study will be presented with subject anonymity. Subjects names will not be presented to any-one but the tester.

7. I, the subject, understand that my questions are welcome and I feel free to ask them at any time during the experiment.

8. Finally, I understand that my participation in this study is greatly appreciated, but if for any reason I wish to discontinue my participation, I may do so at any time without repurcussion.

SIGNED_____

Date_____

APPENDIX B

MUSCLE SORENESS

The purpose of this questionnaire is to evaluate the degree of muscle soreness after exercise

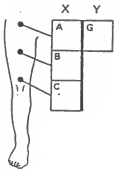
In column X please palpate (press in lightly) your muscle in the areas indicated on the diagram and then rate the degree of soreness

In column Y please indicate the general degree of soreness of the entire muscle when using or moving it (i.e. without pressing in on it).

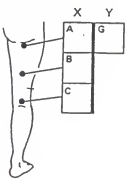
SCALE

1	2	3	4	5	6	7	8	9	10
Normal		Uncomfortable		Sore			Very sore		Very very sore

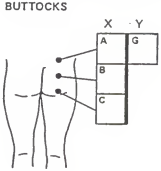
FRONT OF THIGH



BACK OF THIGH



BUTTOCKS



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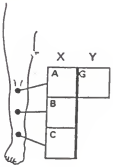

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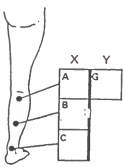

Abdomen general .....

EFFECTS OF DOWNHILL RUNNING ON MOTONEURON POOL EXCITABILITY AS MEASURED BY THE HOFFMANN REFLEX

by

DOUGLAS KENT BOWLES

B.S., Kansas State University, 1984

AN ABSTRACT OF A THESIS

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ABSTRACT

This study was conducted in order to compare alterations in motoneuron pool excitability following eccentric-biased downhill running exercise, with non-biased level running exercise. Motoneuron pool excitability was measured by the Hoffmann reflex, or H-reflex. Electrical stimulation of the tibial nerve in the popliteal fossa results in two distinct muscle action potentials (MAP's) measured at the soleus, the M- and H-waves, respectively. A stable dependent measure of motoneuron pool excitability is obtained by expressing the maximum H-wave relative to the maximum M-wave, the H/M ratio. Exercise-induced reductions in the H/M ratio have been shown in previous studies following both low intensity (40% VO2 maximum) and moderate intensity (75% VO2 maximum) exercise. Exercise treatments employed were always concentric-biased. In order to determine if differences in central nervous system responses occur after eccentric-biased exercise, this study compared H/M ratio alterations in 6 males, aged 25-34, following level (0% grade) and downhill (-10% grade) treadmill running. Relative intensities between exercise bouts were matched at 50% VO2 maximum. Non-biased exercise (0% grade) resulted in a 9.3% reduction in H/M ratio from pre-test values. Eccentric-biased exercise (-10% grade) reduced the H/M ratio 24.6% from pre-test values. Both exercise reductions were significantly different from non-exercise

control (12.6% increase, p=0.0017). Differences between exercise types approached significance (p=0.0647). Twenty-four hours post-exercise, percent changes in H/M ratios were not different from control values or zero (p=0.9673). Downhill running produced significant increases in subjective muscle soreness both immediately post-exercise (+36.1%), and 24 hours post-exercise (+166.7%). Neither control nor level running conditions increased muscle soreness. Subjective soreness appeared unassociated with alterations in the H/M ratio. Thus, both non-biased and eccentric-biased exercise result in a reduction in H/M ratio compared to non-exercise controls. Eccentric-biased exercise did not result in a greater reduction compared to non-biased exercise at similar metabolic intensities, differences being marginally significant (p=0.0647).