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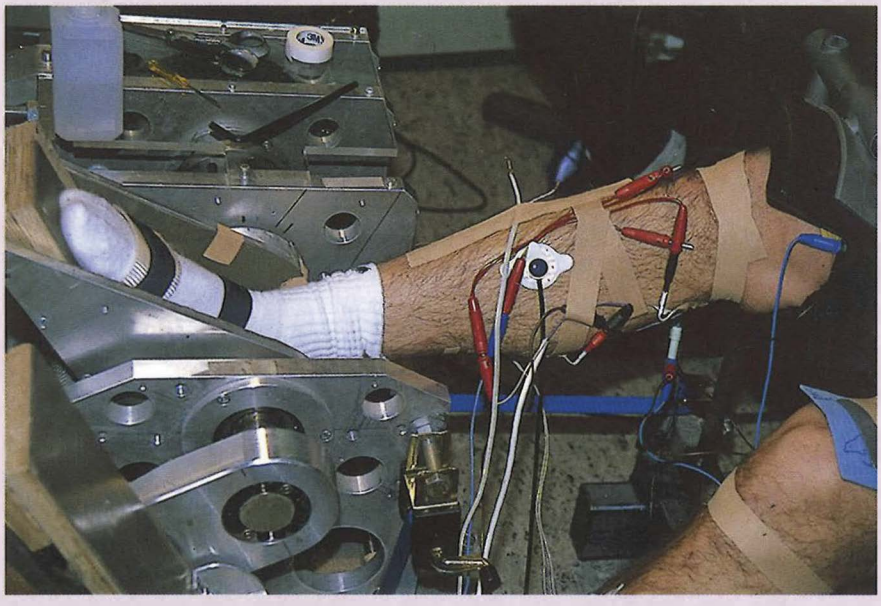
57

Janne Avela

Stretch-Reflex Adaptation in Man

Interaction between Load, Fatigue and Muscle Stiffness



UNIVERSITY OF JYVÄSKYLÄ

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Editors
Harri Suominen
Department of Health Sciences, University of Jyväskylä
Kaarina Nieminen
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ABSTRACT

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The present series of studies were designed to examine the adaptability of the neuromuscular system to different loading conditions and to prolonged stretch-shortening cycle (SSC) exercise. A special interest was in the testing of the hypothesis of central fatigue and in investigating whether there exist more direct fatigue effects on the muscle spindle itself. Reflex and voluntary functions were studied pre- and post-exercise in isolated and non-isolated conditions as well as during the experimental conditions. In the different muscle loading conditions the amount of preactivation depended of the stretch exercise mode, suggesting considerable adaptability of the neuromuscular system not only to normal gravity but even to acute modulation of the muscle loading. In every fatigue experiment, the neuromuscular response was characterized by a clear reduction in force output and by a simultaneous decrement in reflex sensitivity. The changes in reflex sensitivity were associated with changes in the active and passive stiffness properties of the muscle. This suggests that the muscle spindle may be mechanically modulated leading to a reduction in its responsiveness. In consequence, disfacilitation of the α-motoneuron pool may be operative. No recovery in the H-reflex peak-to-peak amplitude was observed as long as the fatigued muscles were kept ischemic. This may indirectly emphasize the role of the small muscle afferents as an inhibitory mechanism. It is suggested that these two central mechanisms are operative during neuromuscular fatigue. However, the relative contribution of these mechanisms depends on the fatigue task; in particular, the amount of metabolic loading, the amount of muscle damage and the amount of mechanical loading.

Key words: Neuromuscular control, preactivity, neuromuscular fatigue, central fatigue, stretch-reflex, muscle stiffness, electromyography

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LIST OF ORIGINAL ARTICLES

The present thesis is based on the following papers, which will be referred to by their roman numerals:

I Avela J, Santos PM, Kyröläinen H and Komi PV 1994. Effects of different simulated gravity conditions on neuromuscular control in drop jump exercises. Aviat Space Environ Med 65: 301-308.

II Avela J, Santos PM and Komi PV 1996. Effects of differently induced stretch loads on neuromuscular control in drop jump exercises. Eur J Appl Physiol 72: 553-562.

III Avela J and Komi PV 1998. Interaction between muscle stiffness and stretch reflex sensitivity after long-term stretch-shortening cycle (SSC) exercise. Muscle Nerve 21: 1224-1227.

IV Avela J and Komi PV 1998. Reduced stretch reflex sensitivity and muscle stiffness after long-lasting stretch-shortening cycle (SSC) exercise. Eur J Appl Physiol 78: 403-410.

V Avela J, Kyröläinen H and Komi PV 1998. Altered reflex sensitivity due to repeated and prolonged passive muscle stretching. Submitted for publication.

VI Avela J, Kyröläinen H and Komi PV 1998. Neuromuscular changes after long-lasting mechanically and electrically elicited fatigue. Submitted for publication.

VII Avela J, Kyröläinen H, Komi PV and Rama D 1998. Reduced reflex sensitivity persists several days after long-lasting stretch-shortening cycle (SSC) exercise. Submitted for publication.

CONTENTS

1 INTRODUCTION

Decline in maximal force has been considered one of the most important signs of fatigue (Asmussen 1979, Bigland-Ritchie and Woods 1984). Despite its simple form of manifestation, the mechanisms of fatigue can be rather complex. Fatigue processes can be developed in different places along the activation-contraction chain of the neuromuscular system (Bigland-Ritchie 1981), depending on the fatigue task. Therefore, fatigue has also been defined as a task-dependent phenomenon (Enoka and Stuart 1992).

In general, during the first six decades of the twentieth century, studies of muscle fatigue focused on changes in the contractile properties of muscle (peripheral fatigue), neurobiology (central fatigue) receiving only minor attention. Then, in the mid to late 1970s, a series of reports appeared on neuromuscular aspects of fatigue (e.g. Bigland-Ritchie et al. 1978, Asmussen and Mazin 1978). The preface to the book on the proceedings of the International Symposia of Muscle Fatigue held in 1994 (Miami, USA) states that "this sudden acceleration of interest in the study of neural and muscular aspects of fatigue is attributable to three factors: the practical importance and relevance of the topic; its long-neglected fundamental and clinical significance; and the type of integrated biological thought and experimentation needed to advance the field".

Usually, neuromuscular adaptation to fatigue has been studied under conditions of voluntary isometric muscle action (Bigland-Ritchie et al. 1986, Fellows et al. 1993, Woods et al. 1993) and electrical stimulation (Garland and McComas 1990, Duchateau and Hainaut 1993). However, pure isometric muscle action exists very rarely in normal human locomotion. On the other hand, muscle action of the repeated stretch-shortening cycle (SSC) type (Komi 1984) is also known to induce fatigue effects which are associated with decrease in muscle output (Gollhofer et al. 1987, Moritani et al. 1990). Such a reduction can occur even after primarily aerobic exercise such as an 85-km skiing race (Forsberg et al. 1978, Viitasalo et al. 1982) or marathon running (Sherman et al.

1984, Nicol et al. 1991). This is the reasoning behind the use of SSC muscle action in the present series of experiments.

In the present study neuromuscular function was studied during natural SSC exercises under different loading conditions and under prolonged fatigue conditions. A special emphasise was placed on studying the spinal control mechanisms of muscle function and their ability to adapt to the prescribed tasks.

2 REVIEW OF THE LITERATURE

2.1 Stretch-shortening cycle (SSC)

Human movement seldom involves pure forms of isolated eccentric, concentric or isometric muscle action. In most human motion, as in walking, running and jumping, the skeletal muscles are subjected to impact forces or conditions where external forces (gravity) lengthen the muscle. In these phases the muscles are acting eccentrically and to obtain positive work (Gavagna et al. 1968), concentric muscle action must follow. The combination of these two muscle actions forms a natural type of muscle function called the stretch-shortening cycle (SSC) (Cavagna et al. 1965, Norman and Komi 1979, Komi 1984). SSC has a well recognized purpose in muscle function: the eccentric part influences the subsequent concentric phase so that the final action is more powerful than that resulting from concentric action alone (Komi 1984).

2.2 Motor control theories

Servo control of human movement has been presented in many theories. The length follow-up servo theory of the 1950s was largely based on experiments performed on decerebrate cats. In this theory, an important role was attributed to the gamma motoneurones, regarded as the initial elements in a servo-controlled system which, via the muscle spindle endings and their stretch-reflex afferents, drive the skeletomotor output to achieve the desired muscle length in voluntary contractions (Eldred et al. 1953, Merton 1953). Later this theory has been disputed. Granit (1975) hypothesized that the skeletomotor output is not servo-driven, but rather servo-assisted by fusimotor-induced activity in muscle spindle afferents. This widely accepted servo-assistance theory implies simultaneous activation by alpha and gamma motoneurons (alpha-gamma coactivation).

Houk (1979) suggested that the spindle feedback is used together with the negative feedback from the Golgi tendon organs to stabilize the stiffness of the muscle; it is the relation between tendomuscular force and the change in muscle length (Nichols and Houk 1976). The statement by Houk (1979) is based on three components: 1) a muscular component which is purely mechanical, 2) a length-feedback component which is based on the potentiation of the stretch-reflex (facilitation of motor output) and tends to increase force, and 3) a force-feedback component which is based on negative feedback from Golgi tendon organs (inhibition of motor output) and tends to decrease force.

Much interest has been shown in the factors which may influence the efficacy of servo-assistance and gain in stretch-reflex. According to Hagbarth and Macefield (1995), such factors may include: 1) the relative strength of the fusimotor output, 2) the extent to which spindle unloading during concentric contractions counteracts the fusimotor-induced internal activation of spindle endings, 3) the extent to which the history of movements and contractions affects the inherent slackness of intafusal muscle fibers, 4) variations in the pre-motoneuronal excitability of segmental and supraspinal stretch reflex arcs and 5) the level of excitability of alpha motoneurons.

Some animal studies (Loeb and Hoffer 1981, Prochazka and Wand 1981) have suggested that a task-dependent dissociation exists between skeletomotor and fusimotor outputs. However, this has not been proven in human studies (Burke 1981, Hagbarth 1993). As judged by these studies, Hagbarth and Macefield (1995) suggested that factors 4) and 5) (see the above paragraph) might play an important role in task-dependent variations in fusimotor-driven assistance of skeletomotor output.

2.3 Neuromuscular fatigue

Fatigue is a very complex phenomenon and it has been described as a loss of force generating capacity or as an inability to sustain further exercise at the required level (Asmussen 1979, Bigland-Ritchie and Woods 1984, Ewards 1981, Enoka and Stuart 1985). Thus, Gandevia et al. (1992) defined fatigue as any reduction in the force- or velocity-generating capacity of a muscle that is alleviated by rest.

2.3.1 Task dependency of neuromuscular fatigue

Neuromuscular fatigue has been studied under a wide range of experimental protocols. These studies have revealed that fatigue is not a consequence of any unique and common set of factors but, rather, that it can be induced by a variety of mechanisms. Therefore, the term "task dependency" has been used to describe the relationship between the details of the task in the underlying mechanisms and the sites associated with fatigue (see also Enoka and Stuart 1992). When the details of the task vary, the mechanisms underlying fatigue

also vary. Bigland-Ritchie et al. (1984) have stressed that the fatigue process begins immediately after the onset of activity. Therefore, it seems that for a given task, the mechanisms contributing to fatigue will vary as the task proceeds. Different tasks can be performed by using muscles in different ways to generate force that may be either sustained or repeated intermittently. In each contraction, the muscle may lengthen and/or shorten or its length may remain unchanged. Furthermore, the intensity and duration of the activity, the speed of contraction and the extent to which the activity is continuously sustained may vary according to the task.

The site of fatigue might be due to a failure anywhere along the bath that finally results in force production, from the descending command that activates the α-motoneurones of a motor pool, to interaction of the contractile proteins within single muscle fibers (Botterman 1995). These major potential sites affected during a fatiguing contraction have been divided into three general categories by Bigland-Ritchie and Woods (1984): those which lie within the central nervous system (CNS), those concerned with neural transmission from the CNS to muscle and those within the individual muscle fibers. Fitts (1994) has defined the affected mechanisms more precisely as being: 1) excitatory input to supraspinal motor centers, 2) excitatory drive to α-motoneurons, 3) modulation of interneuronal circuits, 4) motoneuron excitability, 5) peripheral reflex activity from small diameter afferents, 6) muscle spindle activity, 7) neuromuscular transmission, 8) sarcolemma excitability, 9) excitation-contraction coupling and 10) metabolic energy supply and metabolite accumulation. In general, a reduction of force-producing capacity is mainly due to impairments of peripheral mechanisms (7-10) (Merton 1954, Beelen et al. 1995), while central mechanisms (1-6) have the potential capability to adapt to these changes (Bigland-Ritchie 1981).

2.3.2 Central fatigue

Late last century Mosso wrote perceptively about muscle performance and the role played by the central nervous system (CNS) (Candevia et al. 1995). He measured fatigue in a simple repetitive task in which a weight was moved as far as possible by flexors of a finger at a set rate and realized that the role of the CNS could be deduced by electrical stimulation of the relevant muscle. In the twentieth century, however, physiologists have either ignored the importance of volition or considered it too difficult to measure. For example, Hill (1926) clearly recognized that for other than elite athletes, insufficient voluntary drive could limit performance.

Only few techniques exist to asses the degree to which voluntary isometric performance reaches the optimal maximum. Mosso (1904), Merton (1954) and Bigland and Lippold (1954) suggested that maximal voluntary force could only be judged by reference to what can be achieved by non-voluntary electrical stimulation of motor output. The only problem of this approach is that it leads to considerable pain while stimulating large muscle groups. Interpolation of an electrical stimulus to the motor nerve during a maximal effort should produce an increment in force if the stimulated axons are not all recruited voluntarily or

if the discharge rate is sub-tetanic. This technique has been applied to proximal muscles such as the quadriceps and elbow flexors (Bigland-Ritchie et al. 1983, McKenzie and Gandevia 1991) and tibialis anterior and soleus (Gandevia and McKenzie 1988).

In several studies (Merton 1954, Bigland-Ritchie 1981) twitch interpolation has not been able to overcome the decline in maximal force. However, some alternative results have also been presented (McKenzie and Gandevia 1991, Löscher et al. 1996). McKenzie and Gandevia (1991) demonstrated by means of a twitch interpolation a small degree of central fatigue which developed progressively during a series of limb muscle contractions.

Despite the uncertainty as to the existence of central fatigue in several studies where fatigue has been induced by prolonged SSC exercise (Nicol et al. 1991), repeated eccentric and concentric exercises (Komi and Rusko 1974, Komi and Viitasalo 1977) and sustained isometric contraction (Bongiovanni and Hagbarth 1990), some reduction in neural input to muscle has been observed. This has been confirmed by a decline either in maximal aEMG or in the discharge frequencies of the motor units. This decline has been regarded as an expression of muscle wisdom, since it apparently serves to ensure appropriate economical activation of fatiguing muscle (Marsden et al. 1983): as twitch relaxation times increase during fatigue, the fusion frequency decreases such that lower firing rates are required for individual motor units to generate their maximal force (Bigland-Ritchie and Woods 1984).

The weakness of these two methods of assessing the degree of muscle activation is that they are not able to identify the mechanism which causes possible central fatigue. These potential mechanisms are the following: 1) supraspinal fatigue (Brasil-Neto et al. 1994), 2) peripheral reflex inhibition (Garland and McComas 1990), and 3) disfacilitation of the α-motoneuron pool due to muscle spindle fatigue (Bongiovanni and Hagbarth 1990). In addition, mechanisms such as the late adaptation of motoneurons (Kernell and Monster 1982) and neuromodulation of membrane conductances (Hultborn and Kiehn 1992) have also been suggested.

2.3.2.1 Supraspinal fatigue

Fatigue of the supraspinal centres has been verified in some studies using transcranial magnetic stimulation (Brasil-Neto et al. 1993 and 1994). These experiments suggest that fatigue results from reduced efficiency in the generation of the motor command as a consequence of cortical neurotransmitter depletion. According to Davis and Bailey (1997), these neurotransmitters include serotonin, acetylcholine and dopamine. Of these, serotonin (5-HT) has received most attention recently because 5-HT synthesis is increased during prolonged exercise, and increases in brain 5-HT have been associated with lethargy and loss of motor drive (Davis and Bailey 1997). However, Gandevia et al. (1995) have shown that the changes in cortical excitability can be dissociated from the presence of central fatigue. They suggested, therefore, that it is unlikely that the changes in cortical excitability are necessarily the direct cause of central fatigue.

2.3.2.2 Presynaptic inhibition

According to Andreas et a. (1985), group III afferents are thinly myelinated fibers and conduct impulses between 2.5 and 30 $m \cdot s^{-1}$ in cats and dogs. Group IV afferents are unmyelinated fibers with a conduct velocity of less than 2.5 $m \cdot s^{-1}$. Both groups are free nerve endings surrounded by Schwann cells. A small portion of the ending is bare, and this area is thought to be the site of action for the stimuli that activate them. Many of these muscle afferents have been shown to be stimulated by intraarterial injection of metabolic products of muscular contraction. These products include bradykinin (Mense 1977), arachidonic acid and prostaglandin E_2 (Mense 1981, Rotto and Kaufman 1988), potassium (Kniffki et al. 1978) and lactic acid (Rotto and Kaufman 1988, Sinoway et al. 1985). It should be noted that these metabolic products have non-significant direct effect on the discharge of spindle afferents and Golgi tendon organs.

From animal studies (Cleland et al. 1984) it is known that these small muscle afferents make a powerful input to inhibitory interneurons which could then inhibit either Ia terminals (Duchateau and Hainaut 1993) and/or the α-motoneuron pool as originally suggested by Bigland-Ritchie et al. (1986). The conclusion in favor of presynaptic inhibition by Duchateau and Hainaut (1993) was based on their findings that, firstly, the decrement in H-reflex does not recover as long as the fatigue-induced metabolic accumulation is maintained by ischemia and, secondly, that the time course for the H-reflex decrease during fatigue is rather slow.

2.3.2.2.1 The role of muscle damage in presynaptic inhibition

In addition to the by-products of muscle metabolia, group III and IV muscle afferents are known to be stimulated by biochemical substances, such as bradykinin and prostaglandins, which are known to be released in cases of muscle damage (Mense 1977, Rotto and Kaufman 1988). In general, it is known that eccentric muscle action causes the most soreness and damage to the muscle (McCully and Faulkner 1985, Stauber 1989). According to Warhol et al. (1985), muscle damage may also occur even after a primarily aerobic exercise such as marathon running. It is well documented that the cytoskeletal and myofibrillar abnormalities observed after eccentric muscle action reach a peak 2-3 days after exercise (Friden et al. 1983a, b). Armstrong (1991) divided the whole muscle damage process into four stages: the initial, the autogenetic, the phagocytic and the regenerative.

The initial stage (I) includes the trigger for the whole damage process. Much evidence exists that the initial local damage is mechanically induced (Newham et al. 1983, Lieber and Friden 1993). Histological studies have reported extensive disorganization and disruption of the myofibrillar structure and intermediate filaments, resulting in classically observed Z-line streaming (Waterman-Storer 1991, Newham et al. 1994).

The autogenetic stage (II) corresponds to the first 3-4h following the injury and marks the beginning of the degradation process of the membrane structures (Armstrong 1991).

The phagocytic stage (III) is characterized by an inflammatory response in the muscle tissue which may last for 2-4 days (Kihlsrom et al 1984). The inflammatory response begins with the mobilization of neutrophils and leucocytes (Evans and Cannon 1991). Once activated, neutrophils provide a fresh supply of mediator, which may be partly responsible for amplifying and delaying the inflammation. This stage is also characterized by the presence of indirect indicators of the muscle damage in the blood, such as skeletal troponin I (TnI) and serum creatine kinese activity (S-CK) (Sorichter et al. 1997). It should be noted that although the peak values of S-CK do not reflect the absolute amount of muscle damage (Mair et al 1995), their relative changes might be of some relevance to the detection of tissue inflammation. Faulkner et al. (1993) also defined this stage as secondary injury. It has been suggested that it is responsible for the secondary reduction in some functional parameters (MacIntyre et al. 1996) through the activation of group III and IV muscle afferents (Nicol et al. 1996).

The regenerative stage (IV) begins on days 4-6 and reflects the regeneration of muscle fibers (Armstrong 1991).

2.3.2.3 Disfacilitation

It has been proven that muscle afferent feedback causes a net facilitation of voluntarily generated skeletomotor output in human subjects. Hagbarth et al. (1986) and Bongiovanni et al. (1990) showed that peak motor unit firing rates during brief MVC of nonfatigued muscles were reduced by partial nerve block. Controversially, they also found that the decline in motor unit firing rates, which is normally seen during sustained MVC, was absent during partial nerve block. In addition, in the absence of muscle afferent feedback, the firing rates of single motor axons were lower than those of normally innervated motor units recorded in separate experiments (Gandevia et al. 1990, Gandevia et al. 1992). Direct proof that muscle spindle feedback does decline during constant-force voluntary contraction comes from microneurographic recordings of the discharge of single muscle spindle afferents (Macefield et al. 1991). The discharge of most muscle spindle afferents decreased by 50% during contractions sustained for at least 60 s, when increasing effort was required to maintain the isometric target force. These results have been taken as an evidence that the reflex facilitatory influence provided by muscle afferents declines as fatigue develops. This phenomenon is called disfacilitation.

The reason for the decreased muscle spindle afferent activity is, however, complicated. Owing to technical difficulties, no recordings have been taken from human fusimotor neurons during the development of muscle fatigue. Bongiovanni and Hagbarth (1990) were able by muscle vibration to counteract the declined MVC motor unit firing rates achieved by partial block of the deep peroneal nerve. They took this as a possible evidence for a reduction in fusimotor support to the muscle spindles during fatigue. However, they also

suggested the possibility of some more direct fatigue effects on the intrafusal fibers themselves, as did also Komi and Nicol (1998).

Direct fatigue effects on the intrafusal fibers have not been well established. Glycogen depletion of intafusal fibers have been demonstrated in some animal studies (Yoshimura et al. 1996, Decorte et al. 1984). In addition, Fukami (1988) studied the effect of NH_3 and CO_2 on the sensory ending of cat muscle spindles. He concluded that immediate suppression in impulse activities was most likely caused by changes in the pH of the sensory ending.

2.4 Muscle elasticity in human performance

Elasticity of the tendomuscular complex plays an important role in enhancing both the effectiveness and the efficiency of human performance (Komi 1984). The stretching of an active muscle results in a storage of elastic energy, which can then be utilized during the following concentric muscle action (Cavagna et al. 1965). Classical models (Hill 1938) describe the behavior of muscles in three functional components. The contractile and force generating component is characterized by force-velocity and force-length relationships. The series elastic component (SEC) and parallel elastic component (PEC) represent elastic structures of the muscle according to their geometrical relationship with the contractile component. Tendons, along with connective tissues within the contractile proteins, are a major part of the SEC, while PEC consists of muscle fascia, connective tissue and sarcolemma. These tissues are passive elastic structures, while cross-bridges and myofibrils are the active components of the SEC. However, it is also suggested that the cross-bridges themselves are elastic structures (Huxley and Simmons 1971).

The ability of the muscle to store and utilize elastic energy is dependent on the stretching velocity and the muscle length (Cavagna et al. 1965), as well as the force attained at the end of the prestretch and the coupling time between the eccentric and concentric phases of the SSC (Bosco et al. 1981). A short and rapid stretch with a short coupling time and high force at the end of the prestretch creates a good precondition for utilizing tendomuscular elasticity (Cavagna et al. 1968). In other words, for effective utilization of elastic energy the muscles must show a high degree of elastic stiffness. Gollhofer et al. (1992) emphasized that an effective stretch-shortening cycle can be performed only if there is a marked stiffness of the muscle-tendon complex throughout the total period of contact. However, high stiffness responses have been found to be present only with active reflex functions (Nichols 1974). Dietz et al. (1979) also emphasized the interaction between elastic recoil in the SSC and the reflex contribution during the stretching phase. Burke et al. (1978) reported that during active lengthening contraction (eccentric action) the muscle spindle responses were greater than during passive stretch of similar amplitude and velocity, suggesting increased fusimotor outflow and reflex responses. Horita et al. (1998) as well as Gollhofer et al. (1984) emphasized the importance of the pre-

activity before landing. They suggested that this activity is important in the stiffness regulation of the muscle and contributes to the subsequent high stiffness of the SEC and finally regulates the whole SSC performance.

Komi and Gollhofer (1997) have concluded that an effective SSC depends on three fundamental conditions: 1) well timed preactivation of the muscle before the eccentric phase; 2) a short and fast eccentric phase; and 3) an immediate transition (short delay) between stretch and shortening of the muscle.

2.4.1 The role of titin in muscle elasticity

In addition to actin and myosin filaments inside a sarcomere, vertebrate striated muscle contains a third filament system. Sjostrand (1962) noted in electron micrographs of sarcomeres stretched beyond overlap of thick (myosin) and thin (actin) filaments that very thin (2-4 nm) filaments bridged the region between the nonoverlapping A- and I-bands (figure 1). He thus coined the term gap filaments, to describe those which appeared between the actin and myosin filaments. These filaments have been later labeled titin (also connectin) and nebulin (Wang and McClure 1978). Titin has also been localized in between myosin filaments and the Z line (Maruyama et al. 1985) and, therefore, is of special interest in regard to muscle elasticity. Evidence of titin's elastic nature was demonstrated by Maruyama et al. (1989) when they reported that binding sites of titin monoclonal antibodies returned to their original position after extreme stretch and release of isolated myofibrils. Granzier et al. (1997) also emphasized the elastic nature of titin and suggested that its elastic properties greatly contribute to the passive force in muscle. It has also been suggested that the differences observed in the elasticity of fast and slow fiber types may be related to the percentage of titin found in each (Askter et al. 1989). Labeit and Kolmerer (1995) found that in the I-band region, comparison of titin sequences from muscles of different passive tension identifies two elements that correlate with tissue stiffness, therefore, suggesting that titin may act as two springs in series.

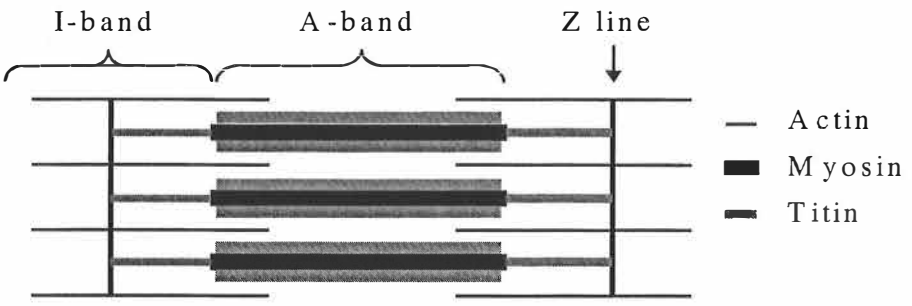

FIGURE 1 A highly schematic representation of the sarcomere (modified from Waterman-Storer 1991).

Horowits and podolski (1987) suggested that the titin filament is also bound to the myosin filament in the A-band region. They thus proposed titin to be responsible also for maintaining the central location of thick filaments in relaxed muscle. Therefore, exercise induced modulation and/or damage of titin filaments can possibly result in Z-disc streaming and irregularities of filament overlapping, which obviously leads to impaired ability of sarcomere to produce force.

3 THE PURPOSE OF THE STUDY

Neuromuscular fatigue has quite extensively been studied in both animals and in humans. Such studies have mostly concentrated on the area of peripheral fatigue. Since 1980s, the aspect of central fatigue, and its peripheral mechanisms in particular, have also been of interest. The fatigue task generally used in these more recent studies has usually been either maximal or submaximal isometric muscle action induced voluntarily or by external electrical stimulation. Because of the unnatural nature of those fatigue models, generalization of those results to normal human movement (SSC) is difficult. Therefore, in the present series of experiments SSC muscle action has been taken as an activation model in the exercises used. However, isometric conditions have also been utilized in some of the pre- and post-fatigue measurements. The detailed purposes of the present study can be characterized as follows:

1) It has been suggested that muscle preactivation is pre-programmed from supraspinal centers, the correct timing and sequence of the muscles having been learned through previous experience (Melwill Jones and Watt 1971). However, Greenwood and Hopkins (1976), have suggested that the vestibular apparatus might modify the central programme. Therefore, the first aim of the present study was to try to clarify further the control mechanisms of muscle precontact activation. In addition, also of interest was to investigate how different modulations of preactivation influence its interactions between stretch-reflex responses and the muscular output itself during the contact period (I and II).

2) Asmussen and Mazin (1978) originally suggested that the decline in the motor unit activation during fatigue depends on the contracting muscle itself through some reflex pathways. This interaction has since been shown more directly by Nicol et al. (1996) in intensive and exhaustive SSC exercise. Therefore, the second purpose of the present study was to investigate whether reduced reflex sensitivity

can be induced by long-term SSC exercise and whether it is associated with reductions in muscle stiffness and also in muscle performance (III, IV and VII).

3) There are in principle two mechanisms which are potentially responsible for the reduced reflex sensitivity induced by fatigue, including disfacilitation of the α-motoneuron pool (Bongiovanni and Hagbarth 1990) and peripheral inhibition of the α-motoneuron pool (Bigland-Ritchie et al. 1986) and/or Ia afferent terminals (Duchateau and Hainaut 1993). On the bases of indirect evidence, Bongiovanni and Hagbarth (1990) have suggested that disfacilitation can be caused, in addition to withdrawal of fusimotor support to the muscle spindle, by direct fatigue processes in the intrafusal fibres of the spindle itself. In order to seek evidence for these more direct fatigue effects on the muscle spindle in the present study, a prolonged passive stretches condition was designed (V) and possible interactions between reflex sensitivity and the compliance characteristics of the muscle-tendon complex were tested (V, VI and VI).

4) Contradictory results exist in the literature on whether central fatigue can reduce muscle force output (McKenzie and Gandevia 1991) or not (Merton 1954). Because of the difficulty in quantifying supraspinal fatigue (Brasil-Neto et al. 1994), the central fatigue hypotheses was investigated under an isolated active condition whereby the possible effects of supraspinal fatigue are minimized by the use of electrical stimulation. Some attempts were also made to characterize the possible peripheral fatigue induced by the same protocol (VI).

5) Several studies have demonstrated a bi-phasic recovery pattern after fatigue stimulation for parameters related to the neuromuscular function (Faulkner et al. 1993; MacIntyre et al. 1996). This pattern involves an initial decline followed by an early recovery and a delayed second decline. Nicol et al. (1996) clearly showed this also to be the case with stretch-reflex responses after intensive SSC fatigue. Therefore, the final aim of the present study was to investigate the control mechanisms behind this delayed secondary impairment of the short latency stretch-reflex function (VII).

4 RESEARCH METHODS

4.1 Subjects

A total of 54 subjects participated in these studies. There were nine subjects in the first experiment, which is reported in papers I and II. The second experiment included in total 18 subjects, nine in the experimental group and nine as controls. The experimental subjects were selected according to their personal best marathon times of the season. This avoided any delay in the post-marathon tests for any of the subjects after the marathon race. The range of their best times was from 2 h and 20 min to 3 h and 30 min. Their results are reported in papers III and IV. For the third experiment, reported in paper V, the number of subjects was 20, ten of whom were randomly selected for the fourth experiment, which is reported in paper VI. Finally, eight subjects, seven male and one female, were selected for the fifth experiment, reported in paper VII. The range of their best times in running the marathon varied from 2h and 45min to 3h and 15min. All the subjects were healthy and none of them had any history of neuromuscular or vascular diseases. They were fully informed of the procedures and the risks involved in these studies and they gave their informed consent (Code of Ethics of the World Medical Association, Declaration of Helsinki). When appropriate, the project plans were approved by the University Ethical Commission. The subjects were also allowed to withdraw from the measurements at will. Table 1 presents the physical characteristics of the subjects in these five experimental series.

TABLE 1 Mean (±SD) of the physical characteristics of the subjects. Exp. = number of experiment, E. group = experimental group and C. group = control group.

Variable	Exp. 1.	Exp. 2. E. group	Exp. 2. C. group	Exp. 3.	Exp. 4	Exp. 5
Age (yr.)	24 ±4	32 ±5	28 ±3	27 ±6	34 ±5	29 ±4
Height (cm)	185 ±8	176 ±5	179 ±5	182 ±7	182 ±5	182 ±5
Body mass (kg)	79.6 ±6.4	69.4 ±6.8	75.7±4.8	78.5 ±9.9	80.7 ±9.3	82.0 ±7.8

4.2 Experimental design

4.2.1 Experiment 1.

In experiment 1 the subjects performed exercises of a stretch-shortening cycle type consisting of three different drop jumps in three to five different loading conditions. The order of the conditions and the type of the drop jump exercise was selected randomly for each subject. In each condition six successful maximal drop jumps were performed with a one-minute recovery period. Recovery phases between the different conditions varied from five to 10 minutes. The jumps were done with the least amount of knee bending and the hands were kept on the hips throughout the entire movement. In paper I only the results of the lifting block jumps (LBJ) with five different loading conditions are reported. Paper II reports all three types of drop jump exercises each with three different loading conditions.

The different drop jump exercises and loading conditions were as follows:

1) Normal drop jumps (DJ) (Komi and Bosco 1978), performed from three different dropping heights: 0.66 m (DJ66), 0.46 m (DJ46) and 0.29 m (DJ29) (figure 1). The selected parameters of these jumps can be seen in table 2.

2) Sledge jumps (SlJ) using a special sledge apparatus (Kaneko et al. 1984) with five different extra loads: body weight +20% (SlJ+20), body weight (SlJ0), and body weight -20% (SlJ-20) (figure 1). The changes in the body weight were obtained by extra loads. Reduced body weight conditions were possible due to a low and constant rate acceleration of 4.68 $m \cdot s^{-2}$. This rate of acceleration was achieved by the inclination of the sledge rail, which was 28.5 degrees. The impact velocity was set constant at 3.0 $m \cdot s^{-1}$. The dropping height was 0.96 m in all three jumping conditions (table 1).

3) Lifting block jumps (LBJ) with three different accelerations: gravity +20% (g+20), gravity +10% (g+10 only in paper I) gravity (g), gravity -10% (g-10 only in paper I) and gravity -20% (g-20). Simulated changes in the accelerations were obtained with a special Lifting Block System (LBS) (figure 2) (designed and

manufactured in our laboratory). With differently placed extra loads (EL) different accelerations could be achieved. The lifting block wheel ratio (LBWR) was three to one, which allowed the EL to pull the subject down at a higher rate of acceleration than the 9.81 $m \cdot s^{-2}$ achieved when putting the EL at the end of the lifting wheel rope. For accelerations less than normal gravity the EL was placed at the end of the upper rope of the system. Dropping velocity at the moment of touchdown was set at 3.0 $m \cdot s^{-1}$ in all three jumping conditions. The dropping height (h) was calculated according to the following formula:

$$h = \frac{v^2}{2a} \quad (1)$$

The EL needed in the different conditions was obtained by:

$$EL = \frac{m(a-g)}{g} + OL \cdot (LBWR) \quad (2)$$

g = gravity (9.81 $m \cdot s^{-2}$)
a = desired gravity condition (g ± x%)
v = constant velocity (3.0 $m \cdot s^{-1}$)
m = mass of the subject
OL = opposite load (2 kg or LBWR · 2 kg)

LBWR was used only when a condition greater than 9.81 $m \cdot s^{-2}$ was required. The corresponding impact parameters for the LBS are shown in table 2.

In all the jumping conditions the extra-loads were blocked-off at the moment of touchdown by means of an electric device. An electric impulse from a resistive platform (Digitest Ltd, Muurame, Finland) placed over the force plate served as a trigger signal for the release. By releasing the extra loads at the beginning of the braking phase, the mechanical effects of the different gravity conditions could be reduced and only the indirect effects on the muscle stretching velocities were obtained through possible changes in the preactivation of the muscles.

TABLE 2 The different jumping conditions and the modified impact parameters.

Jump condition	Dropping height (m)	Acceleration ($m \cdot s^{-2}$)	Impact velocity ($m \cdot s^{-1}$)	Mass (%)	Mean impact power ($Nm \cdot s^{-1}$)
g+20	0.38	11.77	3.0	100	2811
g+10	0.42	10.79	3.0	100	2577
g	0.46	9.81	3.0	100	2343
g-10	0.51	8.83	3.0	100	2109
g-20	0.57	7.85	3.0	100	1875
DJ66	0.66	9.81	3.6	100	2811
DJ46	0.46	9.81	3.0	100	2343
DJ29	0.29	9.81	2.4	100	1875
SlJ+20	0.96	4.68	3.0	120	2811
SlJ0	0.96	4.68	3.0	100	2343
SlJ-20	0.96	4.68	3.0	80	1875

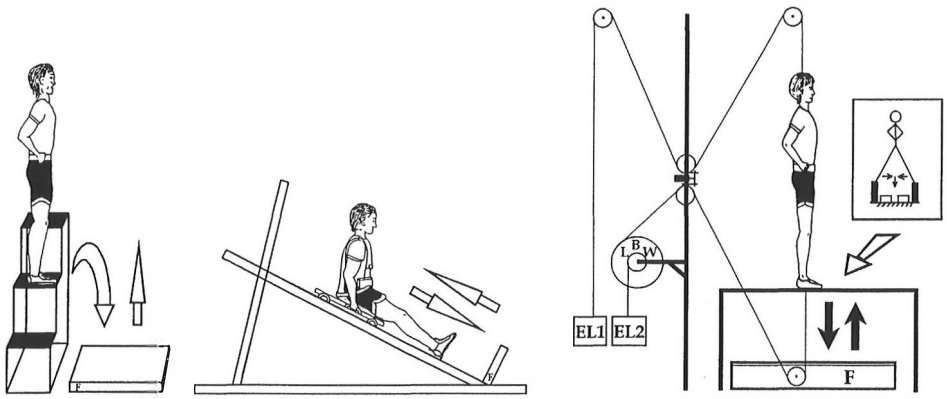

FIGURE 2 Schematic presentation of the different drop jump exercises. Normal drop jumps (DJ) on the left, sledge jumps (SlJ) in the middle and lifting block jumps (LBJ) on the right.

The impact effect of the different jumping conditions was calculated in terms of power according to the following formula:

$$P = v\,(m \cdot a) \qquad (3)$$

P = impact power
v = impact velocity
m = body mass
a = acceleration

Three sets of comparable loading conditions were available with regard to the impact effect: DJ66, SJ+20 and g+20; DJ46, SL0 and g; DJ29, SJ-20 and g-20 were set to correspond to the same impact effects (table 1).

4.2.2 Experiment 2.

In experiment 2 fatigue was induced by marathon running and was, therefore, designed as a before and after marathon study. The marathon run was selected as a way to induce fatigue because by its very nature it involves repeated long term SSC muscle actions (approximately 10 000). The before-marathon tests took place one hour before the actual marathon race and the after-marathon tests were performed immediately after the run. A real competition situation was selected to confirm high levels of motivation, performance and, consequently, fatigue. The control group participated only in the test and retest sessions, separated by a period of one week.

The testing protocol included various jumps on a special sledge ergometer (Kaneko et al. 1984) (figure 2) and controlled patellar reflex measurements in a well stabilized position.

Patellar reflexes were measured immediately before and after the marathon run in the right leg with the subject seated on the dynamometer chair in a relaxed position.

The sledge ergometer jumps (Figure 2) consisted of ten maximal drop jumps performed in a sitting position from three predetermined dropping heights. The optimal dropping height (best rebound height) of each subject was first determined on a sledge using a short series of maximal single SSC repetitions in which the subject was dropped from a progressively increasing heights and instructed to rebound as high as possible. The actual test jumps were then determined as the optimal dropping height minus 30 percent (70%) the optimal dropping height (100%) and the optimal dropping height plus 30 percent (130%). Each of the test jumps was performed with maximal effort. The recovery time between the dropping heights was 3 minutes and between individual jumps 5 seconds. It could be argued that this type of test itself might induce some fatigue effects. However, in the sledge ergometer the subjects were able to relax fully between the individual jumps and the total muscle activation time during the series of ten jumps was only five seconds. The order of the different dropping heights was randomized. The sledge ergometer jumps included also two pure maximal concentric jumps performed from a 90° knee angle. The same protocol was used for both experimental and control groups.

The results of the 100% sledge jumps are reported in paper III and the results for the patellar reflexes and 130% and 70% sledge jumps in paper IV.

4.2.3 Experiment 3.

In experiment 3 all twenty subjects underwent prolonged and repeated passive stretching (RPS) of the calf muscles, which lasted for one hour. RPS was induced by an ankle ergometer (Nicol et al. 1996) which was similar to that used by Gollhofer and Schmidtbleicher (1989). In the ergometer the stretching was applied by a motor torque device (Geisinger, 150Nm) controlled by a digital feedback-system. In all the experimental conditions the subject sat in a chair. Depending on the testing conditions, the thigh of their right leg or left leg was fixed and the foot was mounted on the rotation platform so that the rotation axes of the ankle joint and motor drive coincided, thus only allowing motion around the ankle joint. The initial ankle position was 90 degrees and the knee angle was fixed at 120 degrees. The stretching amplitude of the dorsiflexion of the ankle joint was 10 degrees and the corresponding average velocity of the stretch was 3.5 $rad \cdot s^{-1}$. The frequency of all the stretches was 1.5 cycles per second. An example of the mechanical stretching signal can be seen in figure 4. Throughout the experimental procedure, the legs were warmed with an infra-red lamp and the temperature of the skin was controlled during every test unit. Thus, a constant skin temperature could be ensured by adjusting the distance of the lamp from the skin according to the existing skin temperature (30 ±0.5°C). At the end of the fatigue protocol a blood pressure cuff was wrapped around the middle portion of the right thigh and inflated to at least 200 mmHg. This pressure was maintained while the measurements immediately after fatigue were performed. This procedure served to retain the possible metabolic fatigue substances in the fatigued muscles throughout the measurements.

All twenty subjects performed the one hour RPS (protocol 1). In this test the subjects were instructed not to resist the mechanical stretching of the calf muscles. Six of the 20 subjects were also tested for the effect of ischemia (protocol 2) and for the recovery of reflex excitability (protocol 3). The rather complex experimental protocol is summarized in figure 3.

In the RPS (protocol 1), pre-tests included the measurements of isometric maximal voluntary contraction (MVC) and 50% MVC. These measurements were followed by the measurement of the maximal compound action potential (M-waves) and the Hoffmann-reflex (H-reflexes). The three post-test measurements followed the same protocol; the first of these, however, was performed with ischemia. Blood samples were drawn immediately after and 5 minutes after the RPS from the ulnar vein and from the fingertip.

Stretch reflexes were measured as a bout of ten consecutive stretches for each leg at the very beginning of the RPS, after every 15 minutes and at the very end of the RPS. The RPS signal also served as a stimulus to induce stretch reflexes. Therefore, the experimental leg underwent continuous stretching, while the control leg was treated only during these short bouts of stretch reflex tests. Recovery was tested 15 minutes and 30 minutes after the RPS.

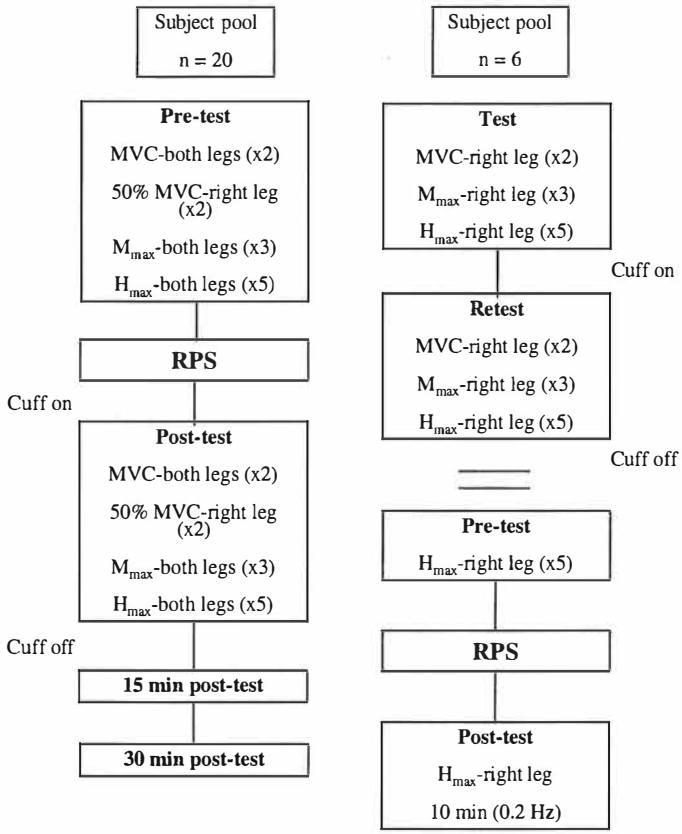

FIGURE 3 Experimental protocol illustrating protocol 1 (RPS) on the left and protocol 2 (ischemia control) and protocol 3 (H-reflex recovery) on the right. MVC = maximal voluntary contraction, M_{max} = maximum M wave and H_{max} = maximum H reflex.

In the first control test (protocol 2), exactly the same pre-test (normal) and post-test (ischemia) protocol was repeated without the RPS. However, in the second control test (protocol 3), the RPS was included and was preceded only by the measure of the maximal H-reflex (five measurements) of the experimental leg. During the post-test, only the recovery of the maximal H-reflex peak-to-peak amplitude was measured, using a stimulus frequency of 0.1 Hz.

In the results and discussion sections, this experiment is also referred as a passive fatigue experiment.

4.2.4 Experiment 4.

In experiment 4, fatigue was induced in the calf muscles of the right leg by active stimulation, consisting of simultaneous electrical stimulation (ES) and

mechanical stretching for one hour. This was performed using the same ankle ergometer as in experiment 3. The mechanical stretching was generated by the servo motor system of the ankle ergometer which was controlled by a microcomputer (A). The stretching was set to produce a 10 degree ankle joint dorsiflexion with an average stretching velocity of 3.5 $rad \cdot s^{-1}$ and a frequency of 1.5 cycles per second (figure 3). The evoked-potential measuring system, including a precision impulse generator (MEB-5304K, Nihon Kohden), was used for the simultaneous ES of the right calf muscles. The stimulator was controlled by a second microcomputer (B), which was synchronized to the mechanical stimuli by computer A. The stimulation intensity was set to a level which would induce 10% of the maximal voluntary contraction (MVC) torque. This intensity was selected to ensure that the exercise was aerobic in energy demand. The mechanical stimulation was party isometric, since, according to Folkow and Halicka (1968) and Sjögard et al. (1986), blood flow becomes restricted and unevenly distributed when the isometric force rises above about 10 to 20% of MVC. The stimulation frequency was 30 Hz (duration 0.2 ms), which is around the initial firing rate during sustained isometric contraction (Bigland-Ritchie et al. 1986). The ES covered each mechanical perturbation unit with a pre-stimulation of 75ms (figure 4). The stimulation electrodes used in the ES, as well as in the low-high frequency test, were self-adhering medical electrodes (5cm x 5cm) (StimTrode, Axelgaard Manufacturing Co., Denmark). The electrodes were placed percutaneously on the right gastrocnemius muscle so that two anodes were attached to the proximal head of the lateral and medial gastrocnemius muscles and one cathode was located distally on the muscle-tendon region.

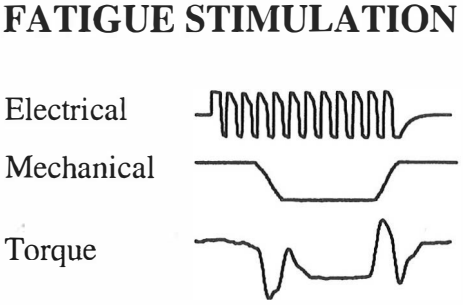

FIGURE 4 An example of one repetition of simultaneous mechanical and electrical stimulation during the fatigue task. The mechanical part of the stimulation represents the signal used in the passive experiment (3) and in the stretch reflex tests of both experiments (3 and 4).

All ten subjects performed the fatigue protocol (protocol 1), consisting of simultaneous mechanical and electrical stimulation of the right calf muscles for one hour. The subjects were then randomized into two groups of five subjects tested for the recovery of the reflex excitability. In these tests the recovery of the maximal H-reflex was followed for ten minutes either with (protocol 2) or

without (protocol 3) a five-minute period of ischemia. The experimental protocols are summarized in Figure. 5.

In the fatigue protocol (protocol 1), pre-fatigue measurements were performed in the following sequence: isometric maximal voluntary plantarflexion (MVC), 50% MVC, MVC with superimposed double twitch, maximal H-reflexes with maximal M-responses and, finally, response of the relaxed triceps surae muscle to electrical stimulation (ES) at frequencies of 20 Hz and 80 Hz. These measurements were performed on the right leg, which served as the experimental leg. Prior to these measurements MVC and maximal H- and M-responses were measured for the non-fatigued left leg, which served as the control leg for these selected parameters.

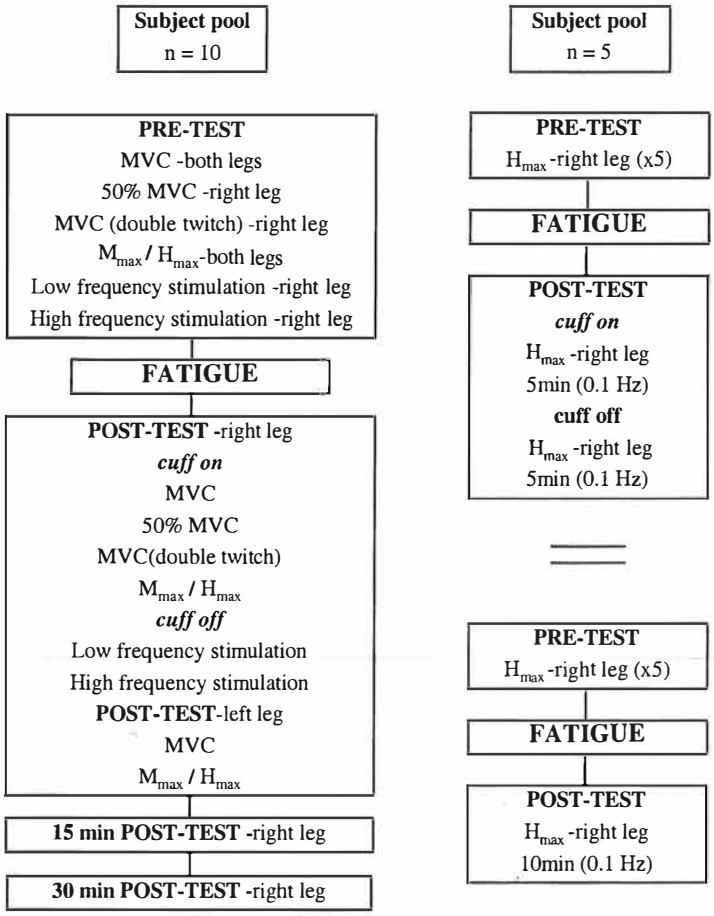

FIGURE 5 Experimental protocol illustrating the fatigue protocol (protocol 1) on the left and recovery tests with ischemia (protocol 2) and without ischemia (protocol 3) on the right. MVC = maximal voluntary contraction, MVC (double twitch) = MVC with superimposed double twitch stimulation of the motor nerve, M_{max} = Maximum M-wave, H_{max} = maximum H-reflex, low- and high-frequency stimulation = electrical stimulation of the passive muscle at 20 Hz and 80 Hz frequencies, respectively.

The post-fatigue measurements repeated the same protocol, except that the immediate ones were performed with ischemia and on the right leg only. Ischemia was induced by a blood pressure cuff which was wrapped around the middle portion of the right thigh and inflated to at least 200 mmHg. This pressure was, as before, maintained while the immediate post-fatigue measurements were performed. This procedure served to retain the possible metabolic fatigue substances in the fatigued muscles throughout these measurements. Blood samples were drawn from the ulnar vein and fingertip before, 5 minutes after and one day after the fatigue task. Throughout the experimental procedure, the legs were warmed with an infra-red lamp, and the temperature of the skin was monitored during every test unit. Thus, it was possible to maintain a constant skin temperature by adjusting the distance of the lamp according to the existing skin temperature (30 ±0.5°C).

The stretch reflexes were measured as a ten consecutive stretches for the right leg at the very beginning, after 30 minutes and at the very end of the fatigue stimulation. During the stretch reflex tests the ES was turned off and stretch reflexes were induced by mechanical stretching only. Recovery was tested 15 minutes (15min+) and 30 minutes (30min+) after the end of the fatigue task.

In the two control tests (protocols 2 and 3) fatigue stimulation was preceded by only the measurement of the maximal H-reflex (five measurements). Therefore, the only post-fatigue measurement was that of the recovery of the maximal H-reflex peak-to-peak amplitude, which was measured at a stimulus frequency of 0.1 Hz. In the first control test (protocol 2) the first five minutes of recovery was measured with ischemia.

4.2.5 Experiment 5.

In experiment five, the marathon run was used for the second time as a fatigue-inducing model, largely because it both contains repetitive (10 000) SSC muscle actions and its long recovery period is very suitable for a follow-up study. The before-marathon tests took place one hour before (Before) the actual marathon and the after-marathon follow-up tests were performed immediately (After), two hours (2Hafter), two days (2Dafter), four days (4Dafter) and six days (6Dafter) after the run. The marathon was run individually and the target times were set according to each participant's best times to confirm high levels of motivation, performance and consequently fatigue. The race was controlled by a cyclist who kept the predetermined speed as constant as possible.

The testing protocol (figure 6) included measurements of isometric maximal voluntary contractions (MVC), maximal M-waves and H-reflexes and stretch reflexes. In all the experimental conditions measurements were carried out using an ankle ergometer (see experiment 3) and both legs were measured separately. The skin temperature of the legs was measured during every test unit. At the end of the marathon a blood pressure cuff, which was wrapped around the middle portion of the right thigh of the subject, was inflated to at least 200 mmHg. This pressure was maintained until the immediate after-marathon measurements were completed. This procedure served to retain the

possible metabolic fatigue substances in the fatigued muscles. Blood samples were drawn before each testing unit from the ulnar vein and 5 minutes after the marathon run from the fingertip. Heart rate was monitored throughout the marathon run (Sport Tester PE-3000, Polar, Finland).

The stretch reflexes of the plantar flexors were measured passively as a bout of ten consecutive stretches at two different stretching velocities. The amplitude of the ankle joint dorsiflexions was 10 degrees in both cases; the mean stretching velocities of the stretches were 1.9 rad·s^{-1} and 3.5 rad·s^{-1}. The frequency of all the stretches was 0.5 Hz and their order was randomized.

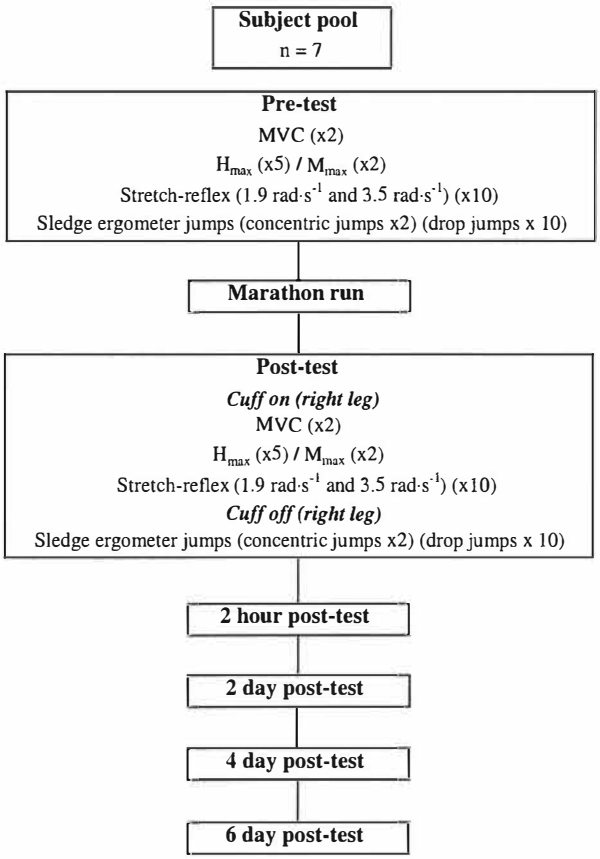

FIGURE 6 Experimental protocol.

After the ankle ergometer measurements, the subjects moved to a sledge ergometer (see experiment 1.), where they performed ten maximal drop jumps from a predetermined optimal dropping height. The optimal dropping height (best rebound height) of each subject was determined one week before the actual marathon run using the method described earlier in experiment 2.

4.3 Recording procedures and analyses

In all the experiments electromyographic (EMG) activity was recorded via bipolar surface electrodes (Beckman miniature skin electrodes) fixed at a constant inter-electrode distance of 20 mm. In the first experiment the electrodes were placed longitudinally on the muscle belly of the lateral head of gastrocnemius (GA), on the edge of the medial side of the soleus (SOL) and on the muscle belly of the vastus lateralis (VL) muscles of the right leg. In the second experiment, in addition to the SOL muscle, electrodes were also placed on the muscle belly of the vastus medialis (VM) muscle. In the third experiment the electrodes were positioned on both legs approximately 6 cm above the superior aspect of the calcaneus on the SOL and between the center of the innervation zone and distal end of the lateral head of the GA. This was also the case for the fourth and fifth experiments, with the additional exception of the muscle belly of the VL in the fifth experiment. To keep the inter-electrode impedance low (<5kΩ) the skin area was dry shaved, rubbed with sandpaper (number 400) and cleaned with alcohol. The longitudinal position of the electrodes on the muscle belly was secured by adhesive strips and also marked carefully on the skin for the later test when necessary.

It is important to ensure that the EMG responses come from the examined muscle only. Therefore, EMG cross-talk measurements, similar to those reported by Moritani et al. (1990), were performed in experiment 3. Near-maximal percutaneus stimulations (Neuropack four mini, 30-50mA, 1ms rectangular pulse wave) were delivered to evoke compound mass action potentials (M-waves) in GA. The extent of cross-talk was determined by the relative amplitudes of the M-wave recorded from the SOL. In these recordings the mean peak-to-peak M-wave amplitude was 9.20 (SD 3.61) mV for the GA and 0.42 (SD 0.35) mV for the SOL, resulting in a cross-talk of 4.76 (SD 4.45) %. This value was lower than the 6% reported by Moritani et al. (1990). The distance between the electrode pairs was the shortest in the GA and SOL and the electrode type was the same in every experiment. It can, therefore, be assumed that the extent of cross-talk in the present experiments in each of the muscles measured was negligible or even nonexistent (Moritani et al. 1990; Koh and Grabiner 1993).

In experiments 3 and 4, EMG activity of the SOL during 50% MVC was also recorded via 50 μm diameter Teflon insulation Evanohm wire electrodes (Wilbur B. Driver Co.). The wire electrode was inserted into the SOL muscle laterally 10 cm above the muscle-tendon region. Careful removal of a section of Teflon at the end of the wire resulted in an exposed, potential-sensitive tip of approximately 2 mm. Two parallel wires, the recording areas separated by approximately 4 mm, were fashioned into a double hook and inserted percutaneously using a needle as described by Komi and Buskirk (1970). Achieving the all-important connection between the electrode and the amplifier conductor was done with a spring-wire coil connector. These EMG signals together with all the H-reflexes were amplified (bandwidth 10-20 Hz to 1 kHz,

10kHz sampling frequency) through an evoked-potential measuring system (MEB-5304K, Nihon Kohden, Japan) and stored in a microcomputer.

In experiments 1,2, 3, and 5 all the EMG activity associated with voluntary contractions, patellar and stretch reflexes were transferred telemetrically, amplified by an FM-microvolt amplifier (Glonner Electronic GmbH, Germany) (bandwidth 3 Hz to 360 Hz, 1 kHz sampling frequency) and finally transferred through an A-D converter, which also full-wave rectified the signals, to a microcomputer. In experiment 4 all the EMG signals were amplified (bandwidth 10 Hz to 1 kHz) by an evoked potential measuring system (MEB-5304K, Nihon Kohden) and then transferred through an AD converter (1 kHz) to a microcomputer.

In experiments where jumping exercises were included (1, 2, 5) vertical reaction (sledge ergometer) or ground reaction forces were measured by strain gauge-type force plates (natural frequency > 150 Hz, designed and manufactured in our laboratory). All the isometric tests (experiments 3, 4 and 5) were performed using the ankle ergometer and the forces were measured as a torque around the rotational axes of the motor by a piezoelectric crystal transducer (Kistler). Angular movements of the ankle and knee joints were measured by electrical goniometers (designed and manufactured in our laboratory) in experiments 1 and 2. In the ankle ergometer the angular movement of the ankle joint was monitored by a linear potentiometer (experiments 3, 4 and 5). All these signals were stored simultaneously with the EMG in the microcomputer for further analyses. In the first experiment the jumps were filmed (100 frames·s^{-1}) with a 16-mm cine camera (Locam 51-0003) from a side view.

4.3.1 EMG and force during voluntary contraction

In all the jumping activities included in the experiments, full-wave rectified EMGs were phase-integrated to pre-activation, impact (eccentric) and push-off (concentric) EMG. The integrated EMG was divided by the integration time and taken to represent the average EMG (aEMG). Impact and push-off forces were calculated following the same principel. In the sledge ergometer jumps the take-off velocity was calculated from the concentric net impulse. In the DJ and LBJ the take-off velocity was calculated on the bases of the following formulae (Bosco et al. 1980):

$$h = \frac{gt^2}{8} \qquad \text{and then:} \qquad v = \frac{h^2}{2a}$$

h = vertical rise of the center of gravity
g = gravity (9.81 ms^{-2})
t = flight time
v = take-off velocity
a = acceleration

In the ankle ergometer measurements (experiments 3, 4 and 5), MVCs with corresponding force-time curves and EMGs were analyzed trial-by-trial as an average for a 500-ms window. The setting of the window was determined so that the maximal force onset occurred in the middle of this time interval. The maximal rate of force production was taken from the MVC force-time curve as the steepest rise of the curve (experiment 5). In experiment 5 the torque values were divided by the moment arm to obtain force values.

In experiments 3 and 4 the EMG activity during 50% MVC was also recorded with fine wire electrodes. This EMG was analyzed trial-by-trial for the number of times that the amplitude of the signal crossed the zero value of the signal (zero crossing rate, ZCR) and for the median frequency (MF), based on the fast Fourier transformation (FFT) (ME3000p, Mega Electronics Ltd, Finland). Individual values were calculated as an average of three overlapping windows (1024ms each). According to Lindström et al. (1973) the relationship between the ZCR and discharge rate of motor unit action potentials is linear for low and constant level contractions. Therefore, the reasoning behind the ZCR analysis was to obtain some estimation of the changes in the motor unit firing rates. However, it should be noted, that a possible increase in the synchronization of the motor unit firing rates and the reduction in conduction velocity of the muscle fiber membrane could lead to some overestimation of these changes.

4.3.2 Reflex sensitivity

In all the passive reflex measurements, background EMG activity was monitored on an oscilloscope throughout the measurements and controlled according to the criterion of absence. This precaution was important since reflex sensitivity is known to change if the muscle is not fully relaxed (Verrier 1985).

4.3.2.1 H-reflex

In recording the Hoffmann-reflex (H-reflex) in experiments 3, 4 and 5 a standard methodology (Hoffmann, 1918) was adapted (figure 7). The H-reflex was evoked in the triceps surae muscle by electrical stimulation of the Ia-afferent fibres of the tibial nerve in the popliteal fossa. With an appropriate pulse duration and stimulus intensity (Hugon 1973), only Ia-afferents are excited by the stimulus. After a delay of approximately 15 ms, this excitation reaches the motoneuron pool and elicits the excitatory postsynaptic potential (EPSP), which attains discharge threshold in a given number of motoneurones. The response of this discharge can later be seen in the EMG as an additional delay of 15-20ms. With increasing stimulation intensity an earlier response (delay 8ms) becomes visible. This response is called the M-wave (muscle compound action potential), which represents direct stimulation of the motor axons.

After preparing the skin, stimulation electrodes (pregelified AG/AGCL electrodes, Niko, Denmark) were positioned bilaterally. The cathode (1.5 x 1.5 cm) was placed over the tibial nerve in the popliteal fossa and the anode (5 x 8 cm) was placed superior to the patella. For H-reflex and M-wave testing, single

rectangular pulses of 1 ms duration (3 for M-wave and 5 for H-reflex) were delivered from an evoked potential measuring system (MEB-5304K, Nihon Kohden, Japan) to the tibial nerve. The position of the stimulus electrodes was tested first in the upright stance, then checked in the experimental position to ensure constant recording conditions. The intensity of the electrical stimulus was set in every testing unit to elicit a maximal H-response (H_{max}) and M-response (M_{max}).

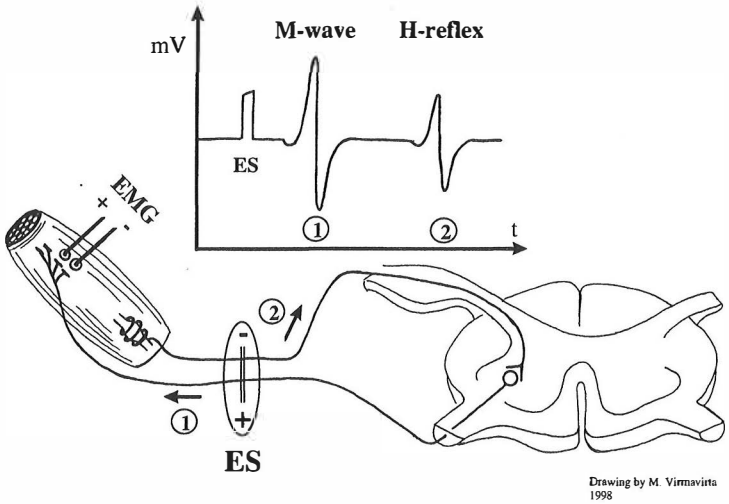

FIGURE 7 Schematic illustration of the neuronal circuitry and the methodology used to elicit the H-reflex. Arrows designate the direction of propagation of action potentials. The trace on top of the figure shows an H-reflex recording from the soleus muscle at rest. The numbers represent the corresponding propagations and EMG responses. ES = electrical stimulation.

Each complete sequence of pre-test and post-test measurements took approximately 240 s. Within this period, at least 30 s were allowed to elapse between the MVCs and H-reflex recordings to avoid the problem of post-contraction depression of the H-reflex (Scieppati and Crenna 1984). The same time delay was allowed to elapse between the M-wave and H-reflex testing. In the fourth and fifth experiments maximal H-reflex peak-to-peak amplitudes were expressed in relation to the maximal M-wave peak-to-peak amplitudes. Theoretically the H:M ratios, so determined, should not be affected by any changes in the peripheral excitability of the muscle fibres consequent on fatigue. All the latencies were also analyzed. However, the long time interval between the pre- and post-measurements could have affected the reflex sensitivity through effects of some behavioral and environmental factors. This was why the contralateral leg was used as a control leg. In the third experiment the reflex excitability was calculated according to the method used by Garland and McComas (1990). First, maximal H-reflex peak-to-peak amplitudes were expressed in relation to the maximal M-wave peak-to-peak amplitudes. Then the difference between the pre-test and post-test H/M ratios was expressed as a percentage of the corresponding pre-test value. Any percentage change in the

H/M ratios of the control leg was then subtracted from the percentage change on the experimental side so as to give overall reflex excitability. Using a control leg in this way eliminated the effects of possible behavioral and environmental factors.

Williams et al. (1992) studied the reliability of individual differences for H-reflex recordings. They found that the reliability of both the H-reflex and M-wave was extremely robust with the majority of coefficients being above 0.95. In addition, the reliabilities remained high when as few as four trials were examined.

4.3.2.2 Passive stretch reflex in ankle ergometer

In general the neuronal circuitry which is responsible for the stretch reflex response is the same than that of the H-reflex (figure 7). However, the H-reflex bypasses the muscle spindles and for the stretch reflex this is not the case. It was Lundberg and Winsbury (1960) who were able to find evidence that the receptor responsible for the classical stretch reflex (Liddel and Sherrington, 1924) is the primary ending of the muscle spindle, the activity of which, together with Ia fibres, leads to activation of the α-motoneurones of their own muscle (autogenetic excitation). The latency for the reflex response in the muscle is usually 30-35ms.

Stretch reflexes of the muscles around the ankle joint (triceps surea) were elicited by an ankle ergometer in experiments 3 – 5 (from 10 to 20 stretches in each testing unit). The description of the ergometer has been presented earlier in the account of the experimental design of experiment 3. Two different types of ankle joint dorsiflexions were selected to evoke stretch reflexes of the plantarflexors (figure 8). The amplitude of the dorsiflexion was 10° in every case. The angular velocity varied from 1.9 rad·s^{-1} (experiment 5) to 3.5 rad·s^{-1} (experiment 3, 4 and 5).

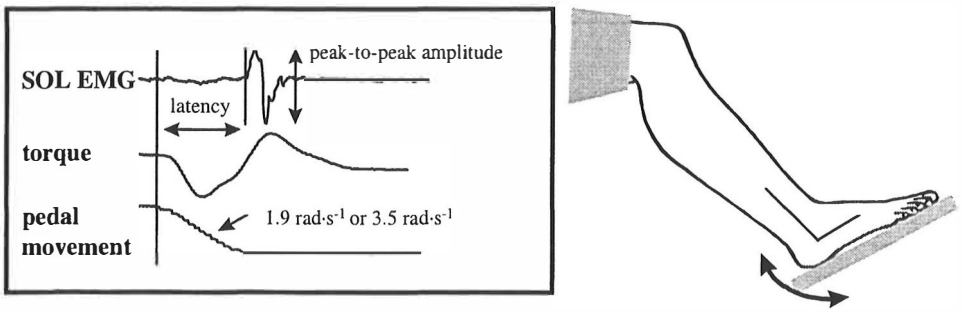

FIGURE 8 Schematic representation of the stretch-reflex stimulus and EMG responses induced by the ankle ergometer.

The stretch reflexes were measured as a bout of ten consecutive stretches. These ten stretch reflexes were first averaged and then peak-to-peak amplitudes were analyzed together with latency times, torques and angular displacements of the ankle joint. The angular displacement of the ankle ergometer pedal

served as a trigger (< 0.5°) for the averaging. In the third experiment the relative changes in the control leg were subtracted from that of the experimental leg.

4.3.2.3 Stretch reflex in sledge ergometer

The short latency stretch-reflex component (M_1) (Dietz et al. 1979) can also be analyzed from normal stretch-shortening cycle type of muscle actions. In experiments 2 and 5 this was done for the sledge ergometer jumps. First the EMG was full-wave rectified and averaged in blocks of ten consecutive jumps. The steep rise in the vertical force was used as a trigger for the averaging (<50 N). After averaging the rectified EMG signal was low-pass filtered at 75 Hz (Butterworth type 4th-order 0-lag digital filter) according to Horita et al. (1996). The M_1 reflex component was then identified according to the original definition of Lee and Tatton (1978). Usually, the first rapid EMG burst appeared with approximately 30 ms latency after ground contact. Bergui et al. (1992) reported 32.2 ms of M_1 latency and 57 ms of M2 latency in the stretch reflex of the quadriceps femoris muscle. Therefore in our analysis the M_1 was quantitated by analyzing the area under the curve from 30 ms to 50 ms after the onset of ground contact and then expressed as M_1 aEMG (figure 9).

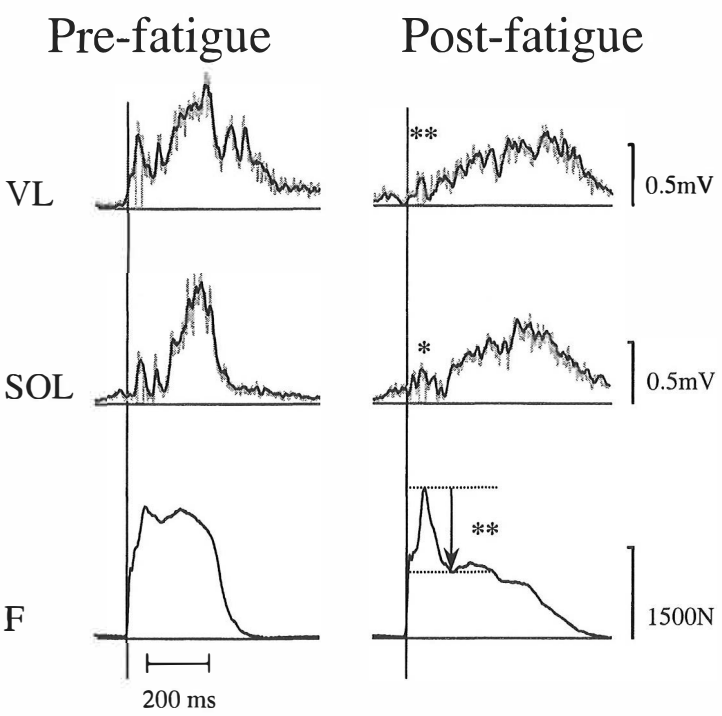

FIGURE 9 An example of the analysis of the M1 reflex component (shaded area). The arrow in the post-fatigue force-time curve indicates the peak force reduction (PFR).

Gollhofer et al. (1990) studied the reproducibility of electromyographic patterns in stretch-shortening type contractions which were repeated several times per day for nine days. In between the test days the surface electrodes were removed and each position was marked by tattooing for later re-attachment. They concluded from the variance-covariance matrix that the reliability coefficients for most of the parameters for the EMG pattern (pre-innervation phase, eccentric phase and concentric phase) was higher that 0.90. Most importantly, the reliability coefficient (day-to-day comparison) for the reflex induced activation phase for the SOL in the jumping exercises was from 0.83 to 0.96. In isometric conditions, the reproducibility of iEMG has been reported to be very high as well (r=0.88, Komi and Buskirk 1970; r=0.88 - 0.91, Viitasalo and Komi 1975; r=0.95 - 0.98, Viitasalo et al. 1980).

4.3.2.4 Patellar reflex

Patellar reflex (T-reflex, tendon jerk) measurements represents a classic form of studying the reflex sensitivity of a muscle. According to Liddell (1960) the knee jerk was introduced by Erb and Westphal as long ago as 1885. In the T-reflex, the reflex is elicited by a hammer strike on the tendon, which leads to a short and rapid stretching of the muscle. The neuronal circuitry is, therefore, the same as that of the stretch reflex (see figure 7). The problem with the T-reflex is that its reproducibility is not very high if certain mechanical features of the stimulus have not been taken into account while measuring. The amplitude and the rate of the stretch of the muscle depend on the site of the impact, angle of impact and force delivered by the impact of the reflex hammer. In addition, the compliance characteristics of the muscle might have an effect on the response.

In the second experiment patellar reflexes of the right leg were measured with the subject seated on the dynamometer in a relaxed position. The subjects were blind-folded and they wore a hearing protector. In this way any behavioral or environmental factors and anticipatory reactions could be minimized. The right leg was placed vertically with a 90° knee angle and was fixed tightly to a force transducer. Special care was taken that the leg extensor muscles were relaxed when the reflex hammer was dropped from an angle of 90° with respect to the patellar tendon. Upon striking the patellar tendon, a microswitch embedded in the hammerhead sensed the tap. In general, the patellar tendon measurements followed those reported by Häkkinen and Komi (1983).

In the analysis of patellar reflexes the three best performances, which produced the highest reflex forces, were chosen for further analysis. The latency time was determined as the time between the hammer strike and the beginning of the EMG response using the threshold of ± 10 μV. The motor time was determined as the time between the beginning of the EMG response and the initial force production (threshold of 1 N). Peak-to-peak reflex amplitudes of the EMG and force responses were analyzed and the EMG-force ratio was calculated.

4.3.3 Evaluation of muscle stiffness

In the second experiment the eccentric peak muscle stiffness values were measured indirectly and calculated as the ratio of the changes in the Achilles tendon force (ATF) and muscle length. This was done for the SOL muscle only. The assumption was that during the early eccentric phase of the sledge jumps rapid knee flexion causes a reduction in the gastrocnemius activity and, therefore, the ATF consisted only of the SOL activity. The ATF was obtained indirectly by calculations from a subject who participated in the ATF measurements of Komi et al. (1987), which were based on the in-vivo measurements from the Achilles tendon with a buckle-type of transducer. The tendon force transducer was implanted under local anesthesia (see Komi et al. 1987) in one subject (not included in the experimental or control groups) who signed a written agreement to participate as a subject. The size of the buckle was matched with that of the tendon. After careful calibration procedures the subject performed sledge jumps corresponding to those of the experimental and control groups. Taking into consideration the geometrical arrangement of the AT transducer, axis of rotation and the calibration system, the exact values of the ATF could be calculated. The ground reaction forces were measured simultaneously with the ATFs. The ratio of these forces was then used to obtain individual ATFs for the experimental and control subjects. The muscle length changes of the SOL were calculated according to the formulas of Frigo and Pedotti (1978).

This method indirectly calculating changes in the stiffness properties of the muscle in the sledge ergometer jumps was not used in the fifth experiment. However, the analysis of this experiment included a parameter called peak force reduction (PFR). This force was determined from the reaction force as the difference in force between the impact peak force and the immediate lowest force level where the peak force falls rapidly. The PFR is illustrated in figure 8. We believe that changes in this parameter indirectly reflects the changes in the active stiffness properties of the muscle.

In experiments 3, 4 and 5, changes in passive muscle stiffness was evaluated directly from the stretch reflex tests in the form of the passive stretch-resisting force of the muscle. This was possible due to the constant stretching amplitude of the muscle. This force was analyzed as the average force of the first 40 ms after the onset of the pedal movement (torque divided by the moment arm). During this 40 ms period, the stretch reflexes are triggered, but do not yet contribute to the force. Therefore, the nature of this force is purely passive. Thus, in the fourth experiment active stretch-resisting force was analyzed similarly from the active fatigue stimulation.

4.3.4 Twitch occlusion technique

In the fourth experiment, changes in the level of activation of the α-motoneuron pool due to fatigue were studied using the twitch occlusion technique. When a twitch is superimposed upon an ongoing contraction, its amplitude represents a measure of the extra force-producing capacity of the muscle. This technique has

been used to assess the maximality of the contraction (Merton 1954). Theoretically, supramaximal stimulation applied during a contraction excites all motor axons, including those not voluntarily able to be recruited or not driven at high enough frequencies to achieve fusion, and thereby adds additional force to the contraction (Candevia 1992).

The superimposed MVC was induced with a double-twitch -technique of interpolated peripheral nerve (tibial nerve) stimuli (McKenzie and Candevia 1991; Strojnik and Komi 1998). For the stimulation of this test the same electrode arrangement was used as in H-reflex testing. The stimulus intensity was set approximately 25% higher than that of the maximal M-wave to ensure maximal response in every testing condition. Special care was taken to ensure that the double twitch was applied during the maximal torque level. The degree of failure of voluntary activation of the muscle was quantitated as the ratio of the superimposed twitch response to the maximal maximal voluntary effort before the twitch. It was expressed as a percentage increment.

4.3.5 Low-high frequency torque test

A failure of excitation-contraction (EC) coupling has long been acknowledged as one of a number of possible causes of fatigue in skeletal muscle. However, EC failure is not a single entity, but can be induced in several places along the EC chain of the muscle fibre. In order to make objective measurements in regard to EC failure, methods of electrical stimulation were developed (Edwards et al. 1977; Jones et al. 1979). Fatigue which leads to loss of force at high frequencies of stimulation (80 - 100 Hz) is called high frequency fatigue (HFF) and at low frequencies of stimulation low frequency fatigue (LFF). In general, HFF indicates impaired action potential transmission along the surface membrane of the muscle fibre and LFF indicates either reduced Ca^{2+} release by the action potential or muscle fibre damage.

In the low-high frequency torque test (experiment 4), the arrangement of the stimulating electrodes was the same than that of the ES of the fatigue task. The relaxed triceps surae muscle was stimulated with two consecutive trains of impulses at a frequency of 20 Hz and 80 Hz (0.2-second duration in both). The stimulation amplitude was set to the level which was three times that of the motor threshold and it was kept the same for both frequencies. The mean value for the leveled torque (500ms window) was obtained.

4.3.6 Blood analyses

From the blood samples, serum creatine kinase activity (S-CK) (experiments 3, 4 and 5), skeletal troponin (TnI) (experiment 5) and blood lactate (B-La) (experiments 3, 4 and 5) were analyzed. S-CK was analyzed using a CK ultraviolet test kit (Boehringer Mannheim, Germany). TnI was analyzed by using two immunoenzymometric assays. The first of these determines the specific human cardiac TnI concentration in serum. The second recognizes all TnI isoforms. By excluding the cardiac TnI concentration, the skeletal TnI

concentration can then be determined (Rama et al. 1996). B-La was analyzed enzymatically using a commercial kit (Biochemica Boeringer GmbH, Germany).

4.4 Statistical methods

Means and standard deviations (SD) were calculated for all the subjects together (experiments 1 - 5) and for groups separately (experiments 2 - 5).

In experiments 1 and 2, statistical significances for different parameters and for group effect (experiment 2) were determined according to the analysis of variance (ANOVA) for repeated measures. When a significant F-ratio occurred for the main effects, the multiple comparison test (Scheffe) in experiment 1 and paired two-tailed t-test in experiment 2 were used to locate the source of the difference. In experiments 3 and 5, statistical significances for the different parameters between tests and between legs (experimental vs. control) were determined according to double multivariate analysis of variance (MANOVA). When a significant F-ratio occurred for the main effects, profile analysis was carried out by MANOVA to locate the source of the difference. In experiment 4, MANOVA was also utilized, although paired students T-test was used to locate the source of differences.

In all the experiments, correlation coefficients (Pearson) were calculated to determine the relationships between selected parameters.

5 RESULTS

The most important findings obtained from the present series of experiments are presented in the following section. For further details the original papers (I - VII) should be consulted.

5.1 Effects of different loading conditions

The angular changes of the knee and ankle joints did not show statistically significant differences between the different conditions in the LBS exercises. They mean values across all conditions were 24.2 ±1.5° and 20.9 ±1.8° for the ankle and knee joints, respectively. In the DJ jump exercises significant ($p<0.01$) differences were observed both in the knee and ankle joint displacements between all the conditions. For the knee joint the angular changes increased with increasing stretch load from 18.9 ±4.0° (DJ29) to 26.9 ±5.9° (DJ66). The corresponding values for the ankle joint were 19.8 ±5.8° (DJ29) and 25.3 ±6.0° (DJ66). This was also the case for the SlJ exercises, in which these changes increased significantly ($p<0.01$) from 36.8 ±4.8° (SlL-20) to 51.5 ±5.8° (SlL+20) in the knee joint and from 26.1 ±5.0° (SlJ-20) to 29.9 ±5.9° (SlJ+20) in the ankle joint. The results concerning the angular changes also indicates other performance differences (table 3).

The eccentric average and peak angular velocities for the ankle joint increased, respectively, by 1.43 rad·s^{-1} and 5.81 rad·s^{-1} from the DJ29 condition to the DJ66 condition. The corresponding values for the knee joint were 1.38 rad·s^{-1} and 3.23 rad·s^{-1}. In the SlJ exercise the eccentric peak angular velocity increased significantly by 1.29 rad·s^{-1} and 5.23 rad·s^{-1} from the SlJ-20 to SlJ+20 condition for the ankle and knee joints, respectively. In the LBS exercise the g condition showed significantly the highest eccentric mean and peak angular velocities as compared to the g+20 and g-20 conditions. The increments from the lowest value were 0.70 rad·s^{-1} and 1.10 rad·s^{-1} for the eccentric mean angular velocities

and 6.21 rad·s^{-1} and 3.21 rad·s^{-1} for the eccentric peak angular velocities for the ankle and knee joints, respectively.

TABLE 3 Mean values and SD of the take-off velocity, contact time and eccentric gross average force (n=9).

	Take-off velocity ($m \cdot s^{-1}$)	Contact time (ms)	Eccentric force (N)
g-20	2.78 ±0.16	288 ±55	2109 ±267
g	2.92 ±0.11**	224 ±40**	2968 ±221***
g+20	2.84 ±0.14*	248 ±56*	2634 ±283**
DJ29	3.05 ±0.15	206 ±33	3027 ±253
DJ46	3.11 ±0.15*	206 ±32	3286 ±357**
DJ66	3.14 ±0.15**	212 ±33	3659 ±343***
SlJ-20	2.39 ±0.32	408 ±62	1453 ±125
SlJ0	2.26 ±0.33	486 ±66**	1551 ±151**
SlJ+20	1.95 ±0.21**	596 ±94***	1596 ±137**

Significantly different from the lowest impact condition (g-20, DJ29 and SlJ-20) of each exercise: *p<0.05, **p<0.01, ***p<0.001

In all the experimental conditions, the preactivation of all the measured muscles started well before the impact of ground contact. For the GA muscle it started always earlier than for the SOL and VL muscles in all the jumping exercises. The preactivation times showed a linear increase with increasing stretching load for the GA and partly for the SOL muscles in the DJ and the SlJ exercises. For the VL muscle there was also a slight trend in this direction. In the LBS exercises preactivation times were longest in the g condition in all the measured muscles. Figure 10 shows the amount of preactivation EMG in the SOL and GA muscles. It could be observed that the amount of preactivation EMG closely followed the pattern of the preactivation times.

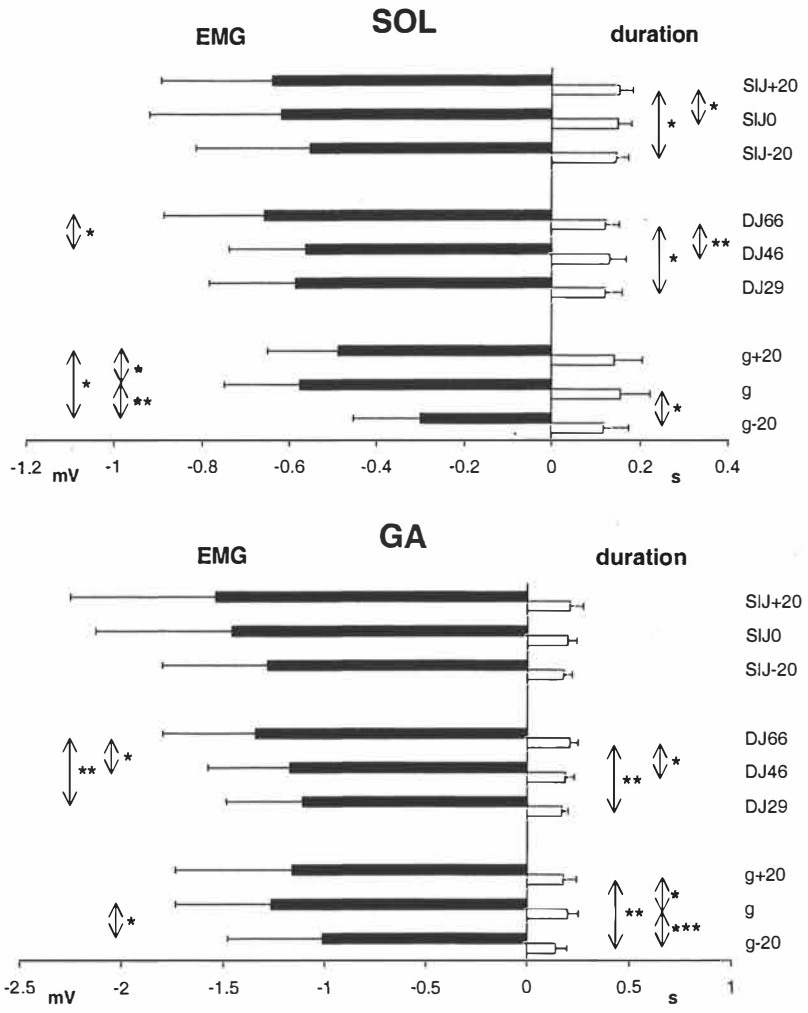

FIGURE 10 Duration (s) and average EMG activity (mV) of the preactivation phase of the SOL and GA muscles (mean +SD). *p<0.05, **p<0.01 and ***p<0.001 (n=9).

Table 4 presents the eccentric activation characteristics of the investigated muscles in the different experimental exercises. The biggest differences in the eccentric phase were be observed for the VL muscle. The increasing stretching load resulted in increased EMG activation. However, in the LBS exercises differences in the eccentric phase also appeared for the GA and SOL muscles. The highest EMG values were measured in the g condition as compared to the g-20 and g+20 conditions. For the concentric phase this analysis revealed only slight EMG amplitude differences between the different jumping conditions within the jumping exercises.

A strong relationship (r=.59 - r=.88, p<0.001) between the amount of preactivation EMG and eccentric EMG was found for the GA and SOL muscles

in most of the experimental jumping exercises. For the VL muscle a corresponding relationship was not be detected.

TABLE 4 EMG values of the eccentric phase for gastrocnemius (GA), soleus (SOL) and vastus lateralis (VL) muscles. EMG values are arbitrary units (n=9).

	GA	SOL	VL
g-20	1.26 ±0.39	1.27 ±0.40	1.31 ±0.23
g	1.46 ±0.42*	1.60 ±0.48**	1.67 ±0.28*
g+20	1.24 ±0.45	1.37 ±0.48	1.68 ±0.31*
DJ29	1.36 ±0.41	1.56 ±0.48	1.57 ±0.22
DJ46	1.41 ±0.31	1.60 ±0.51	1.73 ±0.30
DJ66	1.48 ±0.36	1.64 ±0.45	1.79 ±0.32*
SlJ-20	0.74 ±0.17	0.76 ±0.24	1.02 ±0.39
SlJ0	0.74 ±0.17	0.79 ±0.27	1.10 ±0.37*
SlJ+20	0.61 ±0.17*	0.77 ±0.22	1.26 ±0.39**

Significantly different from the lowest impact condition (g-20, DJ29 and SlJ-20) of each exercise: *$p<0.05$, **$p<0.01$.

The eccentric EMG/force ratio was sensitive to changes in the jumping condition. For the GA and SOL muscles clear decreases in the EMG/force ratio were observed with increases in the stretch load in all the jumping exercises. However, for the VL muscle these changes were more irregular. This was also the case in the concentric phase. Only in the SlJ exercise a corresponding decreasing trend was detected for the GA and SOL muscles.

5.2 Effects of fatigue

In general, the immediate neuromuscular changes due to fatigue were surprisingly similar in experiments 2 - 5. These changes are well demonstrated in figures 11 and 12, which provide an example of the measured parameters of experiments 4 and 3 with the active and passive fatigue stimulations of the calf muscles.

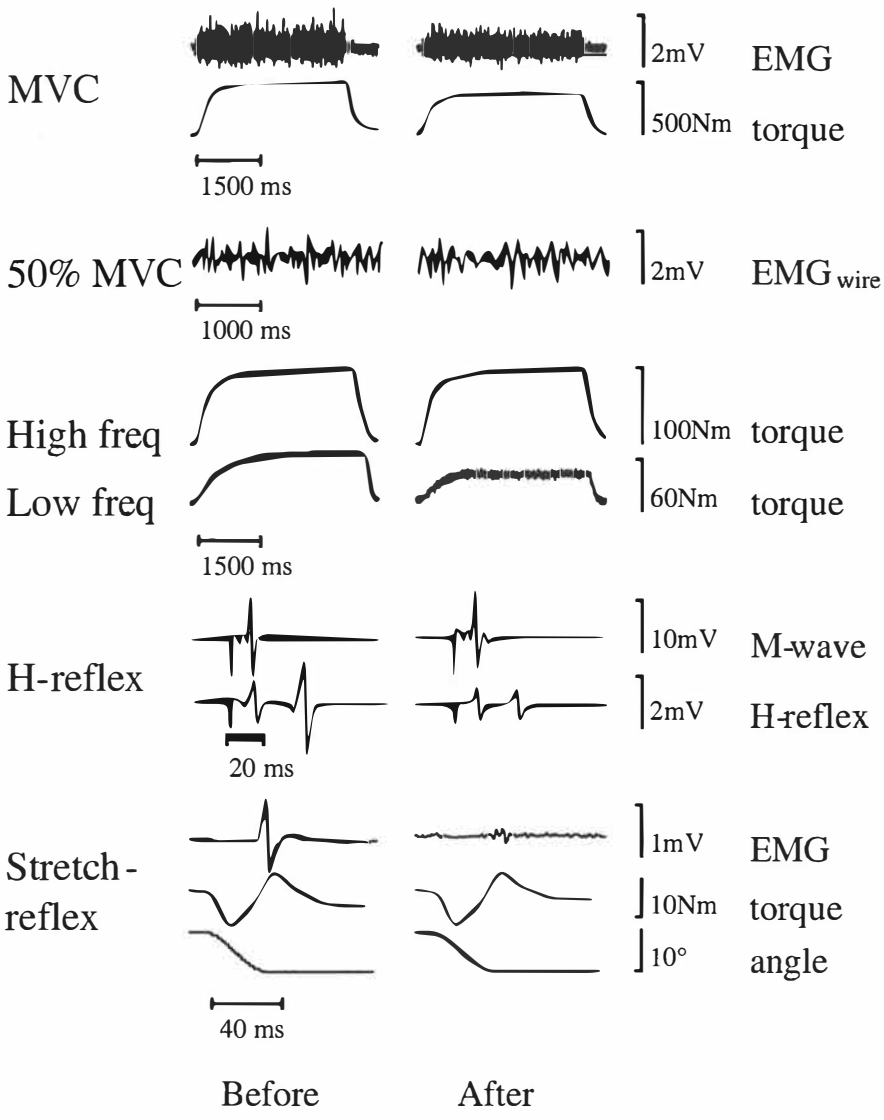

FIGURE 11 Data from one subject illustrating the effectiveness of the active fatigue stimulation. All the signals are from the experimental leg and all the EMG signals represent the activity of the SOL muscle. From top to bottom: SOL EMG and isometric plantarflexion torque from MVC, SOL EMG recorded with fine wire electrodes from 50% MVC, plantarflexion torque due to low and high frequency stimulation, maximal M-waves and H-reflexes and SOL EMG, plantarflexion torque and ankle angle displacement from the stretch-reflex recordings. The time window of the different measurements is not comparable.

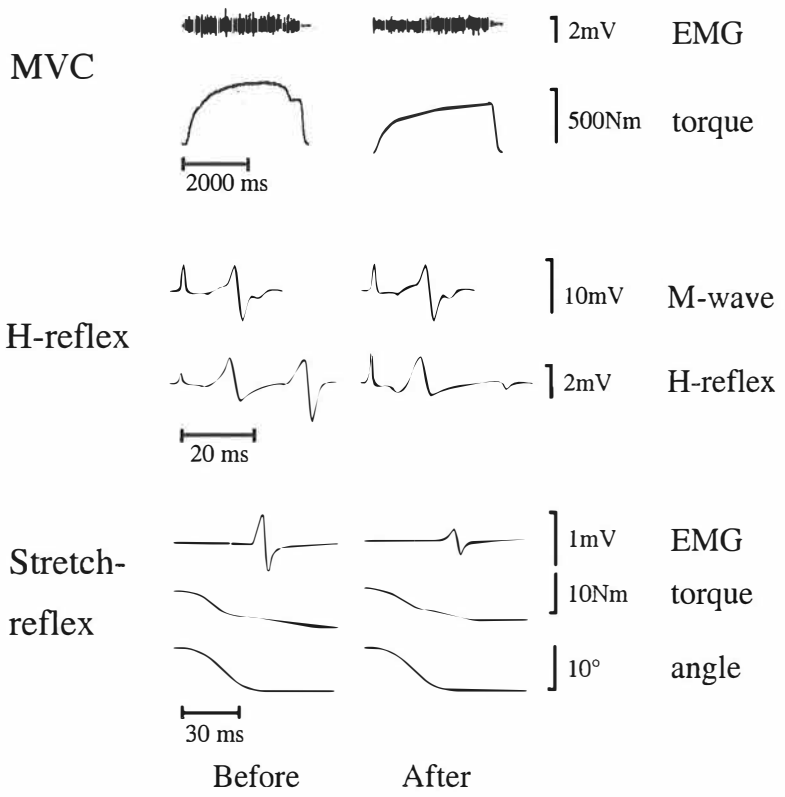

FIGURE 12 Data from one subject illustrating the effectiveness of the passive fatigue stimulation. All the signals are from the experimental leg and all the EMG signals represent the activity of the SOL muscle. From top to bottom: SOL EMG and isometric plantarflexion torque from MVC, maximal M-waves and H-reflexes and SOL EMG, plantarflexion torque and ankle angle displacement from the stretch-reflex recordings. The time window of the different measurements is not comparable.

5.2.1 Maximal voluntary contraction (MVC)

Neuromuscular fatigue was characterized by a clear reduction in most of the measured parameters (figures 11 and 12). A remarkable impairment was seen in the contractile output of the muscle. The MVC torque and/or force decreased immediately after fatigue among all the subjects, averaging 23.2 ±19.7% ($p<0.001$), 18.5 ±7.0% ($p<0.01$) and 29.8 ±10.9% after passive and active fatigue stimulation and after the second marathon run (experiments 3, 4 and 5), respectively. In addition, the maximal rate of force production was reduced by 29.5 ±27.1% ($p<0.05$) in experiment 5 (second marathon). This was also the case for the maximal neural input to the GA and SOL muscles as expressed by the relative reduction in the average EMG values of 19.9 ±29.7% ($p<0.01$) and 16.5 ±24.4% ($p<0.01$) in the passive fatigue experiment (3), 11.9 ±6.9% ($p<0.001$) and

14.8 ±11.7% ($p<0.01$) in the active fatigue experiment (4) and 28.3 ±23.1% ($p<0.01$) and 38.2 ±19.9% ($p<0.01$) in the second marathon experiment (5), respectively. All of these reductions resulted in non-significant changes in the EMG/force-ratio.

The recovery of the MVC parameters differed in different experiments. After passive fatigue stimulation, total recovery was reached in 15 minutes. After active fatigue stimulation, 30 minutes was not sufficient to induce full recovery. In the second marathon experiment (5), maximal isometric force and the simultaneously recorded EMGs of the SOL and GA muscles behaved very similarly during the fatigue follow-up. The 2Hafter values showed a significant recovery ($p<0.05$), which continued until full recovery in these parameters was reached by 2Dafter. However, the maximal rate of force production was still significantly low ($p<0.05$) at 2Dafter.

The decreased amplitude of the maximal EMG was accompanied by a reduction in the frequency properties of the 50% MVC EMG signal in the passive and active stimulation experiments (3 and 4). This was demonstrated by a 12.2 ±11.4% ($p<0.01$) and 8.0 ±7.4% ($p<0.01$) decrement in the ZCR value immediately after the fatigue, respectively. In the passive fatigue experiment (3), it is possible that a reduction occurred in the unit firing rates. Thus, the possibility of increased synchronization of the motor unit firing, which could also be seen as a reduction in the ZCR, seemed to be a less plausible explanation, since there was no increase in the spectral components in the low-frequency range (Bigland-Ritchie et al. 1981) as shown by a reduction in MF of only 1.1 ±1.8% (n.s.) immediately after RPS. According to Hägg (1992) decrease in the firing frequency has correspondingly little effect on the MF. However, in the active fatigue stimulation (experiment 4), there was also a significant reduction in the MF (6.8 ±5.7%, $p<0.01$). The recovery of these rates behaved similarly to those of the MVC parameters.

In the pre-fatigue measurements of the active fatigue stimulation (experiment 4), the MVC was affected by only a 0.54 ±1.56% increment due to twitch interpolation with twin stimuli. This difference increased slightly immediately after fatigue to 4.28 ±3.07% ($p<0.05$). The recovery during the follow-up period was again incomplete with relative values of 1.59 ±1.06% to 1.50 ±1.86% for the 15 and 30 minutes post-fatigue, respectively.

5.2.2 Sledge ergometer jumps

The results of the parameters, which are related to the performance capability of the SSC type of sledge jumps, are summarized in table 4. It shows a clear deterioration of the muscle function of the experimental subjects after the fatigue in the first marathon experiment (2). The most significant changes can be seen in the average forces and, therefore, also in the take-off velocity. The results of the knee and ankle joint displacements did not show any significant change as a consequence of fatigue.

TABLE 5 Basic performance parameters of the SSC sledge jumps for the experimental and control groups.

		Experimental group (N=9)					
		130%			70%		
		before		after	before		after
Contact time	mean	516		601	479		572
(ms)	SD	81		91	96		67
Take-off velocity	mean	2.23	*	1.96	2.09		1.89
($m \cdot s^{-1}$)	SD	0.23		0.28	0.25		0.23
Mean eccentric force	mean	1614		1386	1439	***	1184
(N)	SD	219		210	195		188
Mean concentric force	mean	1108	**	953	1121	***	958
(N)	SD	130		187	152		114

Significant difference after group effect *p<0.05, **p<0.01, ***p<0.001

In the two SSC sledge jumps the EMG activity of both measured muscles decreased (p<0.05 - p<0.01) in the after-marathon test as compared to the before-marathon one. In general, this reduction was most obvious in the pre-activation phase (30.4-38.4% and 26.6-29.6% for the VM and SOL muscles, respectively) and was almost as clear in the eccentric phase (the respective values being 19.5-26.9% and 17.3-31.8%). The concentric phase did not show any significant reduction in any of the loading conditions. However, the EMG ratio of the eccentric and concentric phases decreased during the marathon significantly for the 70% (p<0.01) loading condition. This trend was consistent for both of the muscles. The EMG-force ratio did not demonstrate any significant changes.

5.2.3 Reflex sensitivity

Immediately after passive and active fatigue stimulation and the second marathon run (experiments 3 - 5), the maximal H-reflex peak-to-peak amplitude declined significantly (p<0.001). While this reduction was not associated with any decrease in the maximal M-wave, the maximal H/M ratio was reduced by 43.8 ±41.4% (p<0.01), 50.5 ±37.6% (p<0.001) and 71.2 ±27.3% (p<0.01), respectively. In the passive and active fatigue experiment (3 and 4), the H-reflex recovery seemed to be complete in 15 minutes. However, after the second marathon run (5) full recovery seems to have been reached in two days.

The recovery of the reflex excitability was measured more exactly with the maximal H-reflex stimulation in the passive and active fatigue experiments (3 and 4). The recovery pattern showed two different shapes depending on the test situation (figure 13). Under normal blood supply the recovery of the H-reflex occurred within three to four minutes and then leveled off to a post-exercise level in both experiments. When the leg was kept under ischemia for the first five minutes of the recovery period in the fourth experiment, no recovery was

observed. However, the H-reflex recovered immediately to the post-exercise level when blood circulation was restored to normal.

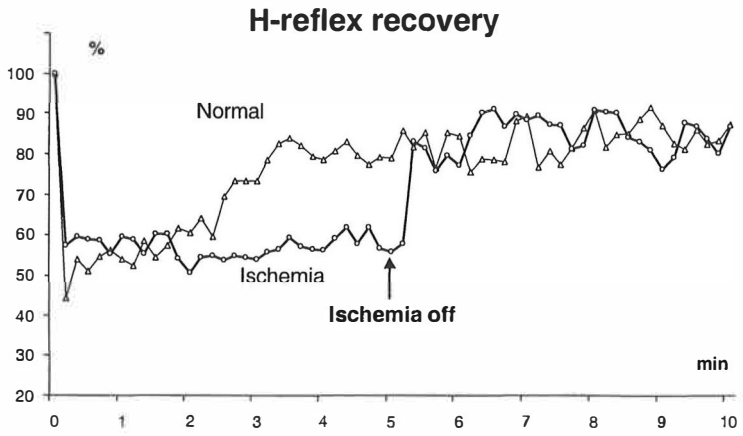

FIGURE 13 The mean recovery curve of the H-reflex peak-to-peak amplitude measured either with ischemia or with normal blood circulation. Arrow indicates the removal of the ischemia cuff (n=2·5).

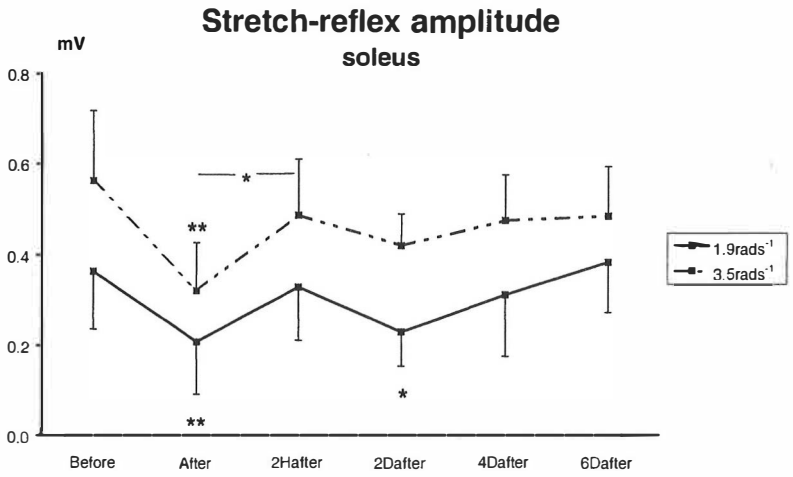

FIGURE 14 Mean values (SD) of the stretch-reflex peak-to-peak amplitude for two stretching velocities. *p<0.05 and **p<0.01 refer to the statistical significances between the values compared to the before-condition or compared to some other marked condition (n=8).

The recordings of the stretch reflexes of the SOL and GA muscles showed a dramatic peak-to-peak reduction already 15 minutes and 30 minutes after the beginning of the fatigue stimulation in the passive and active fatigue experiments (3 and 4). The respective overall reductions for the SOL muscle immediately after fatigue were 76.9 ±21.7% ($p<0.01$) and 59.6 ±28.5% ($p<0.01$). The corresponding reductions in the stretch-reflex peak-to-peak amplitude of

the SOL muscle in second marathon experiment (5) can be seen in Figure 14. In this figure the bi-phasic recovery pattern can easily be seen.

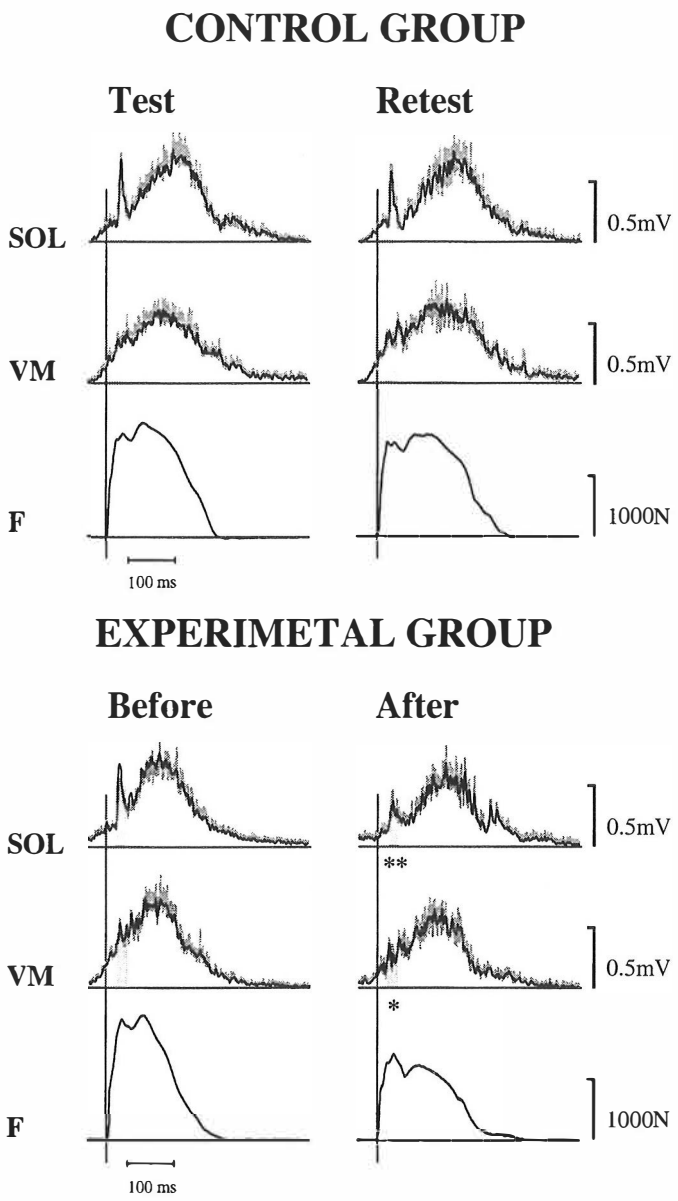

FIGURE 15 The average EMG patterns of the SOL and VM muscles and reaction force for the experimental (top) and control (bottom) groups in the sledge ergometer jumps (n=9). The gray line represents rectified raw EMG and the black line rectified 75 Hz low-pass-filtered EMG. The vertical line indicates the onset of ground contact and the shaded area the M_1 component. *$p<0.05$ and **$p<0.01$ refer to the statistical significances between the M_1 values compared to the before condition.

Figure 15, which shows the EMG patterns of the SOL and VM muscles in the first marathon experiment (2), was constructed to reveal the M_1 reflex component (shaded area) and its possible changes due to fatigue. In the SOL muscle this component was observed for the experimental subjects about 30 ms after the onset of ground contact before fatigue almost disappearing completely ($p<0.05$ - $p<0.01$) after fatigue. It should be mentioned, however, that there was also a reduction in the aEMG of the same muscles throughout the impact phase. For the control subjects a corresponding reflex component was detected in two measurements separated by one week. This was as clear as it was for the experimental subjects in the pre-fatigue measurement. In the VM muscle the M_1 reflex component was not detected with the same clarity. However, there was the same trend towards reduced reflex sensitivity ($p<0.05$) as for the SOL muscle. Again, the changes in the control group were nonsignificant. A similar reduction in the M_1 reflex component after the second marathon run (experiment 5) can be seen in figure 9. In this experiment, the recovery of the M1 component showed a similar two-phase pattern as that for the corresponding passive stretch-reflex.

The patellar reflex measurements also revealed a reduction in the stretch reflex sensitivity immediately after fatigue in the first marathon experiment (2). This reduction was 18.9 ±10.7 % ($p<0.05$) and 30.8 ±16.1% ($p<0.01$) for the peak-to-peak amplitude of the VM and for the peak force, respectively.

None of the latency times measured during the reflex tests showed any significant changes in any of the experiments.

5.2.4 Muscle stiffness

In the first marathon experiment (2), the stiffness properties of the SOL muscle were calculated indirectly for the eccentric phase. These calculations demonstrated a clear reduction in the peak stiffness values immediately after fatigue. It seems that this was more evident at higher muscle loads (130%), showing a reduction of 23.6 ±15.0% ($p<0.05$) as compared to the 18.2 ±17.8% (n.s.) reduction with the 70% load. In addition, the changes in eccentric peak muscle stiffness were related significantly to the changes in the amount of pre-activation EMG at the lower muscle loads (figure 16).

In the second marathon experiment (5), the eccentric stiffness of the muscles was evaluated from the PFR in sledge ergometer jumps. This parameter demonstrated a significant ($p<0.01$) increment immediately after fatigue, indicating impaired eccentric stiffness of the muscle. The recovery of the above-mentioned parameter demonstrated once again the bi-phasic pattern: clear recovery by 2Hafter and a second reduction by 2Dafter. Recovery seemed to be complete by 6Dafter.

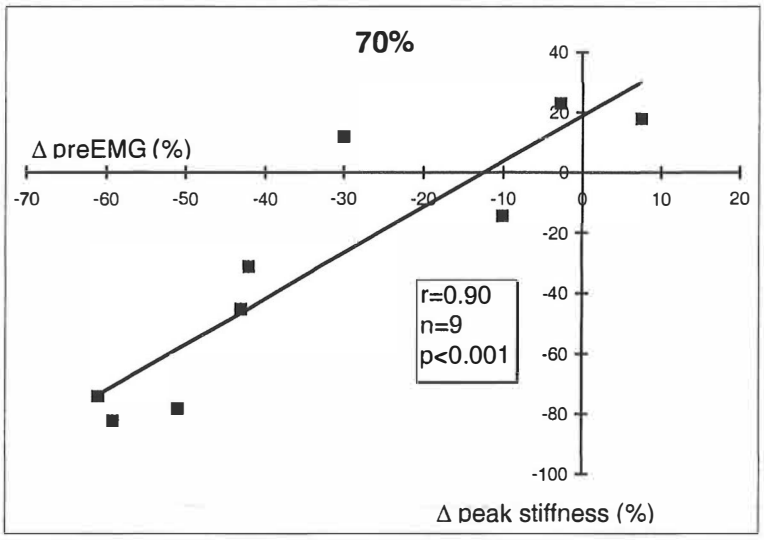

FIGURE 15 Relationship between the relative change in the preactivation EMG and the relative change in the eccentric peak stiffness of the SOL muscle for the experimental group in the 70% sledge jumping condition.

The interpretation of the passive stiffness properties of the muscle was based on the analyses described earlier with reference to the methods in experiments 3 - 5. Table 6 shows the relative contribution of the passive stretch-resisting force in the different test conditions. The pre-fatigue values have been taken as 100 % in each experiment. The active stretch-resisting force showed a remarkable reduction (34.9 ±12.4 %, $p<0.001$) already 30 minutes after the beginning of the active fatigue stimulation (experiment 4). Only a slight recovery was seen during the 30 minutes follow-up.

TABLE 6 The relative contribution of the passive stretch-resisting force in different test conditions. Exp = number of experiment and rad·s^{-1} = velocity of the pedal movement during the stretch-reflex test. *p<0.05, **p<0.01 and ***p<0.001 refer to the statistical significances between the values compared to the before-condition.

	rad·s^{-1}	Before	After	15min	30min		
Exp 3	3.5	100	84.1***	94.2*	98.1		
			±10.1	±9.5	±12.0		
Exp 4	3.5	100	85.9**	103.3	105.4		
			±9.9	±21.8	±21.1		
		Before	After	2Hafter	2Dafter	4Dafter	6Dafter
Exp 5	3.5	100	91.3**	97.7	105.1	115.2	109.9
			±6.3	±4.7	±14.9	±21.1	±13.4
	1.9	100	87.6**	96.9	104.5	112.6	111.1
			±7.6	±7.4	±25.7	±37.5	±17.7

Experiment 3 (passive fatigue) demonstrated a high correlation coefficient between the stretch-resisting force and the maximal H/M ratio ($r=0.70$, $p<0.01$). In addition, immediately after fatigue in experiment 5 (second marathon), the relative change in the stretch-resisting force correlated negatively with the relative change in S-CK. This correlation had turned positive by 2Dafter (figure 17).

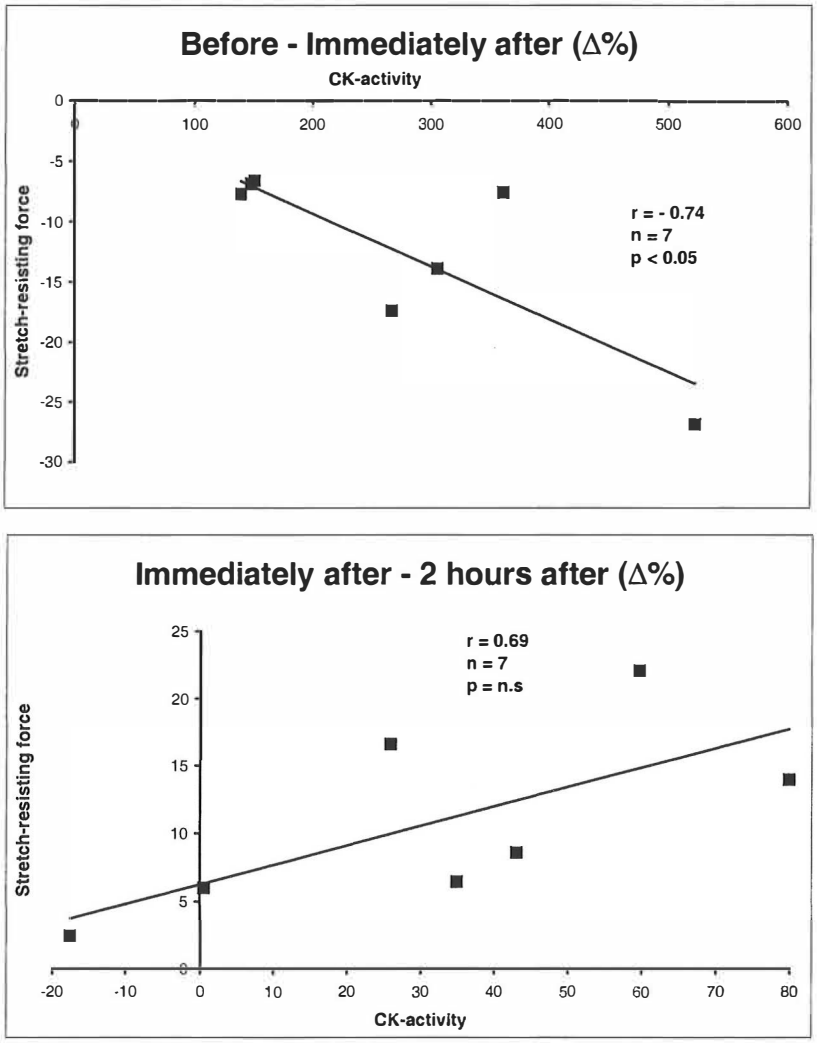

FIGURE 17 Correlation between changes in the stretch-resisting force and S-CK immediately after and 2 hours after fatigue. Stretching velocity is 1.9 rads^{-1}.

5.2.5 Low and high frequency characteristics

In the low-high frequency stimulation test in the active fatigue experiment (4), only the low frequency torque was significantly reduced (from 62 ±26 Nm to 55 ±28 Nm ($p<0.01$). This reduction was even more pronounced in the 15-minute follow-up test (figure 18).

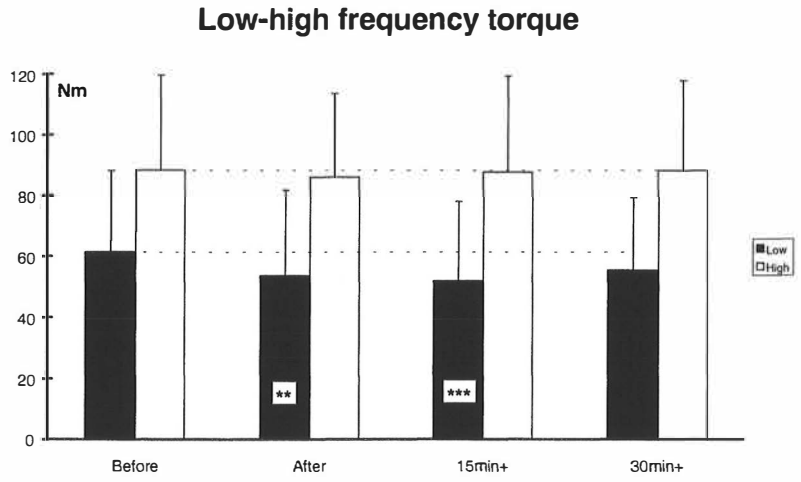

FIGURE 18 The changes in the torques induced by the low and high frequency stimulation (mean and SD). **p<0.01 and ***p<0.001 refer to the statistical significances between the values compared to the before-condition.

5.2.6 Blood lactate, serum creatine kinese activity and skeletal troponin I

In order to evaluate the possible metabolic loading and muscle damage induced by the fatigue tasks in experiments 3 -5, table 7 was constructed. In general, there was a slight but significant increment in the LA values only after active fatigue stimulation (experiment 4). This experiment (4), as well as the second marathon experiment (5), also showed clear increases in S-CK, peaking at 2Dafter in experiment 5. This was also true for TnI, which reached its peak value earlier (2Hafter) as compared to S-CK.

TABLE 7 Changes in blood lactate (LA), serum creatine kinese activity (S-CK) and skeletal troponin I (TnI). Exp = number of experiment. *p<0.05, **p<0.01 and ***p<0.001 refer to the statistical significancies between the values compared to the before condition.

		Before	After	1Dafter			
Exp 3	LA	1.84	2.12				
	($mmol \cdot l^{-1}$)	±0.3	±0.6				
	S-CK	262	284				
	($U \cdot l^{-1}$)	±164	±172				
Exp 4	LA	1.44	2.41***				
	($mmol \cdot l^{-1}$)	±0.33	±0.58				
	S-CK	167	244***	358***			
	($U \cdot l^{-1}$)	±81	±81	±124			
		Before	After	2Hafter	2Dafter	4Dafter	6Dafter
Exp 5	LA	2.23	2.66		1.97	1.57**	1.69*
	($mmol \cdot l^{-1}$)	±0.31	±0.61		±0.43	±0.25	±0.29
	S-CK	251	848***	1147**	1458***	718**	440*
	($U \cdot l^{-1}$)	±115	±245	±520	±551	±364	±148
	TnI	2.2	138.3	164.3	36.2	20.9	9.4
	($ngml^{-1}$)	±3.5	±55.2	±74.8	±19.5	±19.7	±7.5

5.3 Effects of ischemia

The purpose of the induced ischemia at the immediate end of the fatigue tasks in the passive and active fatigue experiments (3 and 4) and in the second marathon experiment (5) was not to induce a nerve block, but rather to prevent any premature metabolic recovery. The ischemia was maintained until the end of the measurements conducted immediately after fatigue. On the average this lasted about 3 minutes. The impulse conduction block progresses according to fibre size, with large myelinated afferents being affected first (McKenzie et al. 1975). Therefore, there is a possibility that the 3-minute ischemia itself had already affected the Ia-afferent activity and reduced the reflex excitability. In addition, the cuff pressure could have induced pain leading to inhibition of the α-motoneuron activity. This was the reasoning behind the test of pure ischemia in the passive fatigue experiment (3).

The results showed clearly that none of the mean values of the measured parameters showed any significant changes due to ischemia. Figure 19, which shows the plotted values and correlation coefficients for the selected parameters, demonstrates in all cases good reproducibility. Most importantly, the unaffected maximal H/M ratio implies that the Ia-afferent activity was not altered by the ischemia. Thus, it is proposed that the 3-minute ischemia was unimportant as a factor in the fatigue measurements.

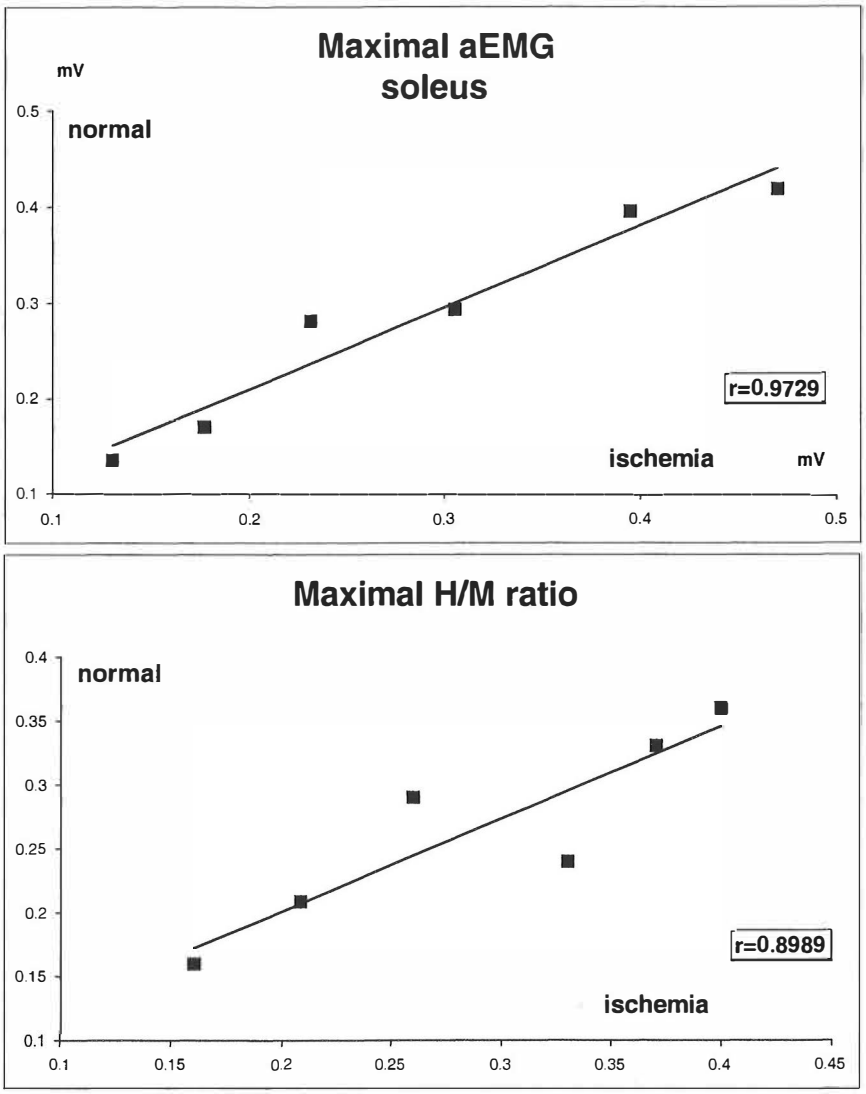

FIGURE 19 Similarity between individual parameters before (normal) and at the end of the three-minute ischemia. Maximal SOL aEMG on the top and maximal H/M ratio on the bottom.

6 DISCUSSION

The main findings of the present study were as follows:

1) In the LBJ, all the results indicated the superiority of the normal gravity condition. In the DJ the results related to the jumping performance were enhanced with increases in dropping height. For the SlJ this was more complicated. In all the different jumping conditions the electrical activity of the muscles started prior to ground contact. However, the duration of the preactivation phase depended on the stretch exercise mode. The amount of preactivity in regard to the loading condition followed the performance parameters. In general, this was also true for the aEMG of the measured muscles.

2) In every fatigue experiment, neuromuscular fatigue was characterized by a clear reduction in force output. This was seen in the sledge ergometer jumps as a decrease in average force and take-off velocity and in the isometric conditions as a reduction in MVC. This was also true for the intensity and frequency domain of the EMG of all the measured muscles.

3) Reflex sensitivity decreased in every fatigue experiment, measured either in passive (ankle ergometer) or in active conditions (sledge ergometer). In general, the amplitude changes due to fatigue were larger in the stretch reflex than the H-reflex.

4) The changes in reflex sensitivity were associated with the changes in the active and passive stiffness properties of the muscle. Interestingly, the highest reduction in passive stretch-resisting force was in the passive fatigue condition.

5) When the recovery of the reflex excitability was measured more precisely, the recovery pattern showed two different shapes depending on the test situation. Under normal blood supply conditions the recovery of the H-reflex occurred within three to four minutes and then leveled off to the

post-exercise level. When the leg was kept under ischemia for the first five minutes of the recovery period, no recovery was observed. However, the H-reflex recovered immediately to the post-exercise level when the blood was once again allowed to circulate freely.

6) When recovery was tested for several days, the recovery pattern of the maximal rate of force production, stretch-reflex peak-to-peak amplitude, M_1 aEMG and PFR showed a bi-modal trend in which the initial decline was followed by early recovery and usually a secondary decline two days after.

6.1 Muscle loading characteristics in SSC muscle action: interaction between muscle preactivity and stretch-reflex

In the present study, the reason for the variation in the parameters related to the neuromuscular function as a result of different muscle-loading conditions (experiment 1) may reflect the functional responses of the whole neuromuscular system or the direct mechanical effect of the load on the contractile part of the muscle.

It has been well documented that electrical activity prior to ground contact is the primary sensitive parameter with respect to varying load conditions (Gollhofer and Kyröläinen 1991, Dietz et al. 1981). This was also the case in the present study. Furthermore, the eccentric part of the contact showed similar mean EMG changes to those of the preactivation phase. There was a high correlation between these two phases, as well as between these phases and the eccentric peak angular velocity of the ankle joint in all the different jumping exercises. The peak angular velocity of the ankle joint can be considered to provide a rough estimation of the changes in the peak stretching velocity of the triceps surae muscle. These variables are known to be closely related to the function of the segmental stretch-reflex. This is in an agreement with the results of Gottlieb and Agarwal (1979) who found a linear relationship between the velocity of the imposed stretch and the magnitude of the spinal stretch reflex. According to Dietz et al. (1981) high stretch velocity as well as the existence of pre-activity at the moment of impact might be responsible for the appearance of the spinal stretch-reflexes. Thus, according to these interactions it can be assumed that the function of the preactivation of the triceps surae muscle is 1) to trigger an adequate segmental reflex activity for producing a corresponding stiffness to support the body and 2) to buffer high initial peak forces (Gollhofer et al. 1984). The first proposal is in line with the suggestion of Gottlieb et al. (1981) who have emphasized that the function of the preactivation is also to increase muscle spindle sensitivity through α-γ coactivation in order to potentiate stretch-reflexes.

Figure 20 was constructed to demonstrate the presence of the short latency component of the reflectory activation. As an example the averaged EMG of the

SOL muscle was drawn together with the instant of touchdown and the short latency M_1 reflex component. The M_1 component was present more than expected in all the jumping conditions and exercises. The reason for this might be that the amplitude of the phasic M_1 response is independent of the level of background EMG activity (Toft et al. 1989). In addition, in all the jumping exercises the imposed stretch seemed to be strong enough to trigger segmental reflex activity. However, there are some other arguments (Matthews 1986, Hunter and Kearney 1982) which emphasize that the automatic gain principle should increase the M1 amplitude proportionally to the background EMG and also, in this case, proportionally with the increasing stretch load.

In the present study, the occurrence of stretch reflex activity could be seen more clearly in the SOL muscle. The reported reduction in EMG activity (Greedwood and Hopkins 1976) in the two-joint muscle GA might be the reason that the M_1 component has not always been observed in this muscle. Therefore, it was remarkable to notice that the mean EMG activity of the SOL muscle increased significantly from the preparatory phase to the eccentric phase and decreased again in the concentric phase in all the jumping exercises. Thus, during the eccentric phase of contact, while the GA EMG activity is reduced, strong background activity from the SOL muscle may be essential. This is in an agreement with the study by Toft et al. (1991) who found that the SOL muscle generates about two-thirds of the maximal torque of the triceps surae muscle with the subjects in a sitting position.

The spinal stretch reflex can also contribute mechanically to the active muscle shortening during the following push-off phase (Dietz et al. 1979). Therefore, for the DJ and LBJ it seems reasonable to suppose that high preactivation level also results in high eccentric activity, leading to powerful output of the muscles in the concentric phase by the re-use of the stored elastic energy, as demonstrated by Asmussen and Bonde-Petersen (1974).

In the LBJ, in the added (g+20) as well as in the reduced (g-20) gravity conditions, the mean amount of EMG was reduced in both the preactivation and eccentric phases. These results might imply adaptation of the neuromuscular system to the natural gravity condition (g). Higher occurrence of stretch reflex activity could also be expected as a consequence of such adaptation. Dietz et al. (1989) reported a decreased muscle proprioceptive reflex mechanism on postural adjustments in underwater conditions (low gravity condition). It might be assumed that unexpected gravity conditions will reduce muscle spindle activity in the low gravity condition. This is in line with the result of a very low amount of preactivation in the g-20 condition. Thus, in the added gravity conditions the increased acceleration might lead to a delay in the central programming. However, in both gases the preparatory function of producing adequate stiffness for resisting high impact forces was weakened. This might also lead to an insufficient utilization of the elastic energy in the concentric phase (Komi 1984) and to decreased ability to jump reactively.

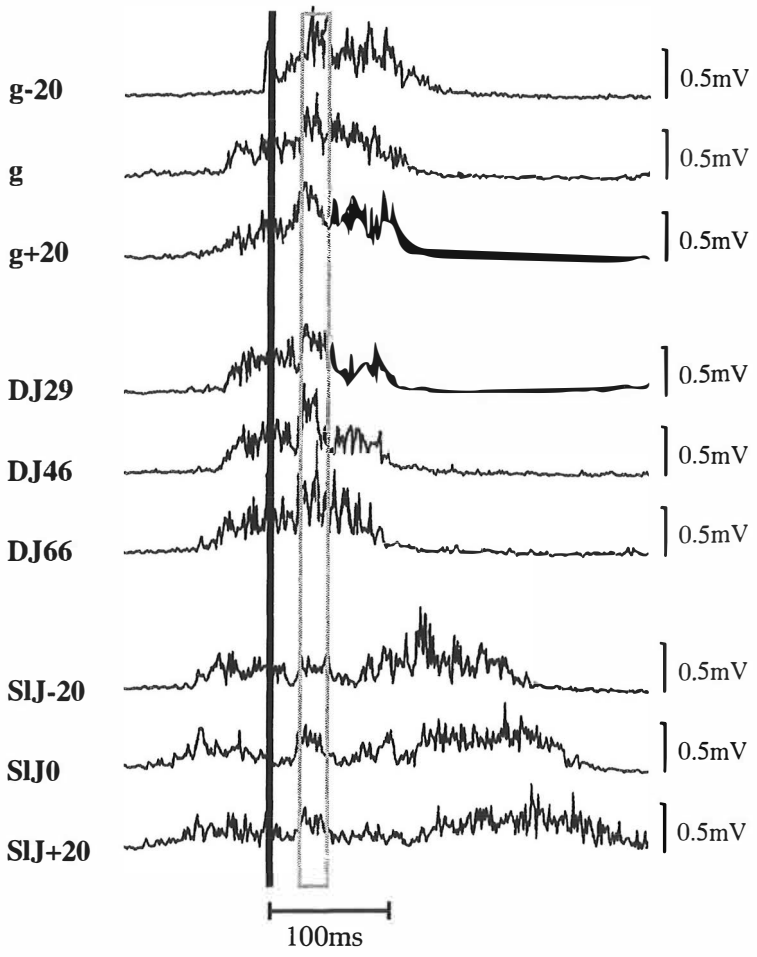

FIGURE 20 Representative example of the EMG pattern of the SOL muscle in all the jumping exercises and conditions. Black vertical line indicates the instant of touchdown and the gray window indicates the time interval for the M_1 reflex component.

As Melvill Jones and Watt (1971) have suggested, it seems that the control of landing is strongly pre-programmed from higher centres and has been, at least partly, learned through previous experience. However, adaptation of neuromuscular control might take place faster in high gravity conditions as compared to low gravity conditions. This idea is supported by the results from the eccentric iEMG-force relationship, which showed equal values in the added gravity condition in respect to the g condition and clearly increased values in the reduced gravity condition.

Schmidtbleicher and Gollhofer (1982) have suggested that muscle preactivation is regulated by the expected muscle load, which is a result of several years of learning. In the present study in all the jumping exercises the

amount of preactivation EMG of the GA and SOL muscles was strongly related to the peak stretching velocity of the muscle and, therefore, the stretch load. Komi et al. (1987) made a similar finding in running, where the preactivation was related to the running velocity. This explains the expected high muscle stretch results in the high preactivation level. However, in the LBJ both of these parameters were reduced even in the low gravity (g-20) condition, although these jumps were performed from higher dropping level. According to Schmidtbleicher and Gollhofer (1982) higher dropping height should result in higher preactivation time and EMG values. This was also the case in the DJ in the present study. Therefore, it is reasonable to assume that the control of landing is not solely regulated by the previous learning process, but is also influenced by the relatively fast adaptation of the vestibular apparatus. The effect of visual control should also not be disregarded. The involvement of the visual system in locomotion is known from animal experiments to be connected to changes in neuronal activity in the motor cortex (Hancock 1985) and possibly to have excitatory actions mediated over the lateral vestibulospinal and reticulospinal tracks, mainly on α-motoneurons.

In the SlJ the subjects could not feel the effect of the extra mass in the starting position of the jump while they were supported up in the sledge trail by an assistant. However, they had knowledge of the expected muscle stretch load. It could be seen from the precontact activation parameters that this earlier non-experienced information could modify the central programme so that the increased stretch load would result in higher preactivation levels.

According to Dietz (1992) the actual loading of proprioceptive, vestibular and visual inputs into equilibrium control is context-dependent and profoundly modifies the central programme. This is well in agreement with the results of this study, concerning also more intensive stretch shortening cycle type of movement. Thus, information about the expected muscle load must also be emphasized in these results even without earlier experience of it.

6.2 Neuromuscular adaptation to fatigue

It could be concluded from experiment 1, that those mechanisms which are related to the control of the SSC type of muscle action demonstrated considerable adaptation ability to changes in the muscle loading characteristics. Particularly evident was adaptation to normal gravity conditions. Therefore, it was of interest to test neuromuscular adaptation to some other forms of muscle modulation performed in normal gravity conditions. Muscle fatigue is one of the phenomena which impose limitations in everyday life. It was thus an obvious choice of model with which to induce modulation in neuromuscular function.

6.2.1 Central fatigue

In the passive fatigue condition (experiment 3), the fatigue protocol induced a 23.2% decrement in MVC. According to Lieber et al. (1991), 13% of the reduction in force output in a similar condition might be due to a failure in the contractile properties of the muscle or in force transfer from the muscle fibres to the tendon. This mechanism does not cover the whole loss of force observed in the present study, but leaves part of the force impairment to be explained by some other mechanisms. As our aEMG and ZCR values suggest, these other mechanisms seem to be related to decreased neural drive to the muscle. Reduced maximal aEMG values of the measured muscles in experiments two, four and five, also support this suggestion. Thus, in several other studies where fatigue has been induced by prolonged SSC exercise (Nicol et al. 1991), repeated eccentric and concentric exercises (Komi and Rusko 1974, Komi and Viitasalo 1977) and sustained isometric contraction (Bongiovanni and Hagbarth 1990) some reduction in neural input to muscle has been observed. This has been confirmed by a decline either in maximal aEMG or in the discharge frequencies of the motor units. These results make it tempting to assume that the reduced neural input to the muscle is partly responsible of the weakened force output.

Several studies have suggested that fatigue in voluntary contractions is most probably due to peripheral fatigue only (Merton 1954, Bigland-Ritchie 1984, Beelen et al. 1995). This argument is based on the fact that in these studies single shocks were not able to induce any superimposed twitches during sustained MVC. The problem with the single twitch technique is that its sensitivity is not high enough to reveal whether more than 95% of the optimal force has been achieved voluntarily (Gandevia et al. 1995). Therefore, a superimposed double twitch technique (McKenzie and Gandevia 1991) was used in experiment four to increase the sensitivity of the method. Our results demonstrated that in our well-trained and motivated subjects torque production increased with interpolated double stimulation by 4.28% ($p<0.05$) immediately after fatigue. Interestingly, this was achieved in conditions where supraspinal effects were minimized during the fatigue task. This result permits the speculation that part of the decline in the force observed in present study could have been caused by the decreased neural drive to the muscle. The origin for this decrement seems to be of peripheral in nature. The possibility of central fatigue has also been proposed by other researchers in a variety of fatigue protocols (Thomas et al. 1989, McKenzie and Gandevia 1991, Lloyd et al. 1991). This is also in line with the suggestion by Bigland-Ritchie & Woods (1984) that in exercise of longer than 60 seconds duration the declining motor drive may well limit force production.

Since our results suggest that, in addition to impairment of contractile properties of the muscle, some level of central fatigue was present due to the fatigue protocol, the possible mechanisms involved must therefore be discussed. According to the literature, central fatigue can be divided into three different mechanisms: A) supraspinal fatigue (Brasil-neto et al. 1994), B) disfacilitation of the α-motoneuron pool (Bongiovanni and Hagbarth 1990) and/or C) peripheral inhibition (Garland 1991). The present experiment did not

include any direct methods for quantifying supraspinal fatigue. The difficulty of accomplishing this task was one of the reasons why the muscle stimulation and perturbation during experiments three and four were induced externally. Hence, we feel that the effect of supraspinal fatigue was negligible in those experiments and our attention shall focus on the two latter hypotheses.

These hypotheses are based on the original suggestion by Asmussen and Mazin (1978), later supported by Bigland-Ritchie et al. (1986), that the decline in motor unit activation during fatigue depends on some reflex response from the contracting muscle itself, resulting in decreased α-motoneuron pool excitability. More direct evidence for reduced reflex sensitivity due to fatigue has since been presented by Nicol et al. (1996) and Horita et al. (1996). The decreased reflex sensitivity in the present study is clearly in line with these suggestions and findings and, therefore, we believe that some forms of reflex mechanisms are responsible for the observed changes in the neural drive.

6.2.1.1 Disfacilitation

In several studies of sustained MVC, muscle fatigue has been associated with a decreased inflow of autogenetic excitatory impulses mediated to the α-motoneurones via the γ-loop (Bongiovanni and Hagbarth 1990, Macefield et al. 1991), a phenomenon which also results in reduced reflex sensitivity. However, the exact mechanism inducing the reduced Ia-afferent activity has not yet been thoroughly explained. Two major possibilities have been presented: 1) withdrawal of the fusimotor support to the muscle spindles, and/or 2) intrafusal fibre fatigue itself (Hagbarth et al. 1986, Bongiovanni and Hagbarth 1990). Bongiovanni and Hagbarth (1990) induced a reduction in MVC motor unit firing rates by a partial anaesthetic block of the deep peroneal nerve, which they were able to counteract by muscle vibration. This could be taken as evidence of the reduced fusimotor role. However, in active muscle fatigue it seems very difficult to separate the pure function of the fusimotor system from that of the passively stimulated muscle spindles. Therefore, in experiment three, we tried to isolate the pure effect of the muscle spindles by inducing muscle fatigue passively. This was based on the presumption that when the intrafusal fibres are stimulated only by external stretching force, Ia-afferent activity is induced without assistance from the fusimotor system. However, the aim was not to try to disregard the possible role of the withdrawal of fusimotor support, but rather to reveal some more direct effects on the muscle spindle itself.

The results of experiments four and five could be cited in support of the role of the γ-loop system in reducing reflex sensitivity. However, the withdrawal of fusimotor support to the muscle spindle may not be the only explanation possible, even in active muscle fatigue, since some of the reflex measurements were performed under passive muscle conditions. A more attractive explanation might be to attribute at least part of the impaired γ-activity to other more direct fatigue effects on the muscle spindles themselves. These effects could be either metabolic or mechanical in nature.

The possibility that metabolic fatigue processes occur not only in extrafusal but also in intrafusal muscle fibres have not been well demonstrated. Some signs of intrafusal fibre fatigue have been observed following prolonged stimulation of static γ-axons in cats (Decorte et al. 1984) or during the prolonged swimming of mice (Yoshimura et al. 1996). Komi and Nicol (1998) have also proposed the possibility of direct mechanical damage in the intrafusal fibers themselves, a mechanism which could also reduce muscle spindle sensitivity and should not therefore be underestimated. Such explanations would be ideal for the results of the present study, especially in experiment four where significant increases in B-LA and S-CK were observed and in experiment five where a corresponding increment was seen in S-CK and TnL. According to Sorichter et al. (1997) the increased concentration of S-CK and TnL can be taken as indirect markers of ultrastructural muscle damage.

The theory of intrafusal fatigue relies on a fatigue-induced decline in intrafusal contraction force, which reduces the afferent discharge. In experiment three, the incompleteness of the explanation regarding metabolic fatigue raises the question of whether the reduced intrafusal contraction force could be induced by mechanical factors. In a study of repeated passive stretch of the rabbit tibialis anterior muscle, Lieber et al. (1991) measured the peak force which passively resisted the muscle stretch. This force declined after 30 minutes of stretches by 19.5 %. In the present study the average stretch-resisting force was measured for the first 40 ms after the onset of the pedal movement. The behavior of the stretch-resisting force was very much similar to that of the stretch reflexes in experiment three, demonstrating a 16% reduction immediately after fatigue. These results suggest therefore that repeated passive stretching of a muscle modifies the muscle tissue so that its compliance increases. This results in an impaired external force response of the muscle to stretch and can lead to a reduced stretch response of the muscle spindle. The resulting decrement in the intrafusal force would then decrease the inflow of autogenetic excitatory impulses mediated to the α-motoneurones via the Ia-afferents. It is of interest that this mechanical modification could also increase the intrafusal fibre compliance. In such a case, passive stretching of a muscle could lead to a direct decrease in intrafusal force. Thus, in the presence of γ-motoneuron activation the contractile properties of these fibres would also have been impaired, leading to reduced intrafusal force. In both cases, the final result would be disfacilitation of the α-motoneuron pool.

In the experiment four, fusimotor support to the muscle spindles was also minimized by using external electrical stimulation. In addition, we observed a similar reduction in the passive stretch-resisting force of the muscle (14.1%) immediately after fatigue. Hence, this result allows speculation that the mechanical modification (possibly slackening) of the muscle could also take place in the active SSC type of muscle fatigue. The 8.7 to 12.4% (for stretching velocities of 3.5 and 1.9 $rads^{-1}$, respectively) reduction in the stretch-resisting force in experiment five (second marathon) could also verify this speculation. However, it is interesting that the decrement in the stretch-resisting force was smaller after active compared to passive fatigue conditions. This could possibly be explained by the fact that during the stretch of an active muscle the strain

also rests on the cross-bridges and not only on passive components of the muscle. In addition, during long-term SSC fatigue some muscle swelling might already take place due to some damage processes as proven by the immediate increases in S-CK and TnI. This swelling could compensate for part of the reduction in stretch-resisting force.

The stretch-resisting force and stretch-reflexes were measured here under passive conditions. Consequently, it could be argued that the mechanical modification hypothesis does not necessary hold in active muscle function. However, the stretch-reflexes were also measured during sledge ergometer jumps in experiments two and five (both marathon experiments). These results showed that the M_1 reflex component declined significantly in the measured muscles immediately after fatigue. This was also the case for eccentric peak muscle stiffness and PFR, which may indirectly reflect changes in the active stiffness properties of the muscle. It seems apparent, therefore, that the theory regarding the mechanical modification of the muscle tissue and/or muscle spindle may also have relevance to active muscle function.

It should also be taken into account that the mechanical modification of the muscle might have impaired the direct force transfer from the muscle fibers to the tendon. This was also suggested by Lieber et al. (1991), who were able to induce a 13% reduction in the MVC by stretching passively the tibialis anterior muscle of the rabbit. In their study the maximal tension was induced by electrical stimulation (100 Hz) of the isolated peroneal nerve. They proposed, therefore, that the origin of the force deficit could be impaired force transfer from the muscle fibres to the tendon. In more detail, they implied that repeated passive stretching could damage portions of the myotendinous junction, a location which has been shown to be susceptible to acute injury because of stress concentration at the ends of the tapered muscle fibres (Garret et al. 1987). However, they did not observe any abnormalities within the muscle fibres. In any case, the reduced active stretch-resisting force (34.9 %) in experiment four supports this theory. Part of this large decrement in the active resisting force can be explained by events related to the low frequency fatigue (18.0 %) (discussed later). However, the remaining force deficit seems to fit within the range of the reduced passive component.

It would be of interest to discuss the exact origin of this possible modification in passive muscle tissue. Unfortunately, our results only permit indirect speculations. However, it is most likely that the strain is directed to several elements in the muscle tissue, the total effect depending on the compliance characteristics of the element. Edman and Tsuchiya (1996) studied the strain on passive elements during force enhancement by stretch in frog muscle fibres. They suggested that the origin of the elastic elements affected by the stretch is in the longitudinal filaments which link together the Z- and M-lines. These filaments have been termed titin (also known as connectin) (LaSalle et al. 1983) and nebulin (Wang and Wright 1988). The role of titin is of interest, especially as Granzier et al. (1997) has suggested that the elastic properties of titin greatly contribute to the passive force of the muscle. In addition, Horowits and Podolsky (1987) proposed that titin is responsible for maintaining the central location of the myosin filaments inside a sarcomere in relaxed muscle.

Therefore, modification of titin could result in some irregularities of filament overlapping. This could lead to increased compliance of the sarcomere and also to a decrease in the number of attached cross-bridges. If this also happens in intrafusal fibres, the direct effect will be reduced intrafusal contraction force. However, the logical result of the modification of titin is a reduced external force response to stretch and, therefore, a decrease in the mechanical effect on the muscle spindles.

6.2.1.2 Presynaptic inhibition

The hypothesis concerning peripheral inhibition relies on metabolically induced activity of the small myelinated and unmyelinated afferents (group III and IV) (Bigland-Ritchie et al. 1986, Duchateau and Hainaut 1993). Bigland-Ritchie et al. (1986) demonstrated a decline in motoneurone discharge rates with sustained maximal voluntary contraction. Almost full recovery took place 3 minutes after the exercise under normal blood supply conditions. However, they did not find any recovery in the firing rates when the fatigued muscle was kept ischemic. Therefore, they hypothesized that during fatigue the motoneurone firing rates may be regulated by a peripheral reflex originating in the fatigued muscle. Furthermore, they suggested that the muscle afferent for such a reflex could be group III and IV free nerve endings. This suggestion has since been supported by Garland (1991). These small myelinated and unmyelinated muscle afferents are polymodal and known to be sensitive to several parameters associated with either metabolic fatigue or muscle damage (Kniffki et al. 1978, Rotto and Kaufman 1988). In case of muscle damage, biochemical substances, such as bradykin and prostaglandins, are known to be released, and these stimulate the spontaneous discharge of both group III and IV mechanoreceptors and nociceptors (Kniffki et al. 1978, Rotto and Kaufman 1988, Yoshimura et al. 1996). It is known that these muscle afferents have a powerful input to inhibitory interneurones (Cleland et al. 1982) stimulation of which could lead to presynaptic inhibition of the Ia terminals and/or inhibition of interneurones in the oligosynaptic pathways (Duchateau and Hainaut 1993). In both cases this could result in a reduced neural drive to the muscle.

The metabolic parameters in experiment four demonstrated that the fatigue stimulation induced some level of metabolic loading (B-LA) and muscle damage (S-CK) and, therefore, that there was indeed a strong possibility for an increase in the activity of the III and IV afferents. The most convincing evidence for the peripheral inhibition hypothesis in the present experiment was the recovery pattern of the maximal H-reflex peak-to-peak amplitude. When the blood supply was not restricted, the recovery of the H-reflex took place in about three to four minutes similarly to the period reported by Bigland-Ritchie et al. (1986). However, no recovery was observed while the fatigue-induced metabolic accumulation was retained through ischemia. The same phenomenon has also been demonstrated earlier by Duchateau & Hainaut (1993) and Bigland-Ritchie et al. (1986) (see the above paragraph).

In the passive stretches condition (experiment three) it seems to be very difficult to identify the agent which could trigger the discharge of both group

III and IV muscle afferents. Our results (B-La and S-CK), as well as the literature (Lieber et al. 1991, Armstrong et al. 1993), do not support the occurrence of metabolic fatigue or muscle damage due to passive stretching.

The second marathon experiment (5) did not show any significant changes in B-LA. However, glycogen depletion, which is likely to take place during marathon running (Warhol et al. 1985), is known to reduce muscle metaboreceptor-mediated responses (Sinoway et al. 1992). Therefore, the mechanism of presynaptic inhibition remains a tenable explanation in this fatigue condition, especially since the lowered reflex sensitivity as demonstrated by the H-reflex and stretch-reflex measurements were associated with increases in the immediate CK-activity and skeletal TnI post-exercise values. However, the recovery patterns of these parameters did not follow each other, implying possible activity on the part of other additional mechanisms.

It has been shown that the small muscle afferents (group III and IV) have also an effect on gamma motoneuron discharge. Most studies have demonstrated that this effect is facilitatory in nature (Johansson et al. 1993). Therefore, it seems that this facilitation works as a mechanism which tries to compensate for the loss of γ-loop and/or α-motoneuron pool activation. However, according to our results, it seems that this mechanism is not sufficient enough to compensate for the total loss of reflex activity.

6.2.2 Secondary reduction in maximal rate of force production, stretch-reflex and PFR

If it can be agreed that the increased compliance of the muscle acts, at least partly, as an operative mechanism in the reduction of immediate post-exercise reflex sensitivity, then it could be suggested that the opposite direction of the correlation between the changes in the stretch-resisting force and CK-activity immediately after and two days after the marathon run (experiment 5) implies the involvement of other forms of mechanisms during the recovery phase. The recovery pattern of the maximal rate of force production, stretch-reflex peak-to-peak amplitude, M1 aEMG and PFR showed a bi-modal trend which involves an initial decline followed by early recovery and a secondary decline. This same trend has been reported earlier by Faulkner et al. (1993) in animal studies and MacIntyre et al. (1996), Nicol et al. (1996) and Horita et al. (1997) in human studies. Faulkner et al. (1993) suggested that the bi-modal recovery pattern could be due to muscle damage, which can be divided into initial injury and secondary injury. They implied that the initial injury is more mechanical in nature and the secondary injury is related to the phagocytic activity after the acute inflammatory response at the site of the initial damage. The delayed increase in the stretch-resisting force, which could be due to muscle swelling caused by various inflammation processes, together with the delayed increase in CK-activity, might support the existence of secondary injury in the present study. Hence, in addition to the impaired contractile properties of the muscle, such secondary damage could also activate the group III and IV free nerve endings and result in presynaptic inhibition at the site of the Ia afferent terminal (Nicol et al. 1996). The reduced stretch-reflex peak-to-peak amplitude two days

after the marathon run supports this suggestion. However, the maximal isometric force, corresponding maximal aEMG and H/M ratio did not demonstrate the bimodal pattern. This could imply that the inhibitory agent is operative during active or passive lengthening or shortening action of the muscle. If this is the case, the most likely possibility is that the group III and IV mechanoreceptors are more heavily involved than has been earlier thought.

6.2.3 Other sources of fatigue mechanisms

The neuromuscular block has been assumed to be one of the causes of fatigue during voluntary contractions (Krnjevic and Miledi 1958). This phenomenon has been associated with the effectiveness of electrical propagation across the neuromuscular junction and along the muscle surface membrane (Bigland-Ritchie and Woods 1984). However, decline in the mass action potential (M-wave) and force loss during high frequency stimulation, which have been connected with the above mechanisms (Edwards et al. 1977), were not observed after any of the present fatigue protocols. The reason for the force impairment must therefore be due to some failure in the contractile properties of the muscle. This possibility is discussed only briefly in respect to low frequency fatigue.

Active fatigue experiment (4) demonstrated a significant reduction (18.0%) in the low frequency stimulation torque. This decline has been defined as low frequency fatigue, which is known to be pronounced during fatigue induced by eccentric muscle action (Newham et al. 1983). Low frequency fatigue is thought to signify an impairment in excitation-contraction coupling, a process linking the action potential in the surface membrane with the activation of actomyosin by calcium (formation of cross-bridges) (Warren et al. 1993). This type of fatigue has been found to exist even after the muscle metabolites have recovered (Edwards et al. 1977), and it has also been described as long-lasting fatigue (Jones 1996). It has therefore been suggested that low frequency fatigue is not simply a consequence of an impaired excitation-contraction coupling mechanism, but is also due to muscle damage caused by the exercise (Jones 1996). In the present fatigue experiments this was supported by a significant immediate and delayed increase in S-CK (4 and 5) and TnI (5) after fatigue, parameters which have been considered an indirect indicators of muscle damage (Newham et al. 1987, Sorichter et al. 1997).

6.2.4 The role of task dependency

An extensive number of fatigue studies have demonstrated that a clear dependency exists between the fatigue task and the mechanisms underlying the fatigue processes. In the present series of experiments a common feature in the fatigue tasks was a prolonged stretch-shortening cycle type of muscle action. However, the involvement of the supraspinal effect and the type and the intensity of the muscle activation varied. Therefore, it is of importance to discuss the effect of the different fatigue tasks in regard to the present results.

In general, it was surprising that most of the changes in the results were nearly identical despite the differences in the fatigue task. However, this does not necessarily mean that the mechanisms related to fatigue should be the same.

We have suggested that disfacilitation, which seems to be an operative mechanism in passive fatigue stimulation (experiment 3), could also be involved in active fatigue stimulation (experiment 4). This disfacilitation hypothesis was made on the assumption that the reduction in the passive stretch-resisting force, which could be seen after both fatigue experiments (3 and 4), results in reduced muscle spindle sensitivity to mechanical stretch. However, for example, MVC decreased less in experiment four than in experiment three, although experiment four showed clear marks of metabolic loading and peripheral fatigue (reduction in low-frequency torque). This discrepancy can probably be explained by some compensatory mechanism of the active muscle. This difference could also be taken as evidence for our suggestion regarding the modulation of titin during passive muscle stretching. If that hypothesis holds true, then it seems obvious that during active muscle stretching, the strain, which is due to the stretching of the muscle, is directed over a larger amount of structures because of the cross-bridge formation. Therefore, the contribution of disfacilitation due to increased compliance of the tendomuscular system decreases during active compared to the passive muscle stretching. However, this still does not exclude the possibility of intrafusal fiber fatigue (Bongiovanni and Hagbarth 1990).

The aim of the fatigue task in active fatigue stimulation (experiment 4) was that its mechanical and metabolic loadings correspond roughly to the ones of the marathon experiments (2 and 5). Marathon experiments represent a natural form of the SSC type of fatigue task which, however, also involves the possibility of supraspinal fatigue. If supraspinal fatigue is evaluated purely on the bases of the muscle's ability to produce maximal force, and if the only difference between the fatigue tasks in experiments four and five is the supraspinal effect, then a simple subtraction implies that about 10% of the reduction in the MVC after marathon running is due to supraspinal fatigue. It is difficult to estimate how close to reality we can get with this naive approach. However, it is very likely that the possibility of supraspinal fatigue increases along with the duration of the fatigue task. This is in accordance with the suggestion by Bigland-Ritchie & Woods (1984) that in exercise lasting longer than 60 seconds the declining motor drive may well limit the force production.

6.3 Functional significance of fatigue in SSC

The EMG results of the sledge ergometer jumps (marathon experiments) showed that the centrally mediated pre-activation of the muscles as well as that of the eccentric phase, which is largely controlled by the supraspinal inputs and stretch reflex modulation, were clearly affected by fatigue. In addition, the present study showed a clear relationship between the function of stretch-reflexes and tendomuscular stiffness. In several of our experiments, both these

parameters were reduced due to fatigue. Thus, these changes were associated with impaired force output in both isometric conditions and SSC conditions. It was notable that the pre-activation and eccentric EMG changes were closely related to the reduced stiffness properties of the muscle. Gollhofer et al. (1992) emphasized the role of high muscle stiffness as a prerequisite for the effective utilization of elastic energy. The study by Horita et al. (1997) pointed out the importance of the preprogrammed preactivity of the muscle. The authors reported that in the drop jump (DJ) exercises muscle pre-activity correlated with muscle stiffness during the initial impact phase of the contact ($p<0.05$) and with the short latency stretch reflex area of the VL muscle ($p<0.001$). These relationships suggest that the prelanding stiffness enhanced by the preprogrammed activity could initiate high postlanding stiffness. This could then regulate the entire movement in the DJ, as revealed by the high series-elastic component (SEC) stiffness, the short contact time and more economical work (Horita et al. 1997). Nichols and Houk (1976) proposed that the stiffness of the tendomuscular complex is not only dependent on the range of motion, but also on the efficacy of the reflex system. Therefore, it is tempting to suggest that central and peripheral fatigue can, in addition to the direct effects (discussed earlier), also impair muscle performance through modulation of the stiffness-mediated utilization of muscle elastic energy. This accords well with the results of Komi et al. (1986), who suggested that repetitive impact loads may decrease the ability of the leg extensor muscles to sustain the impact loads with the result that the muscle may lose its recoil characteristics.

In general, the results of the present study supports the suggestion by Komi and Gollhofer (1998) that the stretch-reflexes can have an important role as a partial control mechanism in force-enhancement during SSC exercise.

7 PRIMARY FINDINGS AND CONCLUSIONS

The observations of the present series of studies confirm many of the hypotheses presented in chapter 3. The findings and conclusions can be summarized as follows:

1) The preactivation level of the muscle was strongly related to the stretching velocity and eccentric activation of the muscle. These did not necessarily result from the highest expected muscle load, as was observed in the LBJ, which showed considerable adaptation of the neuromuscular system to the normal gravity (g) condition. The observed results also suggest strong interaction between the preactivation and reflexively controlled muscle stiffness as well as the subsequent utilization of the stored elastic energy.

2) It is obvious that the preactivation part of the landing is, at least partly, preprogrammed. However, the total control mechanism may also depend on proprioceptive, vestibular and visual inputs, which might modify even the earlier learned central programmes. Thus, the conscious modification of the expected muscle load, even without the earlier experience, cannot be disregarded.

3) The neuromuscular system was also strongly adapted to fatigue. All the fatigue tasks induced significant impairments in neuromuscular function. This could be seen in a clear interaction between reduced force output of the muscle and decreased neural input to the muscle. In addition, these changes were associated with reduced reflex sensitivity, measured in both active and passive conditions. This result strongly emphasizes that the mechanism responsible for the reduced neural input could be the fatigued muscle itself through certain reflex pathways.

4) A mechanism reducing reflex sensitivity could also be activated due to repeated and prolonged passive stretching of the muscle. The origin of such a system is probably the reduced activity of the large diameter afferents (Ia), resulting from the reduced sensitivity of the muscle spindles

to stretch. It is suggested that in this situation of passive stretches the decreased spindle sensitivity is mechanical in nature due to some modification (increased compliance) of the extrafusal and/or intrafusal fibres

5) Simultaneous electrical and mechanical fatigue stimulation of the calf muscles induced a clear impairment in neuromuscular function, the principal cause of which seemed to be related to low frequency fatigue. The possibility of central fatigue is also apparent, as shown by the increased enhancement in force immediately after fatigue due to the interpolated double stimuli. The results of the H-reflex recovery appear to verify that presynaptic inhibition of the Ia terminals through the activation of the group III and IV afferents is of importance. However, the possibility of reduced spindle sensitivity leading to disfacilitation of the α-motoneuron pool can not be excluded. This was also true after natural long-lasting SSC exercise.

6) In addition to the initial decline in muscle function and reflex sensitivity, natural long-term SSC exercise also induced a delayed second decline in the reflex parameters associated with the lengthening action of the muscle. This second decline may be attributable to secondary injury, which might well point to the involvement of the group III and IV mechanoreceptors.

7) Reduced stretch reflex sensitivity was also associated with decreased active muscle stiffness after natural long term SSC exercise. This strengthens the suggestion that in the early eccentric phase muscle stiffness is partially controlled by the segmental reflex potentiation. Thus, it can be suggested that during fatigue reduced muscle stiffness is at least partly responsible for weakened muscle performance, resulting in impaired utilization of elastic energy.

8) Finally, the origin of the decreased neural input to the muscle seems to be the fatigued muscle itself via two reflex pathways: disfacilitation of the α-motoneuron pool as a result of reduced Ia afferent activity and presynaptic inhibition through stimulation of group III and IV afferents. The relative contribution of these two mechanisms depends on the fatigue task; in particular, the amount of metabolic loading, the amount of muscle damage and the amount of mechanical loading.

8 YHTEENVETO

Väsymyksen vaikutuksia hermolihajärjestelmän toimintaan on tutkittu hyvin paljon sekä eläimillä että ihmisillä. Lihasväsymyksen osalta tutkimukset ovat keskittyneet perifeerisesti lihastason tapahtumiin, keskushermoston mekanismien jäädessä vähemmälle huomiolle. Suurimmassa osassa tutkimuksia väsymysmallina on käytetty isometrista lihastyötapaa, jolloin lihaksen aktiivisuus on myös usein aiheutettu ulkoisella sähköisellä stimulaatiolla. Epäluonnollisten lihastyötapojen takia näiden tutkimusten tuloksia on hyvin vaikea soveltaa ihmisen normaaliin liikkumiseen.

Tämän tutkimussarjan tarkoituksena oli selvittää ihmisen hermolihasjärjestelmän kykyä adaptoitua erilaisiin kuormitusmalleihin ja väsymykseen lihaksen normaalissa työssä, eli venymis-lyhenemissyklin (SSC) aikana. Erityisenä tavoitteena oli selvittää keskushermoston väsymismekanismeja, ja lihasssukkulassa (lihaksen liikereseptori) mahdollisesti ilmeneviä väsymysoireita, jotka osaltaan vaikuttaisivat keskushermostoon.

Tutkimussarja koostuu viidestä osatutkimuksesta, joissa koehenkilöitä oli yhteensä 54. Ensimmäisessä tutkimuksessa keskityttiin erilaisten kuormitustapojen vaikutuksiin hermolihasjärjestelmän toiminnassa. Kuormitusmalleina toimivat normaalit pudotushypyt eri pudotuskorkeuksilta, pudotushypyt kelkkaergometrissä, jossa koehenkilön massaa voitiin muuttaa sekä pudotushypyt taljaergometrissä, jossa voitiin varioida koehenkilöön kohdistuvaa kiihtyvyyttä. Tämän tutkimussarjan muissa osatutkimuksissa käytetyillä kuormitusmalleilla aiheutettiin normaalisti palautuva lihasväsymys. Maratonjuoksu toimi väsytysmallina tutkimuksissa 2. ja 5. Muissa väsytystutkimuksissa hyödynnettiin nilkkaergometriä, jolla simuloitiin vakioidussa tilanteessa lihaksen venymis-lyhenemissykliä. Tämä tehtiin joko passiivisesti (mittaus 3) tai aktiivisesti yhdessä ulkoisen sähköisen stimuloinnin kanssa (mittaus 4). Lähtökohtana nilkkaergometrin väsytysmalleissa (3 ja 4) oli eliminoida motorisen aivokuoren aiheuttama lihasaktiivisuus ja siten eliminoida mahdollisen supraspinaalisen (keskushermoston ylempi osa) väsymyksen vaikutus tuloksiin. Väsymystutkimuksissa mittaukset suoritettiin

ennen/jälkeen asetelmalla. Lisäksi toisessa maratontutkimuksessa (mittaus 5) hermolihasjärjestelmän palautumista seurattiin kuuden päivän ajan.

Tutkittaessa erilaisten kuormitustapojen vaikutusta hermolihasjärjestelmään havaittiin, että lihaksen esiaktiivisuuden määrä oli riippuvainen käytetystä kuormasta. Yleensä suurempi ennakoitu kuorma aiheutti määrältään suuremman esiaktiivisuuden. Näin ei kuitenkaan käynyt, kun varioitiin koehenkilöön kohdistuvaa kiihtyvyyttä. Tämä tulos osoitti odotetusti, että hermolihasjärjestelmä on voimakkaasti adaptoitunut normaaliin maan vetovoiman aiheuttamaan kiihtyvyyteen. Alhaisempi kiihtyvyys selkeästi heikensi lihaksen liikereseptorien (lihassukkula) toimintaa. Tällä tuloksella voi olla soveltavaa merkitystä esimerkiksi vesikuntoutuksessa. Tulokset osoittivat lisäksi, että lihaksen esiaktiivisuudella oli selvä yhteys lihaksen jäykkyysominaisuuksiin ja sitä kautta lihaksen kykyyn hyödyntää lihakseen varastoitunutta elastista energiaa. Esiaktiivisuuden kontrollimekanismeja pohdittaessa näyttäisi selvältä, että esiaktiivisuus on ainakin osittain esiohjelmoitu tahdonalaisesti. On kuitenkin mahdollista, että kokonaiskontrollia voidaan modifioida palautevasteilla liikereseptoreista, tasapainoaistista ja jopa silmästä.

Tutkimuksen kaikki väsymysmallit aiheuttivat selkeästi hermolihasjärjestelmän toimintakyvyn laskua. Yllättävää oli, että tulokset olivat hyvin samankaltaiset riippumatta väsymysmallista. Isometrinen tahdonalainen voimantuotto heikkeni eri mittauksissa 18.5 – 23.2%. Aktiivisessa nilkkaergometriväsytyksessä tahdonalaisen voimantuoton laskua voitiin kompensoida n. 4% ulkoisen sähköstimulaation avulla. Voimantuoton aleneminen oli selkeästi yhteydessä lihasten neuraalisen ohjauksen (EMG aktiivisuus) heikkenemisen kanssa (11.9 – 38.2%). Lisäksi tutkimuksessa voitiin osoittaa samanaikainen refleksiherkkyyden lasku. Passiivisessa ja aktiivisessa tilanteessa mitatut venytysrefleksit laskivat kaikissa väsytystilanteissa erittäin merkittävästi. Samanlaiset muutokset havaittiin myös sähköisesti aiheutetulle H-refleksille passiivisessa tilanteessa, joskin muutokset olivat aina hieman pienemmät kuin mekaanisesti aiheutetulle venytysrefleksille. Näiden tulosten perusteella voidaan olettaa, että tutkimussarjan kaikki väsytysmallit aiheuttivat jonkinasteista keskushermostollista väsymystä, jonka aiheuttajamekanismit näyttäisivät olevan lähtökohdaltaan perifeerisiä.

Kaikissa väsytystilanteissa muutokset venytysrefleksissä olivat yhteydessä lihaksen passiivisen vastustavan voiman laskun kanssa. Tämä viittaa epäsuorasti siihen, että käytetyt väsymysmallit modifioivat lihaksen rakennetta siten, että sen joustavuus lisääntyy. Tämä voi mahdollisesti tapahtua myös lihassukkulan sisällä oleville intrafusaalisoluille. Joka tapauksessa, tuloksena on lihassukkulan venytysvasteen heikentyminen, Ia afferenttien hermosyiden syttymisfrekvenssin lasku ja lopulta α-motoneuronialtaan disfasilitaatio. Tämän tuloksena lihaksen neuraalinen aktiivisuus vähenee.

Toinen perifeerinen mekanismi, joka voi vähentää lihaksen aktiivisuutta on lihaksessa sijaitsevien vapaiden hermopäätteiden (ryhmät III ja IV) aiheuttama Ia afferenttien hermosyyterminaalien presynaptinen inhibitio. Nämä hermopäätteet reagoivat erilaisiin metaboliatuotteisiin, joita voi vapautua kuormituksen ja lihasvaurion aiheuttamina. Aktiivisessa

nilkkaergometriväsytyksessä ja maratontutkimuksessa voitiin osoittaa selviä metaboliamuutoksia (laktaatti) ja mahdollisia lihaksen mikrovaurioita (troponiini ja kreatiinikinaasi). Lisäksi havaittiin, että aktiivisen nilkkaergometriväsytyksen jälkeen H-refleksissä ei tapahtunut palautumista sillä aikavälillä kun väsytetty lihas pidettiin iskemisessä tilassa. Palautuminen tapahtui kuitenkin välittömästi kun verenkierto normalisoitiin. Nämä tulokset vahvistavat epäsuorasti presynaptisen inhibition mahdollisuuden.

Lihasväsymyksen jälkeen hermolihasjärjestelmän toiminnan palautuminen tapahtui kaksivaiheisesti. Lihaksen toiminnan ja refleksiherkkyyden akuutin laskuvaiheen lisäksi refleksitoiminnassa havaittiin toinen lasku kahden päivän viiveellä. Tämän tutkimuksen ja kirjallisuuden perusteella voidaan olettaa toisen laskun johtuvan väsytysmallin aiheuttaman lihasvaurion tulehdusvaiheesta. Tällöin refleksitoiminnan lasku aiheutuisi vapaiden hermopäätteiden aiheuttamasta presynaptisesta inhibitiosta.

Yhteenvetona todetaan, että lihasväsymyksessä lihaksen neuraalisen aktiivisuuden vähenemisen voi aiheuttaa myös väsytetty lihas itse. Tämä voi tapahtua lähinnä reflektorisesti, joko disfasilitaatiolla ja/tai presynaptisella inhibitiolla. Näiden kahden perifeerisen mekanismin suhteellinen vaikutus keskushermostollisiin muutoksiin riippuu kuormitusmallista, erityisesti metabolisen kuormittumisen määrästä, lihasvaurion suuruudesta ja mekaanisen kuormittumisen määrästä.

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ORIGINAL PAPERS

I

Effects of different simulated gravity conditions on neuromuscular control in drop jump exercises.

by

Janne Avela, Pedro M. Santos, Heikki Kyröläinen and Paavo V. Komi

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II

Effects of differently induced stretch loads on neuromuscular control in drop jump exercises

by

Janne Avela, Pedro M. Santos and Paavo V. Komi

European Journal of Applied Physiology and Occupational Physiology
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III

Interaction between muscle stiffness and stretch reflex sensitivity after long-term stretch-shortening cycle (SSC) exercise

by

Janne Avela and Paavo V. Komi

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IV

Reduced stretch reflex sensitivity and muscle stiffness after long-lasting stretch-shortening cycle (SSC) exercise

by

Janne Avela and Paavo V. Komi

European Journal of Applied Physiology and Occupational Physiology
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V

Altered reflex sensitivity due to repeated and prolonged passive muscle stretching

by

Janne Avela, Heikki Kyröläinen and Paavo V. Komi

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VI

Neuromuscular changes after long-lasting mechanically and electrically elicited fatigue

by

Janne Avela, Heikki Kyröläinen and Paavo V. Komi

(submitted)

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VII

Reduced reflex sensitivity persists several days after long-lasting stretch-shortening cycle (SSC) exercise

by

Janne Avela, Heikki Kyröläinen, Paavo V. Komi and Daniel Rama

(submitted)

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