STUDIES IN SPORT, PHYSICAL EDUCATION AND HEALTH

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JYVÄSKYLÄ 1998

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Esitetään Jyväskylän yliopiston liikuntatieteellisen tiedekunnan suostumuksella
julkisesti tarkastettavaksi yliopiston Liikunnan salissa (L304)
toukokuun 23. päivänä 1998 kello 12.

Academic dissertation to be publicly discussed, by permission of
the Faculty of Sport and Health Sciences of the University of Jyväskylä,
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UNIVERSITY OF JYVÄSKYLÄ

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Publishing Unit, University Library of Jyväskylä

URN:ISBN:978-951-39-7921-8
ISBN 978-951-39-7921-8 (PDF)
ISSN 0356-1070

ISBN 951-39-0227-7
ISSN 0356-1070

Jyväskylä University Printing House, Jyväskylä
and ER-Paino Ky, Lievestuore 1998

ABSTRACT

Oksa, Juha
Cooling and neuromuscular performance in man.
Jyväskylä: University of Jyväskylä, 1998, 61 p.
(Studies in Sport, Physical Education and Health,
ISSN 0356-1070;53)
ISBN 951-39-0227-7
Diss.

The quality and quantity of the problems arising in neuromuscular performance during work or leisure time activities in cold should be known better. Especially, the possible dose - dependent relationship between different levels of cooling and rewarming exercise in relation to changes in neuromuscular performance have remained obscure. This thesis focuses on the effects of a standard cold exposure and different levels of cooling as well as the effect of rewarming exercise on muscular performance and co-activation of agonist - antagonist muscle pairs (reflected by EMG) during stretch-shortening cycle. During the standard cold exposure 48 male subjects (age 25±2 yr, height 176±4 cm and weight 72±2 kg) sat at 10°C for 60 min wearing shorts and jogging shoes. The exposure of same duration to 27°C was considered as thermoneutral reference. Exercise type which prominently utilises the elastic properties of the muscles (very fast stretch-shortening cycle exercise), drop jump, was found to be most susceptible for cooling. To study the effects of different levels of cooling the subjects sat at 27°C, 20°C, 15°C and 10°C for 60 min on different occasions. After the exposure to 10°C the subjects performed rewarming walking exercise. The results showed that muscular performance and its variables decreased when ambient exposure temperature was lowered (decreasing muscle temperature) but recovered with rewarming exercise (increasing muscle temperature). An unexpectedly low level of cooling, exposure to 20°C, was found to be sufficient to significantly decrease muscular performance (17 % · $°C^{-1}$ in T_m). In relation to muscle temperature a dose - dependent change in the co-activation of the working agonist-antagonist muscle pairs was found. The activity of the agonist muscle during preactivity and stretch phases increased (ca. 34 % and 32 %, respectively), whereas during the shortening phase it decreased (ca. 21 %). During the shortening phase, on the contrary, the activity of the antagonist muscle increased (ca. 47 %). This "braking effect" of agonist-antagonist muscle pairs during shortening phase is, in part, responsible for the decreased muscular performance. Alterations in the T- and H-reflexes could be related to the EMG-activity changes of the lower leg muscles observed during a stretch-shortening cycle after cooling.

Key words: Neuromuscular performance; motor co-activation; cooling; agonist; antagonist

ACKNOWLEDGEMENTS

This study was carried out at the Oulu Regional Institute of Occupational Health, ORIOH, during the years 1991-1998. I wish to thank Research Professor Juhani Hassi, previous director of ORIOH and present director Professor Lauri Pyy, for placing the facilities of the Institute at my disposal.

Docent Hannu Rintamäki, PhD, the Head of the Laboratory of Physiology at ORIOH, my colleague and dear friend, has guided and inspired me in the difficult paths of science throughout this study. Through discussions in the lab and in the wilderness, his unfailing support has been a great source of strength for me. I also wish to thank my other supervisors Professor Paavo Komi, the Head of the Department of Biology of Physical Activity, University of Jyväskylä and Professor Heikki Rusko, the Director of Research Institute for Olympic Sports for fruitful collaboration and support.

I am grateful to Professors Ulf Bergh and Albert Gollhofer for their valuable comments on the manuscript of this thesis.

There are several persons who have contributed to my work. I would particularly like to thank my co-authors: Research Professor Juhani Hassi, the Director of Cold Work Action Programme also for his support in different phases of this work, Researcher Sirkka Rissanen M.Sc., ORIOH, for fruitful discussions while being "the spirit in the same situation", preparing her thesis, Tero Mäkinen M.Sc., ORIOH, especially for assisting in the measurements and analysis of the different studies, Mr Vesa Martikkala M.Sc., Oulu Deaconess Institute, especially for skilful mathematics, Professor Uolevi Tolonen, Oulu Central Hospital, Department of Clinical Neurophysiology, for making the H-reflex measurements possible and MD Seppo Rytky, Oulu Central Hospital, Department of Clinical Neurophysiology, for measuring and analysing the H-reflex in the last study. I am grateful to MD, PhD Timo Lauri, MD Raija Kerätär and MD Veikko Kujala for their medical assistance in muscle temperature measurements.

I would especially like to thank the work physiology group of ORIOH and also the rest of the personnel for creating such a wonderful working atmosphere.

Finally I wish to thank my family and relatives for their wonderful support. My daughters, Tuuli and Kaisli, never failed in wondering; what does dad really do? And sometimes, especially in the nights in front of the PC, I found myself wondering the same thing.

This study was financially supported by the Finnish Work Environment Fund.

LIST OF ORIGINAL ARTICLES

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

I Rintamäki, H. Oksa, J. Rissanen, S. Hassi, J. and Rusko H. Power output, fatigue and recovery in one minute jumping test in cooled and warmed men. (submitted).

II Oksa, J. Rintamäki, H. Mäkinen, T. Hassi, J. and Rusko, H. (1995) Cooling-induced changes in muscular performance and EMG-activity of agonist and antagonist muscles. Aviat Space Environ Med 66: 26-31.

III Oksa, J. Rintamäki, H. Mäkinen, T. Martikkala, V. and Rusko, H. (1996) EMG-activity and muscular performance of lower leg during stretch-shortening cycle after cooling. Acta Physiol Scand 157: 71-78.

IV Oksa, J. Rintamäki, H. and Rissanen, S. (1997) Muscular performance and EMG-activity of the working muscles with different levels of cold exposure. Eur J Appl Physiol 75: 484-490.

V Oksa, J. Rintamäki, H. and Rissanen, S. (1996) Recovery of muscular performance and EMG-activity with rewarming exercise in the cold. Human Movement Science 15: 591-603.

VI Oksa, J. Rintamäki, H. Rissanen, S. Rytky, S. Tolonen, U. and Komi, P.V. Leg T- and H-reflexes after cooling and local rewarming (submitted).

Some unpublished results will also be presented.

ABBREVIATIONS

BCG = body center of gravity
BL = blood lactate
CD = contraction duration
Fpush-off = average force production during the shortening phase
EMG = electromyography
aEMG = averaged integrated electromyography
GAL = gastrocnemius lateralis muscle
GAM = gastrocnemius medialis muscle
H_{amp} = maximum amplitude of H-reflex
H_{lat} = H-reflex latency
H_{max}/M_{max} = relation between maximum H- and M-amplitudes
HR = heart rate
ISI = inter spike interval of T-reflex
T_{amp} = maximum amplitude of T-reflex
M_{amp} = maximum amplitude of M-response
MPF = mean power frequency
MVC = maximal voluntary contraction
Q_{10} = the change in the velocity of chemical reactions in the tissue
Q = body heat content
RD = T-reflex duration
RL = T-reflex latency
TA = tibialis anterior muscle
TMA = time to reach maximal EMG-activity
TS = triceps surae muscle
T_b = body temperature
T_c = calf skin temperature
T_m = muscle temperature
T_{re} = rectal temperature
T_{sk} = mean skin temperature
T_s = shin skin temperature
t_{conc} = duration of shortening phase during ground contact
t_{ecc} = duration of stretch phase during ground contact
t_f = flight time of a jump
t_t = total time of the jumps
S = heat storage
SOL = soleus muscle
SSC = stretch-shortening cycle
Vel = take-off velocity
VO_2 = oxygen consumption
P_{jump} = mechanical power of jumping

CONTENTS

1 INTRODUCTION

Temperature, whether cold or hot, is a fundamental modifier of human function. In polar and circumpolar areas people are often exposed to cold. In Finland approximately 400.000 workers have been estimated to be exposed to cold in their work (Rantanen and Lehtinen 1992). At the same time when the number of outdoor workers decrease the number of people spending their leisure time outdoors increase. This thesis focuses on the interaction of cold temperature and neuromuscular performance and thus the central feature of this thesis is to combine the problems and methods of thermal and exercise physiology. A short review of the area is given in the next paragraphs.

Cold causes uncomfortable thermal sensations and decreases muscular performance (Bergh 1980; Bennett 1984; Sargeant 1987). Several parameters describe the decreased capacity to perform muscular work. In dynamic work maximal force production, the power of short term exercise and movement velocity decreases, especially in fast exercises ((Bergh and Ekblom 1979a; Davies and Young 1983; Sargeant 1987). The time to reach the maximal force level and relaxation rate in isometric work increases (Faulkner et al. 1990).

The effects of cooling on various components of neuromuscular performance such as elasticity, duration and velocity as well as co-activation of agonist-antagonist muscle pairs (co-ordination) are less known. Usually in cold research isometric (e.g hand grip, Petrofsky and Lind 1980) or concentric (e.g. bicycle ergometer, Petrofsky 1979) exercise types have been used. A comparative search for cold sensitive exercise types that would ease to study the effects of cooling has not been done.

In the majority of the studies the level of cold exposure has been severe in order to assure performance decrement. However, the minimum level of cooling which is sufficient to deteriorate performance has not been identified. Rewarming exercise has been used to recover decreased skin, core or muscle temperatures (Sawka et al. 1984; Rintamäki et al. 1992). However, it is not known how rewarming exercise may recover decreased performance.

Mainly biochemical mechanisms has been found to cause the decrement in muscular performance when the tissues are cooled. Cooling slows the hydrolysis of ATP (e.g. Blomstrand 1985; Ferretti et al. 1992a), the release and uptake of Ca^{2+} from and to sarcoplasmic reticulum. Also, the Ca^{2+} sensitivity of the actomyosin complex is weakened (Hartshorne et al. 1972; Stephenson and Williams 1985). The velocity of the chemical processes in the cooled muscles are slower, the Q_{10} values varying between 1.5 - 3.0 (Rome 1990). This means that when the temperature of the tissue decreases 10°C there is 1.5 - 3.0 times change in the velocity of its chemical processes. Also the increased viscosity and stiffness of the muscles may deteriorate performance (Hunter 1952). Neural changes may as well decline performance: the decreased nerve and/or muscle conduction velocity may weaken muscle contraction (Paintal 1965; Bigland-Ritchie et al. 1981).

One possible mechanism which may deteriorate muscular performance due to cooling is the changes in the co-activation of agonist-antagonist muscle pairs (reflected by EMG). The effects of cold on co-activation are not well understood. Faulkner (1990) suggested that cooling may decrease the power production of agonist muscle and increase the power absorption of antagonist muscle. Bawa et al. (1987) found that during light work the antagonist muscle was activated during cold-induced shivering whereas at thermoneutrality the same was not observed. Bergh and Ekblom (1979a) showed that cooling did not affect the EMG-activity of the thigh muscles during jumping exercise. However, only one subject was used in their EMG measurements. Thus, it is important to know to what extent cooling can cause changes in the co-activation of agonist-antagonist muscle pairs, therefore possibly affecting muscular performance.

The present study was designed to evaluate the interaction between body cooling and neuromuscular performance.

2 REVIEW OF THE LITERATURE

Cold ambient temperature is a very basic modifier of human life especially in polar and circumpolar regions. Beside on man itself cold has had and has all the time a tremendous effect on e.g housing, transportation and industry. One of the first physiological effects of cold-induced cooling on man is the reorganisation of blood circulation. The periphery is vasoconstricted thus, leading to a diminished blood flow in the hands and legs but assuring an adequate blood flow in the central part of the body around the vital organs (Alexander 1974). Cooling has an adverse effect on physical performance capacity. The following literature review will focus on the effects of cooling on body heat balance, muscular performance and its components, functional properties of the muscles, EMG and T- and H-reflexes.

2.1 Body heat balance in the cold

Body heat balance is a sum of two factors: internal heat production and heat transfer between the body and ambient environment. This relationship is described by heat balance equation:

(1) $S = M - (\pm W_k) \pm E \pm R \pm C \pm K$ $(W \cdot m^{-2})$

where, M = metabolic heat production
W_k = energy leaving (positive for concentric exercise) or entering (negative for eccentric exercise) the body as external work
E = evaporative heat loss
R = radiative heat loss
C = convective heat loss
K = conductive heat loss

The sum of these processes is the storage of body heat (S) which is positive if heat is gained and negative if heat is lost.

When humans at rest are exposed to cold air heat flows from the body core to the surrounding environment primarily via dry (radiative, conductive and convective) heat loss mechanisms. Wind increases convective heat loss (Santee and Gonzalez 1988) and since water has a much higher thermal capacity than air (approx. 25-fold in still water) conductive heat transfer is greater when immersed to water than when exposed to air of the same temperature (Nadel et al. 1974; Gonzalez 1988). Clothing provides thermal insulation between the body and environment therefore limiting dry heat loss. However, if clothing is wet (due to e.g. rain or sweating) it provides considerably less insulation than dry clothing. Therefore, environmental characteristics besides temperature influence heat loss and the resulting physiological responses.

2.2 The effect of cooling on neuromuscular performance

Performance is here considered as a sum of the function of neural and muscular components. Though they can not be operationally separated, from hereon in this review the first three chapters has more emphasis on muscular component and the latter three more on neural component.

It is well established that cooling decreases muscular performance (Pugh 1967; Kaijser 1970; Davies et al. 1975; Bergh and Ekblom 1979a and b; Davies and Young 1983; Blomstrand et al. 1984; Sargeant 1987). To what extent muscular performance decreases depends mainly on the exercise type and the level of cooling. The exercise types can be roughly divided into isometric and dynamic exercises.

2.2.1 Isometric exercise

The deterioration in performance is usually expressed as absolute (%) or muscle temperature (% $\cdot$ °C^{-1} in T_m) related decrease. In human studies maximal isometric force level has been found to be relatively stable within the muscle temperature range from 27 to 40°C (Clarke and Royce 1962). Within that temperature range Bergh and Ekblom (1979a) found a decrease of 2 % MVC $\cdot$ °C^{-1} T_m (maximal voluntary contraction, per degree of change in muscle

temperature). Very small decrease or no effect on MVC was observed by Clarke et al. (1958) and Bundschuh and Clarke (1982) and an increase in MVC was found by McGown (1967). On the other hand, literature quite uniformly reports that with muscle temperatures below 27°C the isometric MVC decreases, the absolute decrement being within the range from 11 to 19 % (Johnson and Leider 1977; Coppin et al. 1978; Oliver et al. 1979; Davies et al. 1982; Barnes 1983; Buller et al. 1984). A prerequisite for this is however, a very pronounced lowering in muscle temperature. For example, in the study of Johnson and Leider (1977) the 30 min local cold water exposure (10°C) of the forearm has been reported to result in a 21°C lowering in muscle temperature (Fischer and Solomon 1965) which leads to a local muscle tissue temperature of approximately 11°C. The measuring depth of the muscle temperature was not, however, reported. It also seems that gender has an effect: the women being less susceptible for loss of muscle force due to cooling (Cornwall 1994).

During sustained isometric exercises cooling, on the contrary, seems to have a beneficial effect. The endurance time is increased and the rate of fatigue is slower (Clarke et al. 1958; Clarke and Stelmach 1966; Petrofsky 1979; Bundschuh and Clarke 1982).

2.2.2 Dynamic exercise

In general, the ability to perform dynamic exercises is more readily disturbed by cooling than is the isometric exercise. Most often used dynamic exercise types are bicycling and jumping (Asmussen et al. 1976; Bergh and Ekblom 1979a; Bergh 1980; Sargeant 1983; Blomstrand et al. 1984; Sargeant 1987; Crowley et al. 1991; Ferretti et al. 1992a). Less often used exercise types are e.g. sprinting, isokinetic leg exercise or manual performance (Haymes and Rider 1983; Bergh and Ekblom 1979a; Giesbrecht and Bristow 1992). The literature reports, however, quite uniformly decreases in dynamic performances regardless of the exercise type, the decrement in general being approximately of the order of 2 - 10 % · °C^{-1} in T_m (Bergh 1980; Sargeant 1987). However, even greater values have been reported. Bergh and Ekblom (1979b) found that cooling produced a 55 % absolute decrement in maximal working time while muscle temperature decreased by 3.4°C, corresponding to 16 % · °C^{-1} in T_m.

There are reports that the more the temperature of the working muscle tissue is decreased over a rather wide range (ca. 36 - 25°C) the more is the amount of deterioration in muscular performance increased (Clarke and Royce 1962; Bergh and Ekblom 1979b; Petrofsky and Phillips 1986; Ferretti et al. 1992a). However, due to rather large declines in the reported muscle temperatures, the lowest level of cooling which is sufficient to deteriorate muscular performance has not been identified. Moreover, it has been reported that passive rewarming returns muscle force back to thermoneutral level during three hour recovery period (Oliver et al. 1979). How active rewarming exercise recovers the deteriorated performance in dynamic exercises is not known.

2.2.3 "Traditional" components of muscular performance

This chapter focuses on parameters conventionally regarded as basic components of muscular performance namely: endurance, force, power, velocity and co-ordination (in this thesis referred to as co-activation of agonist-antagonist muscle pairs), or their combination.

Cooling affects all the components of muscular performance. The amount of cooling-induced decrease in various components of muscular performance varies to some extent from study to study depending e.g. on the type, duration and intensity of the cold exposure. The intensity of cold exposure is often expressed in terms of muscle temperature. In the studies cited below the lowest muscle temperature reported was ca. 24°C, but the rather usual muscle temperature caused by various exposures was around 30°C.

Endurance during bicycling (determined as time to exhaustion), for example, has been reported to decrease 55 % (Bergh and Ekblom 1979a) and 38 % (Blomstrand et al. 1984) by cooling. On the contrary, Haymes and Rider (1983) reported a 20 % increase in endurance during isokinetic leg exercise, the difference being possibly due to different kind of exercise used.

Maximal muscle force (expressed as force, torque or instantaneous power) has been reported to decrease by 29 % and 27 % during jumping exercise (Asmussen et al. 1976; Ferretti et al. 1992a). During isokinetic leg exercise maximal muscle force declines from 4.4 % to 10 % (Haymes and Rider 1983) and from 4.7 to 4.9 % $\cdot$ °C^{-1} in T_m (Bergh and Ekblom 1979b). During bicycling Sargeant (1987) reported a 20 % and Crowley et al. (1991) a 30 % decrease in power. Hart et al. (1985) reported that muscle force declines during both eccentric and concentric phases of stretch-shortening cycle due to cooling, but after cooling the force oscillation from the predetermined level is less.

Cooling decreases both metabolic and mechanical power (Ferretti 1992; Shephard 1993). Bergh (1980) found that anaerobic power declined by 4 - 6 % $\cdot$ °C^{-1} in T_m. The mechanical power has been reported to decrease during bicycling by 17 % (Sargeant 1987) and by 26 % (Crowley et al. 1991) and during jumping by 27 % (Ferretti et al. 1992a).

Velocity of movement is also deteriorated due to cooling. With lowered muscle temperature (from 38.3 to 31.4°C) the velocity of the fly - wheel during cycling was 32 % slower (4.7 % $\cdot$ °C^{-1} in T_m, Bergh and Ekblom 1979b). Sargeant (1983 and 1987) demonstrated that the decrease in muscular performance is dependent on muscle contraction velocity. With faster muscle contraction velocities (bicycling 144 rpm) muscular performance was more deteriorated than with slower muscle contraction velocities (bicycling 54 rpm). Lakie et al. (1986) also found that fast wrist movements are more easily reduced due to cooling than slower ones. Asmussen et al. (1976) reported that during ground contact (jumping) the positive velocity (upwards) slowed from 2.86 m $\cdot$ s^{-1} to 2.41 m $\cdot$ s^{-1} due to cooling. At the same time the average duration of stretch phase was uninfluenced but the shortening phase increased from 118 to 140 ms due to cooling (Asmussen et al. 1976).

Cooling also affects the relationship between force production and velocity. Force-velocity curve is shifted to the left (Binkhorst et al. 1977; Bergh and Ekblom 1979a), which means that with a given force the velocity of movements or muscle contraction decreases after cooling. A similar shift is also seen in force-time curve. In a given time less force is produced after cooling (Clarke and Royce 1962).

The studies concerning the co-activation of the agonist-antagonist muscles after cooling are sparse and mainly attributed to increased thermoregulatory muscle tonus or shivering. During increased thermoregulatory muscle tonus motor units are firing asynchronously to produce heat but not motion. During vigorous shivering the number of active motor units may become so high that the firing becomes synchronous, which can be seen as motion (Pozos et al. 1986). This synchronous firing may disturb the preciseness of motion (co-ordination) (Meigal et al. 1998), but can be momentarily stopped (increasing preciseness) e.g. by dynamic work, changing body posture and mental concentration (Freund 1983; Lupandin and Meigal. 1994; Meigal et al. 1994). It has been found that during light exercise (extension of the elbow) after cooling the co-activation of the antagonist muscle (*biceps brachii*) occurs simultaneously with the agonist muscle (*triceps brachii*), whereas in thermoneutrality only the agonist is active (Bawa et al. 1987). In their study with one subject, Bergh and Ekblom (1979a) reported that no changes occurred in the relationship of agonist and antagonist EMG-activity onset during jumping in relation to changes in muscle temperature (from ca. 38 to 30°C).

2.2.4 Functional properties of skeletal muscle

In addition to decreased muscular performance cooling has also a profound effect on functional properties of skeletal muscle (Elmubarak and Ranatunga 1984; Faulkner et al. 1990). It has been well verified that the rate of tension development in the beginning of muscle contraction i.e. the time to maximum force level (twitch or tetanic tension) is temperature dependent (Clarke and Royce 1962; Ranatunga 1982; Ranatunga 1984; Ranatunga et al. 1987; Wylie and Ranatunga 1987). The temperature sensitivity (Q_{10}) of the rate of tension development in humans has been shown to be approximately 1.5 (Ranatunga et al. 1987), whereas in animal studies Q_{10} values over 2 have been reported (Bennett 1985; Kössler and Kuchler 1987).

A similar temperature dependence has also been found for the rate of relaxation at the end of muscle contraction (Ricker et al. 1977; Ranatunga 1982). It is generally described as half relaxation time i.e. the time from the maximum tension to 50 % of the maximum tension (Davies et al. 1982). The Q_{10} of the rate of relaxation in humans has been reported to be approximately between 1.7 - 2.3 (Wiles and Edwards 1982; Ranatunga et al.1987), whereas in animal studies Q_{10} values over 2.5 have been reported (Bennett 1985; Kössler and Kuchler 1987).

The velocity of muscle contraction itself, shortening and lengthening, is also slower in a given time when muscle tissue is cooled (Faulkner et al. 1990).

Therefore, the power production of the muscle during shortening is less and power absorption during lengthening is greater thus leading to a less powerful action of a muscle.

The biochemical reasons underlying the increased time to peak tension, half relaxation time and velocity of muscle contraction have been related to decreased ATP-hydrolysis (Edwards et al. 1971; Ferretti 1992), slowed Ca^{2+} release and uptake from the sarcoplasmic reticulum (Kössler et al. 1987) and decreased calcium sensitivity of the actomyosin (Hartshorne et al. 1972; Sweitzer and Moss 1990). These changes may also cause impaired cross-bridge formation and breakdown or decreased force per cross-bridge (Sweitzer and Moss 1990).

There are only few studies concerning the effects of cooling on elasticity and stiffness of the muscles. It has been reported that the stiffness (i.e. the ratio between force and length changes) of the muscle-tendon entity does not significantly change due to cooling (Asmussen 1976), which is unexpected because at lower temperatures the stiffness of the tendons and joints has been reported to increase (Hunter et al. 1952; Rice 1967; Apter 1972). However, Lakie et al. (1986) reported increased stiffness of the wrist for large and moderate, but not for small amplitude movements when the forearm was cooled.

Asmussen et al. (1977) studied the effect of cooling on the capacity of the muscles to utilise their elastic properties by comparing the jump height of static and countermovement jump. The height of the static jump describes the force produced without the "interference" of elastic components of the muscles, whereas the countermovement jump also utilises the elastic components. It was found that the "gain" in height i.e. the increase in the countermovement jump height in relation to the height of the static jump increased after cooling. Simultaneously the EMG-activity of the working muscles increased. These results led to the conclusion that the utilisation of elastic components of the muscle are enhanced after cooling (Asmussen et al. 1977). Unfortunately, the contribution of tendon on the elasticity of muscle - tendon entirety has remained obscure.

Cooling also slows nerve and muscle conduction velocity which may result in a slower and less powerful muscle contraction (Paintal 1965; Vanggaard 1975; Denys 1991; Bigland-Ritchie et al. 1992). The decrease in conduction velocity has been reported to have a Q_{10} of approximately 1.4 (Lowitzsch et al. 1977). The absolute reduction in nerve conduction velocity has been reported to vary between 1.1 - 2.4 $m \cdot s^{-1}/°C$ (Denys 1991).

The motor unit recruitment pattern is also affected by cooling. At a given submaximal work level after cooling more and faster motor units are being recruited in order to maintain the work level (Rome et al. 1984; Rome 1990; Faulkner et al. 1990; Rissanen et al. 1996). On the other hand, the greater decrease of fast maximal exercise in comparison to slower exercise (Sargeant 1987) after cooling would suggest that fast motor units are first dropped out during maximal exercise after cooling (Faulkner et al 1990).

2.2.5 EMG

Cooling clearly has a modulating effect on electromyographical (EMG) activity of the muscles (Ricker et al. 1977; Hart et al. 1985). The two conventionally used parameters to describe muscular activity are the amplitude and frequency components of EMG. The literature rather uniformly reports that cooling changes the various parameters of the frequency component: mean power or center frequency (Petrofsky 1979; Winkel and Jørgensen 1991), median frequency (Merletti et al. 1984) or firing rate of individual motor units (Wolf and Letbetter 1975). The decrement seems to depend rather linearly on the level of cooling, the muscle temperature varying approximately between 24 - 40°C (Petrofsky and Lind 1980; Merletti et al. 1984). For example, a 30 min exposure of the forearm to 10°C water in relation to 40°C water decreased the mean power frequency (MPF) from approximately 180 Hz to 100 Hz (Petrofsky and Lind 1980). The effect of cooling seems to be similar regardless of the exercise type (dynamic or isometric) or cooling procedure (water or air) (Petrofsky and Lind 1980; Mucke and Heuer 1989; Winkel and Jørgensen 1991). The decrement in MPF has been connected with simultaneous decrease in nerve conduction velocity (Mucke and Heuer 1989).

The EMG-amplitude does not seem to be as uniformly affected by cooling as the frequency of EMG. There are studies reporting decreased amplitude of the EMG due to cooling (Wolf and Letbetter 1975; Petrofsky and Lind 1980; Mucke and Heuer 1989; Bell 1993) while others report increased amplitude (Sellers et al. 1954; Zipp 1977; Winkel and Jørgensen 1991). In the above cited studies muscle temperature was clearly reduced. At normal muscle temperature range (34 - 39°C) Petrofsky (1979) found no significant difference in EMG-amplitude. The difference may be to some extent explained by different exercise types and cooling procedures. Cold air exposure and dynamic exercise seems to produce increased amplitude (Zipp 1954; Winkel and Jørgensen 1991), whereas cold water and isometric exercise results in a decreased amplitude (Petrofsky and Lind 1980; Mucke and Heuer 1989). These differences are not however, consistent. In the study of Bell (1993) cold air exposure and isometric exercise was used and the amplitude of EMG increased.

The relationship between EMG-activity and force production also seems to be affected by temperature. Bell (1993) found that for a given force level at 10°C the EMG-activity increased whereas at 40°C it decreased. On the other hand, there are studies where no difference between EMG-activity and force production at different temperatures has been found (Holewijn and Heus 1992; Yona 1997).

2.2.6 T- and H-reflexes

Tendon reflex (T-reflex) is a monosynaptic, ipsilateral spinal reflex which is activated by stretching the muscle spindles (during the stretch phase of stretch-

shortening cycle, tapping the tendon or causing a flexion of a joint), which in turn facilitates the following contraction of the agonist muscle and inhibits the contraction of the antagonist muscle (Matthews 1964). Hoffman reflex (H-reflex) is also a monosynaptic spinal reflex which is induced by electrical stimulation of e.g. tibial nerve in popliteal fossa (Funase et al. 1996). The T-reflex depends on both alpha-motoneuron excitability and muscle spindle sensitivity whereas H-reflex is considered to mainly reflect the excitability of the alpha-motoneuron pool (Bishop et al. 1968; Bell and Lehmann 1987; Funase et al. 1996). Many of the studies concerning the effects of cooling on T- and H-reflexes have shown that cooling supresses T-reflex amplitude and enhances H-reflex amplitude (Petajan and Eagan 1968; Knutsson and Mattsson 1969; Urbscheit and Bischop 1970; Mecomber and Herman 1971; Miglietta 1973; Lightfoot et al. 1975; Denys 1990). There is evidence that the suppressed T-reflex amplitude is due to decreased activity of the muscle spindles (Eldred et al. 1960; Ottoson 1960; Michalski and Sequin 1974; Bell and Lehmann 1987) and this may lead to a decreased force production of a muscle (Petajan and Watts 1962; Knutsson and Mattsson 1969). In H-reflex measurements evidence has been presented that cooling enhances the excitability of the alpha-motoneuron pool (Landau et al. 1966; Knutsson et al. 1969, Denys 1990; Arsenault et al. 1993), although opposite results have also been presented (Bell and Lehmann 1987). A decrease in muscle temperature does not seem to be a prerequisite for the possible increase in the excitability of the alpha-motoneuron pool, rather it seems possible that only skin cooling may be sufficient (Arsenault et al. 1993).

3 THE PURPOSE OF THE STUDY

The research of the effects of cooling on neuromuscular performance has mainly focused on isometric or submaximal exercise types. Therefore, the effects of cooling on dynamic maximal exercises and on the various components (force, power and velocity) of muscular performance are less understood. Also, the co-activation of the working agonist-antagonist muscle pairs (representing co-ordination) during dynamic efforts need to be clarified. Therefore, the purposes of the present study were:

1. To evaluate the effect of cooling on muscular performance and its components.

- What is the amount of muscular performance decrement in slow, fast and very fast stretch-shortening cycle exercises?
- Which exercise type is especially susceptible for cooling?
- What is the level of tissue cooling which is sufficient to alter neuromuscular performance?
- How much rewarming exercise is needed to recover from the altered neuromuscular performance?

2. To evaluate the effect of cooling on EMG- and reflex activity of the working muscles

- Does the co-activation of working agonist-antagonist muscle pairs change during different phases of stretch-shortening cycle?
- Can the T- and H-reflex responses in cold be related to possible changes in the co-activation of agonist-antagonist muscle pairs?

4 MATERIAL AND METHODS

4.1 Subjects

A total of 48 healthy male subjects volunteered for the study. Before participating the subjects were medically examined, the experimental protocol was explained and their written consent was obtained. The experimental protocol was in accordance with the Declaration of Helsinki. Table 1 illustrates the physical characteristics of the subjects in different studies.

TABLE 1. The mean (±SD) physical characteristics of the subjects in different studies. Body fat was estimated according to skinfold thickness measurements (Durnin and Womersley 1974)

Study	Age (yr)	Height (cm)	Weight (kg)	Body fat (%)
I (n=9)	22±1	181±4	75±10	18±3
II (n=11)	24±4	174±7	70±7	12±3
III (n=12)	27±2	171±2	70±3	12±5
IV and V (n=8)	26±4	175±8	72±6	15±3
VI (n=8)	24±5	177±5	73±8	14±3

4.2 Thermal exposures and temperature measurements (I - VI)

Before the exercises, the subjects sat motionless for 60 min in a climatic chamber at 27°C (thermoneutral reference) and 10°C dressed in shorts and jogging shoes. During the exposures and following exercise bouts, local skin tempera

tures from seven (I - III) or eight (IV - VI) sites (skin temperatures of calf and shin are denoted as T_c and T_s, respectively), rectal temperature (T_{re} 10 cm depth) (Yellow Springs Instruments, YSI 400 series) and muscle temperature (T_m) with an elastic wire thermistor from *gastrocnemius medialis* (III - VI), *deltoideus* and *triceps brachii* muscles (II, 3 cm depth underneath the skin surface, YSI 511) were recorded with one minute interval in a data logger (Squirrel 1200, Grant, UK). Mean skin temperature (T_{sk}) was calculated by weighting the 7 - 8 local skin temperatures by representative areas (Hardy and DuBois 1938). In study V body temperature (T_b) was calculated by the equation:

(2) $$T_b = T_{sk} \cdot 0.35 + T_{re} \cdot 0.65.$$

Body heat content (Q) was calculated by the equation:

(3) $$Q = 3.48 \cdot T_b \cdot \text{body weight (kg)},$$

where 3.48 = is the specific heat of the body ($kJ \cdot kg^{-1} \cdot °C^{-1}$) (Minard 1970).

In study IV the subjects were exposed also to ambient temperatures of 15°C, 20°C and two subjects to 25°C. In study V the effect of rewarming exercise on decreased muscular performance was studied. After the 60 min exposure to 10°C the subjects performed a drop-jump. Thereafter, the subjects were allowed to walk on a treadmill at 10°C for 5 min with the speed of 1.4 $m \cdot s^{-1}$ (5 $km \cdot h^{-1}$) and after each walking bout they repeated the drop-jump. This cycle was repeated until the thermoneutral reference value in the flight time of the jump was achieved. In study VI the subjects were exposed to 10°C twice. During the second exposure to 10°C ($10°C_{lw}$) the subjects were dressed similarly but their lower legs were kept warm with electrical pillows wrapped around their lower legs from knee to toe.

4.3 Measures of muscular performance

4.3.1 Rebound jumping exercise (I)

The subjects performed a 60 s maximal rebound jumping test on a contact mat (Digitest, Muurame, Finland) with the advise to jump as many times and as high as possible. The hands were kept at the hips and the subjects were advised to bend their knees approximately to 90° during each ground contact. During the test the accumulated flight time and total time were recorded. The number of jumps could be calculated afterwards from the accumulated flight time recordings. The mechanical power of jumping was calculated according to Bosco et al. (1983):

(4) $P_{jump} = (g^2 \cdot t_f \cdot t_t)/4 \cdot n \cdot (t_t - t_f)$

where P_{jump} = the mechanical power of jumping ($W \cdot kg^{-1}$)
$g = 9.81\ m \cdot s^{-2}$
t_f = flight time (s)
t_t = total time (s)
n = number of jumps

After the test a recovery period was continued as long as the oxygen consumption returned to a resting level. During ground contact performance consists of stretch phase (downward movement, eccentric contraction) and of shortening phase (upward movement, concentric contraction). In normal human locomotion this combination of eccentric and concentric contractions forms a natural type of muscle function which is called the stretch-shortening cycle (SSC, Komi 1984). This exercise type is referred to as fast SSC exercise because the duration of the cycle was approximately 700 ms.

4.3.2 Ball throwing exercise (II)

The subjects performed a full effort overhead throwing test with both arms, where five balls, weighing 0.3, 0.6, 1.0, 2.0 and 3.0 kg were thrown. The diameter of the balls ranged from 18 to 25 cm. Before the tests the subjects were allowed to get accustomed with the throwing exercise (they performed the test as many times as they wanted, at least three times) in order to avoid a learning effect during the experiments. The flight times of the balls were measured (Newtest 1500, Oulu, Finland) by using ten infra-red beams, located 10 cm apart from each other in a vertical line and a contact mat functioning with an on/off basis (Newtest, Oulu, Finland). Time measurement was activated when the ball reached the infra-red beams and stopped when the ball hit the contact mat attached to the wall 250 cm behind the infra-red beams. The throwing spot was 100 cm in front of the beams.

The test was performed in a standing position, legs slightly apart. The subjects were holding the ball with both arms and after a signal they lifted the ball above their head and threw it with full effort. During the test a recovery time of approximately 30 s was allowed between consecutive balls. The subjects were allowed to have one trial and the performance was accepted if the throwing spot was not overstepped. The velocity (V) of each ball was calculated:

(5) $V = D / t,$

where D = distance (m)
t = time (s)

This exercise type is referred to as slow SSC exercise because the duration of the cycle was close to 1 s, the stretch phase being particularly slow (700 ms or more).

Two factors in the measuring system may cause variation in the ball velocities: the diameter and the flight path of the ball. Balls with small diameter hitting directly in the middle of two IR beams causes shorter flight distance and flight path with an inclination downwards causes longer flight distance. However, the reproducibility of the ball velocities, expressed as coefficient of variation, was found to be good, varying between 1.3 - 3.1 % (II). The test was randomised by exposure and ball weight.

4.3.3 Drop jump exercise (III - VI)

The subjects performed a drop-jump exercise (Komi and Bosco 1978) from a 40 cm bench two times. In study III various stretching velocities (drop heights) were used in order to find an optimal stretching velocity for potentiation of elastic energy. The subjects dropped from six different bench heights 10, 20, 30, 40, 50 and 60 cm. The exercise was performed in the climatic chamber at the exposure temperature. They dropped freely from the bench onto a force plate (Kistler 9287A, Switzerland or Digitest force plate, Finland) and performed an instantaneous maximal rebound jump.

In the drop-jump exercise *triceps surae* (TS) muscle complex (agonist muscle) generates force to extend the ankle, whereas *tibialis anterior* (TA) muscle flexes the ankle (antagonist muscle).

Stretch and shortening phases were determined from the motion analysis and force plate data. Stretch phase started when the tip of the shoe touched the force plate (plate reading positive) and ended when the motion analysis marker attached to the knee stopped moving downwards. Shortening phase started when motion analysis knee marker began to move upwards and ended when the tip of the shoe left the force plate (plate reading zero). All the measuring devices were started simultaneously.

The subjects were asked to perform the jump with as straight legs as possible (knee angle was between 150-170°) in order to fully utilise the capacity of TS muscle complex. Before the tests the subjects thoroughly practised the drop-jump with an instructor in order to avoid learning effect during the experiments. This exercise type is referred to as very fast SSC exercise because the duration of the cycle was less than 300 ms. The reproducibility of the jump height (5 subjects, 5 consecutive jumps), expressed as coefficient of variation, was 2.3 %. Thermal exposures and consequently drop-jump exercises were performed in a random order.

4.4 Measurements

4.4.1 Heart rate, oxygen consumption and blood lactate (I)

Before and during the jumping exercise as well as during the recovery period heart rate (HR) was measured (Sport Tester, Polar Electro, Finland) continuously and stored at 5 s intervals. The resting oxygen consumption (VO_2, 202 Ergospirometer, Medikro, Finland) level was obtained before the jumping exercise began. Oxygen consumption was also measured during the exercise (VO_{2aver}) and recovery period. Recovery had occurred when oxygen consumption returned to the resting level and to illustrate recovery 4 minute postexercise cumulative oxygen consumption (VO_{2recov}) is reported in the results section. Blood samples for lactate (BL) determination (UV-method, Boehringer, Mannheim, Germany) were collected from the tip of the finger just before (BL1) the exercise, immediately after it (BL2) and on the third minute of recovery period (BL3).

4.4.2 EMG-activity (II - VI)

Table 2 illustrates from which muscles the electromyographic activity (EMG) was measured in different studies.

TABLE 2. The muscles studied by EMG

II	III - VI
Triceps brachii (TB)	Triceps surae (TS)
Deltoideus (anterior part, DA)	Gastrocnemius medialis (GAM)
Rectus abdominis (RA)	Gastrocnemius lateralis (GAL)
Erector spinae (ES)	Soleus (SOL)
	Tibialis anterior (TA)

Surface EMG signals were collected with a sampling rate of either 1250 Hz (Mespec 4001, Mega Electronics, Finland, II - V) or 1000 Hz (ME4000, Mega Electronics, Finland, VI), using pre-gelled bipolar electrodes (Medicotest, M-OO-S, Denmark). Before entering the climatic chamber the electrodes were placed in the middle part, over the belly of the muscle, except in SOL muscle where the electrodes were placed in the descending lateral portion, approximately 15 cm above *lateralis malleolus*. The measuring height for RA and ES muscles was approximately at the L_3 vertebra level. The distance between recording contacts was 2 cm. To ensure the accuracy of replacing the electrodes, their places were carefully marked on the skin with waterproof drawing ink. The markings were clearly visible throughout the experiments.

The measured EMG signal was amplified 2000 times (preamplifier situated 6 cm after the measuring electrodes) and signal band between 20 and 500 Hz was full wave rectified and integrated with a 13 ms time constant and further averaged by dividing the integral by measuring time (averaged inte-

grated EMG, aEMG II - V). The technical qualities of the amplifier were: common mode rejection ratio (CMRR) 130 dB, noise 0.5 μVrms and input impedance 20^9 ohm. The EMG results that are presented from study VI in this thesis are raw EMG values. To assess the frequency component of the EMG, the power spectrum was estimated by moving Fast Fourier Transform (FFT window, 256 points). From the power spectra mean power frequency (MPF) was calculated to describe the changes in the frequency component. Butterworth filtering was used in the 20 - 500 Hz measuring band.

Due to the close geometrical arrangement of EMG measurements in TS muscle crosstalk between the different heads of this muscle is inevitable. It is also quite likely to occur to some extent between TS and TA muscles. However, to minimise this effect the electrodes at TS muscle were placed as far as possible from the electrodes at TA muscle (Winter et al. 1994).

In the ball throwing exercise the EMG measurement started simultaneously from the same signal that was given for the subjects to throw the ball. The EMG measurement was stopped automatically by a switch when the ball left the hands. The time to reach the maximal level of EMG-activity (TMA) during contraction of TB and DA muscles were analysed. The threshold for the beginning of contraction was set at 20 % of maximal activity. Contraction duration of TB muscle (CD, started from 20 % of maximal activity and ended when the ball left the hands) was also analysed. The EMG analysis of all data covered the same period as the CD of TB muscle.

In the drop jump exercise the EMG measurement started from a signal that was given for the subjects to perform the drop-jump. An on - off connector was attached to the sole of the shoe which gave a signal of the contact phase to the EMG measurement device. The EMG data was analysed during the preactivity phase (100 ms before the beginning of stretch phase) and during the stretch and shortening phases of SSC. The results from the three agonist muscles are presented as a sum divided by three thus representing the activity of TS muscle complex. The results of the aEMG and MPF were compared intra-individually against the results obtained at 27°C.

4.4.3 Ground reaction forces (III - VI)

To measure ground reaction forces during the contact phase of the drop-jump a piezo-electric force plate system was used (Kistler 9287A, Switzerland), except in study III where strain gauge force plate (measuring frequency 200 Hz and amplification 1000 times, Digitest, Finland) was used. The measuring frequency of the Kistler force plate was 200 Hz and the obtained signal was amplified 50000 times (Kistler, charge amplifier 9865C). The linearity and hysteresis of the force plate were %FSO < ±1 (full scale output). From the data the average force production ($F_{push\text{-}off}$, in vertical plane) during the shortening phase was calculated, and the flight time (t_f) of the subjects was analysed.

The flight time of the jump is mostly affected by the amount of force produced during the shortening phase (in a vertical plane) and therefore average force production was analysed during this specific phase. An example of a

typical force plate curve and the phases where $F_{push-off}$ and t_f were determined are shown in Fig. 1. The flight time started when the tip of the shoe took off from the force plate (plate reading zero) and ended when the tip of the shoe again touched the force plate (plate reading positive). At each temperature exposure the jump with longer flight time from two trials was chosen to represent the particular temperature exposure.

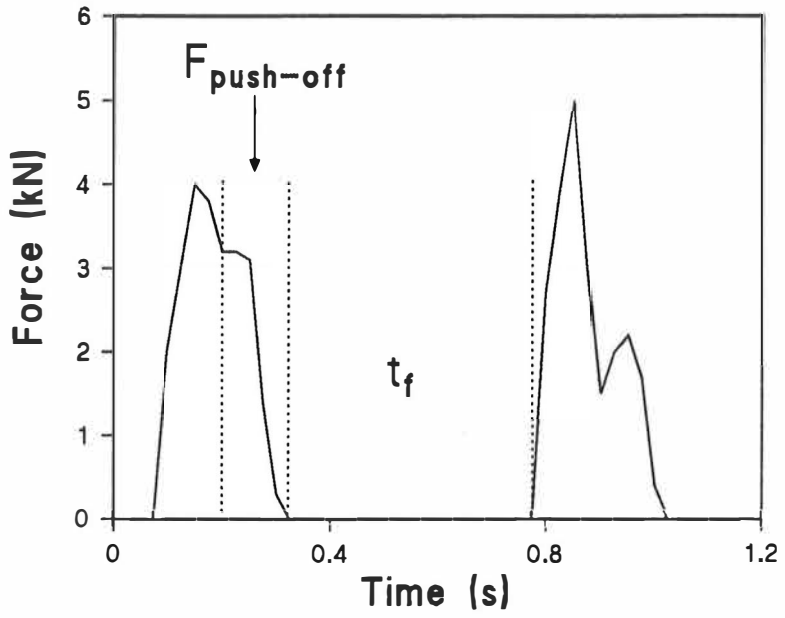

FIGURE 1. An example of a typical force plate curve of one subject at 27°C. The dotted lines indicate the parts from where the average force production ($F_{push-off}$) and flight time (t_f) were determined.

4.4.4 Motion analysis (III - VI)

To determine the duration of the contact (t_c), stretch (t_{ecc}) and shortening (t_{conc}) phases and the take-off velocity (Vel) of the subjects an infra-red optoelectronic motion analysis system was used (MacReflex, Qualisys, Sweden). Take-off velocity was defined as the velocity of the ankle marker at the moment of take-off, tip of the shoe leaving the force plate (on-off connector at the sole of the shoe "off"). The motion analysis system consists of two cameras emitting infra-red light pulses each with a frequency of 50 Hz, with a 10 ms delay in relation to each other (total frequency 100 Hz), videoprocessors and a computer. Before entering the climatic chambers five infra-red light emitting markers were attached to the hip, knee, ankle and to the tip of the shoe. The fifth marker served as a reference and was attached to the side of the force plate. The camera-system sends infra-red light pulses which are reflected back to the camera system from the markers, thus providing a two dimensional

spatial co-ordinate information of the movements of the markers to the motion analysis system. The data was delivered in a digital form to a computer and processed using a WingZ MacReflex-table calculation program. In order to ensure exactly the same distance of the cameras from the subject and to avoid the possibility of losing the marker reflection the cameras were oriented perpendicularly to the measurement plane and were placed one above another. Resolution of the system is 1/30000 of FOV (field of view = 3150 mm at this study) and spatio-temporal noise (STN) 0.1 % thus possible error due to noise is 0.14 mm, calculated as FOV/(FOV/STN).

4.4.5 T- and H- reflexes (VI)

Immediately after thermal exposures the tendon reflex and drop jump measurements were performed. Tendon reflex was elicited (at the exposure temperature) by a mechanical hammer striking with a force of 30±2 N. The subjects were lying prone and relaxed on an examining bed. Their dominant leg was supported from the ankle so that the foot was hanging freely. The place of hammer tap was approximately 2 cm above the tendocalcaneous junction of the Achilles tendon. The reflex was elicited ten times and EMG-activity was measured from TS muscle complex during the last seven. An on - off detector giving a marker to the EMG-device was placed above the Achilles tendon for determining the reflex latency (RL). In addition to RL, the duration of the reflex (RD), peak to peak amplitude (T_{amp}) and time between positive peaks (ISI, inter spike interval) were analysed. RL was defined as the time between the hammer tap and the beginning of the reflex EMG. The reflex was considered to have started when the EMG-activity increased above 10 µV and ended when the activity went below 10 µV. Maximum peak to peak amplitude was defined as the difference in µV from the lowest negative to the highest positive activity during the reflex.

Due to the duration of H-reflex measurements they were done on a separate occasion after similar thermal exposures. The subjects (n=5, from the total group of eight participating the study) were lying prone and relaxed on an examining bed. The Dantec Neuromatic 2000 electromyograph (Dantec Ltd, Denmark) was used for the measurements. A pair of surface electrodes (silver/silver chloride cups) were placed on the calf with the active in the middle of the calf and the reference on the Achilles tendon. In the H-reflex measurement the tibial nerve was stimulated at the popliteal fossa with the cathode proximal to the anode. When determining the M-amplitude and latency the cathode was distal to the anode. The high frequency cut-off was 10 kHz and the low frequency cut-off 10 Hz. In the H-reflex measurement the time window was 100 ms, pulse duration 0.5 ms and stimulus frequency less than one per 5 seconds. Stimulus intensity was gradually increased until the maximum M-response was obtained.

In the first examination the recording and stimulating places were marked with a waterproof pen. Therefore, in the following measurements the measuring site was the same. The latencies of H and M-responses were measured to

the take-off from the baseline and the maximum amplitudes (H_{amp} and M_{amp}) from peak to peak. The ratio between maximum H- and M-amplitudes (H_{max}/M_{max}) was calculated. In the results section the H and M-latencies are not expressed separately but together as H-reflex latency (H_{lat}).

4.5 Statistical methods

The results obtained after the exposure to 27°C were considered as thermoneutral reference values. The results obtained from other exposure temperatures were compared against the results at 27°C. In the statistical analysis paired t-test (I - III, VI) and analysis of variance (ANOVA) (IV, V) were used. After using ANOVA when significant F-ratio was obtained Bonferroni´s (IV) or Duncan´s (V) post hoc test was applied. Pearson´s product moment correlation coefficient (IV, V) was calculated. The significance was accepted at the $p<0.05$ level.

5 RESULTS

5.1 Thermal responses

The standard cooling used in this study (60 min at 10°C) significantly decreased mean and local skin temperatures and muscle temperature but had no effect on rectal temperature. The results of study IV and V show that thermal responses depend on the level of ambient exposure temperature and amount of rewarming exercise. With decreasing temperature all the parameters, except for rectal temperature, decreased.

While doing rewarming exercise at 10°C the muscle and local skin temperatures increased. However, the mean skin and rectal temperature and therefore also body temperature and body heat content were unaffected. Results of the thermal responses illustrating both the effect of the standard cooling and dynamic changes due to different ambient exposure temperatures and rewarming exercise are given in tables 3 and 4.

TABLE 3. Rectal (T_{re}), mean skin (T_{sk}), calf (T_c), shin (T_s) and GAM muscle temperature (T_m) at the end of exposures to 27°C, 20°C, 15°C and 10°C

	T_{re} (°C)	T_{sk} (°C)	T_c (°C)	T_s (°C)	T_m (°C)
27°C	37.0±0.1	32.6±0.3	31.6±0.2	31.8±0.3	32.9±0.5
20°C	37.0±0.1	28.1±0.5***	28.9±0.1***	28.8±0.3***	32.0±0.8
15°C	37.0±0.1	27.5±0.4***	27.1±0.5***	26.5±0.4***	31.0±0.4*
10°C	37.0±0.1	25.8±0.6***	24.0±0.3***	24.1±0.4***	29.5±0.7**

The values are mean±SE of 8 subjects, except muscle temperature (n=6). The significance in relation to 27°C is denoted by * = $p<0.05$, ** = $p<0.01$ and *** = $p<0.001$.

TABLE 4. Rectal (T_{re}), mean skin (T_{sk}), calf skin (T_c), shin skin (T_s) and GAM muscle temperature (T_m) after the exposure to 27°C (reference values), 10°C and after each walking bout

	T_{re} (°C)	T_{sk} (°C)	T_c (°C)	T_s (°C)	T_m (°C)
27°C	37.0±0.1	32.6±0.3	31.6±0.2	31.8±0.3	32.9±0.5
10°C	37.0±0.1 [a]	25.8±0.6	24.0±0.3	24.1±0.4	29.5±0.7
1 walk	36.8±0.1 [a]	24.8±0.5	23.7±0.7	24.9±0.6	30.5±1.0
2 walk	36.8±0.1 [a]	25.1±0.5	25.2±0.8	26.7±0.5*	32.5±1.3 [a]
3 walk	36.9±0.2 [a]	25.0±0.5	25.6±0.3*	28.0±0.3*	33.0±1.2* [a]
4 walk	37.2±0.2 [a]	25.1±0.6	26.3±0.2*	27.8±0.4*	33.4±1.5* [a]
5 walk	37.0±0.2 [a]	24.9±0.4	27.1±0.3*	28.2±0.2*	33.8±0.7 * [a]

The values are mean±SE of 8 subjects, except at 4, n = 7 and at 5, n = 3. In T_m (n = 6), except at 4, n = 5 and at 5, n = 1. The significance in relation to 10°C is denoted by * = $p<0.05$. Letter [a] indicates the points where the data does not significantly differ from the 27°C thermoneutral values.

5.2 Muscular performance

The three different exercise types used in this study consisted of throwing exercise (II, slow SSC), rebound jumping exercise (I, fast SSC) and drop jump exercise (III - VI, very fast SSC). The average decrease in performance due to standard cooling (60 min at 10°C) was 7 % (5 - 9 %), 11 % and 27 % (19 - 31 %), respectively. Expressed as muscle temperature related values the mean decrease in performance was 2.8 % · °C^{-1} in T_m, 4.8 % · °C^{-1} in T_m and 6.8 % · °C^{-1} in T_m for slow, fast and very fast SSC exercise, respectively. Consequently, the most susceptible exercise type for cooling seems to be the very fast stretch-shortening cycle exercise. In all the exercise types a rather steady shift towards lower levels of performance could be seen (Fig. 2).

5.2.1 The components of muscular performance

The mechanical power decreased by 11 % due to cooling (Fig 2). In the rebound jumping exercise blood lactate levels after cooling were significantly lower. Mean oxygen uptake during the exercise did not differ but the oxygen consumption after the exercise was significantly lower with cooled subjects (Table 5).

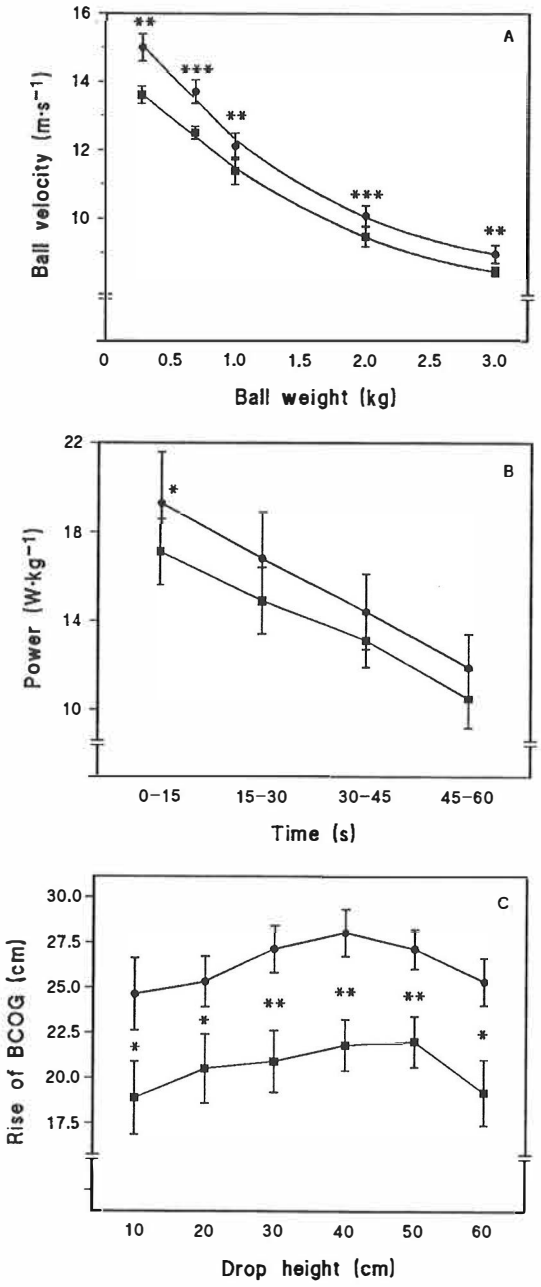

FIGURE 2. The effect of standard cooling (60 min at 10°C) on different maximal exercise types. Slow eccentric - concentric exercise (A, throwing), fast eccentric - concentric exercise (B, rebound jumping exercise) and very fast eccentric - concentric exercise (C, drop jump). Filled circles represent 27°C and squares 10°C. The values are mean±SE, * = $p<0.05$, ** = $p<0.01$ and *** = $p<0.001$.

TABLE 5. Blood lactate before (BL1), after (BL2) and 3 min after rebound jumping exercise (BL3, mmol · l^{-1}), mean oxygen uptake (VO_{2aver} l · min^{-1}) and 4 min cumulative recovery oxygen consumption (VO_{2recov}) after the exposures to 10°C and 27°C

	BL1	BL2	BL3	VO_{2aver}	VO_{2recov}
27°C	2.3±0.3	8.3±1.0	11.9±0.8	2.17±0.06	4.30±0.11
10°C	2.2±0.2	4.8±0.3*	8.5±0.6	2.17±0.12	3.78±0.17*

The significance in relation to 27°C is denoted by * = $p<0.05$.

Heart rate during the exercise after cooling was also lower (Fig 3).

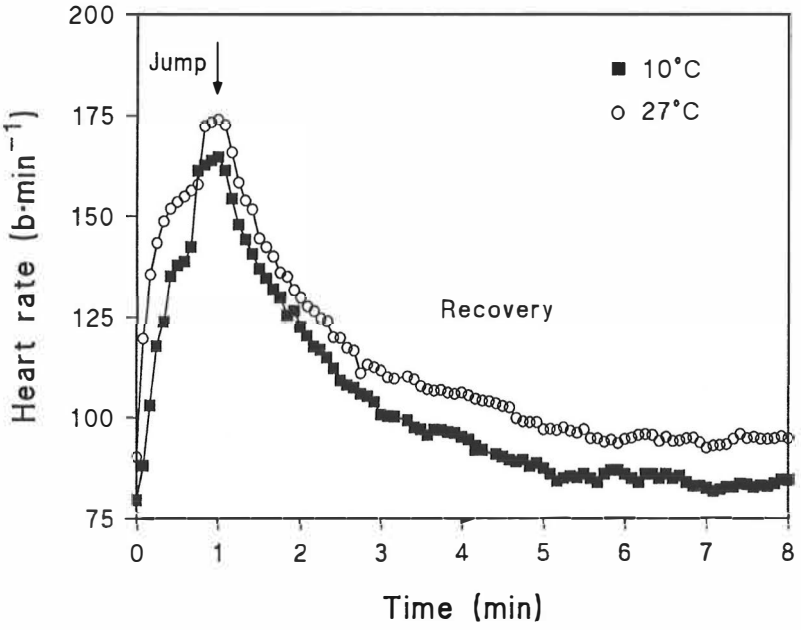

FIGURE 3. The effect of standard cooling on heart rate during rebound jumping exercise.

In the ball throwing exercise (II) the velocity of the balls depicts also the velocity of the arms which, on the other hand, is produced by the muscles involved in the throwing exercise. The use of different ball mass had a different effect on the velocity of the arms. While using the light balls the velocity effect was the greatest, which is seen as a more pronounced decrease in the 0.3 and 0.6 kg ball velocities as compared to that of the heavier balls (Fig 4).

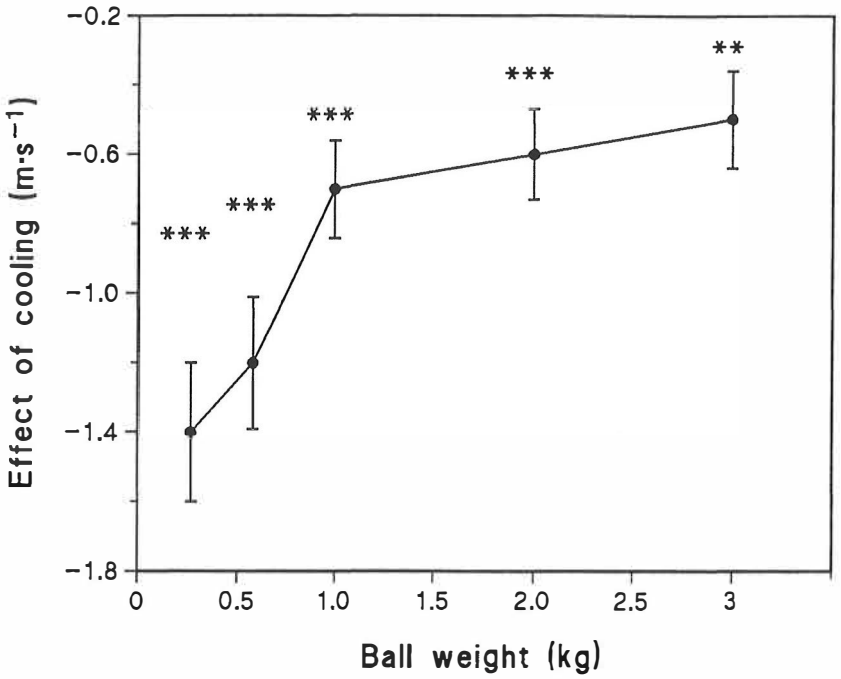

FIGURE 4. The cooling-induced decrease in ball velocities in relation to ball weight. ** = $p<0.01$ and *** = $p<0.001$.

The study III demonstrated that force production during the shortening (push-off) phase of the drop jumps also decreased due to standard cooling in all drop heights. The average force production of all the drop heights at 27°C was 1841±192 N and at 10°C 1348±83 N, the difference being 27 %. At the same time the mean duration of stretch phase increased from 73±3 ms to 101±4 ms and shortening phase from 103±3 to 126±2 ms. Thus, due to cooling the total duration of the stretch-shortening cycle was 29 % (51±3 ms) longer. Simultaneously, knee and ankle angles changed similarly during the contact phase after different thermal exposures, the largest difference being at the lowest point (knee angle <3°, ankle angle <4°).

Different drop heights were used in order to evaluate if cooling has an effect on the optimal stretching velocity for potentiation of elastic energy (III). As expected, there were individual differences so that the optimal stretching velocity after cooling (illustrated as the highest rise of BCG) was slower in four subjects, faster in five subjects and unchanged in three subjects in comparison to drop jumps in thermoneutrality. Therefore, significant group differences due to cooling were not found (Fig. 2c).

5.3 Temperature dependence of muscular performance

In studies IV and V the muscle as well as mean and local skin temperatures of the subjects decreased with passive cooling and increased with rewarming

exercise (Table 3). Muscular performance of the subjects (flight time of the drop jump, 40 cm bench) decreased with lowering the ambient exposure temperature and increased with rewarming exercise (Fig. 5). In a similar manner behaved all the other performance variables, too. Tables 6 and 7 illustrate the changes in the duration of the stretch and shortening phases, total contact time, average force production during the shortening phase and take-off velocity with decreasing ambient exposure temperature and rewarming exercise.

TABLE 6. The duration of stretch phase (t_{ecc}), shortening phase (t_{conc}) and total contact phase (t_c) of the drop jump, average force production during shortening phase ($F_{push-off}$) and take-off velocity (Vel) after exposures to 27°C, 20°C, 15°C and 10°C

	t_{ecc} (ms)	t_{conc} (ms)	t_c (ms)	$F_{push-off}$ (N)	Vel ($m \cdot s^{-1}$)
27°C	104±9	103±7	206±9	2429±111	2.57±0.09
20°C	115±5	114±5	229±4*	1756±82***	2.32±0.06
15°C	131±8*	131±7*	262±13**	1480±56***	2.29±0.08*
10°C	133±6*	146±9**	278±13***	1268±100***	2.10±0.06***

The significance in relation to 27°C is denoted by * = $p<0.05$, ** = $p<0.01$ and *** = $p<0.001$.

TABLE 7. Changes in performance parameters after the exposure to 10°C and after each walking bout. 27°C are reference values

	t_{ecc} (ms)	t_{conc} (ms)	t_c (ms)	$F_{push-off}$ (N)	Vel ($m \cdot s^{-1}$)
27°C	104±9	103±7	206±9	2429±111	2.57±0.09
10°C	133±6	146±9	277±13	1268±100	2.10±0.06
1 walk	136±11	141±10	277±17	1612±105	2.19±0.06
2 walk	117±8 a	120±8 [a]	237±12 [a]	1886±144*	2.30±0.08 [a]
3 walk	116±8* [a]	110±8 [a]	226±13 [a]	2104±94* [a]	2.37±0.1* [a]
4 walk	107±7* [a]	107±6* [a]	214±7* [a]	2305±133* [a]	2.52±0.1* [a]
5 walk	110±7* [a]	90±5 * [a]	203±5* [a]	2520±38 * [a]	2.35±0.06* [a]

The significance in relation to 10°C is denoted by * = $p<0.05$. Letter [a] indicates the points where the data does not significantly differ from the 27°C thermoneutral values.

When combining the results concerning muscle temperature and flight time of the drop jump from study IV and V a correlation between these two factors can be seen (Fig 6), showing that muscular performance is mainly dependent on muscle temperature.

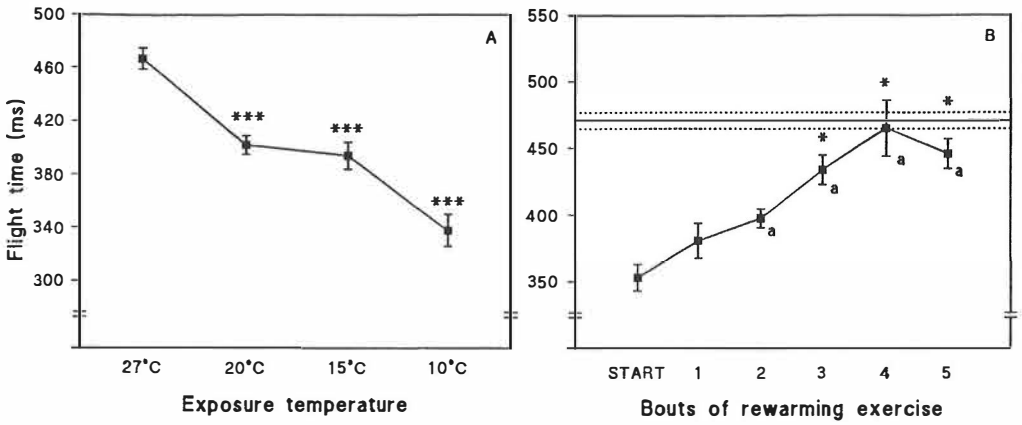

FIGURE 5. (A) The flight time of the drop jump after the exposures to 27°C, 20°C, 15°C and 10°C. The values are mean±SE of eight subjects. *** = $p<0.001$. (B) The flight time after the exposure to 10°C (START) and after the following five walking bouts (1 - 5). The values are mean±SE of eight subjects, except at 4, n = 7 and at 5, n = 3. The statistical difference in relation to START is denoted by * = $p<0.05$. The solid and dotted lines indicate the thermoneutral value (mean±SE). Letter [a] indicates the points where the data does not significantly differ from the 27°C thermoneutral reference value.

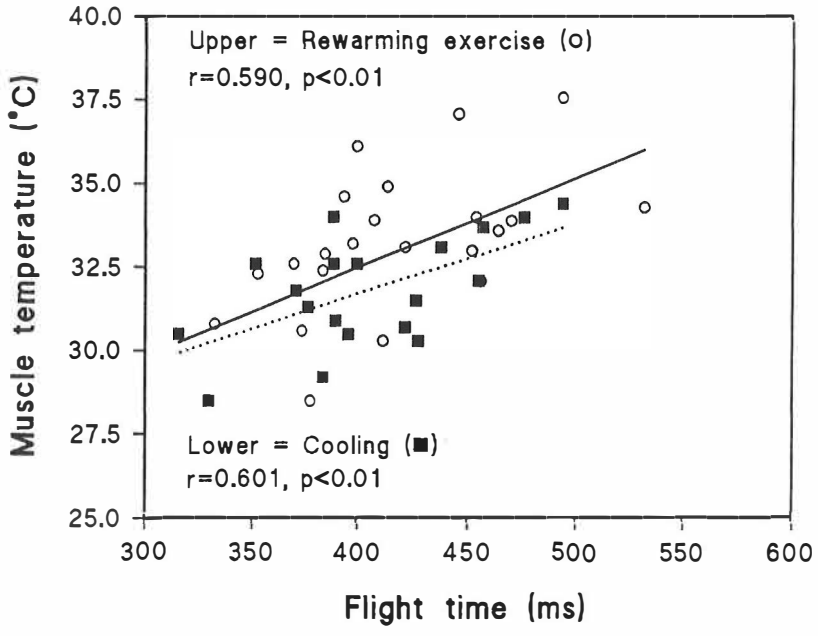

FIGURE 6. Correlation between muscle temperature and flight time of the drop jump.

5.4 Neuromuscular function

5.4.1 EMG-activity

In studies II - VI alike changes in the EMG-activity of the working agonist-antagonist muscles were observed during a stretch-shortening cycle. As an example of these changes Figure 7 shows the EMG-activity of TS and TA muscles of one subject during different phases of SSC.

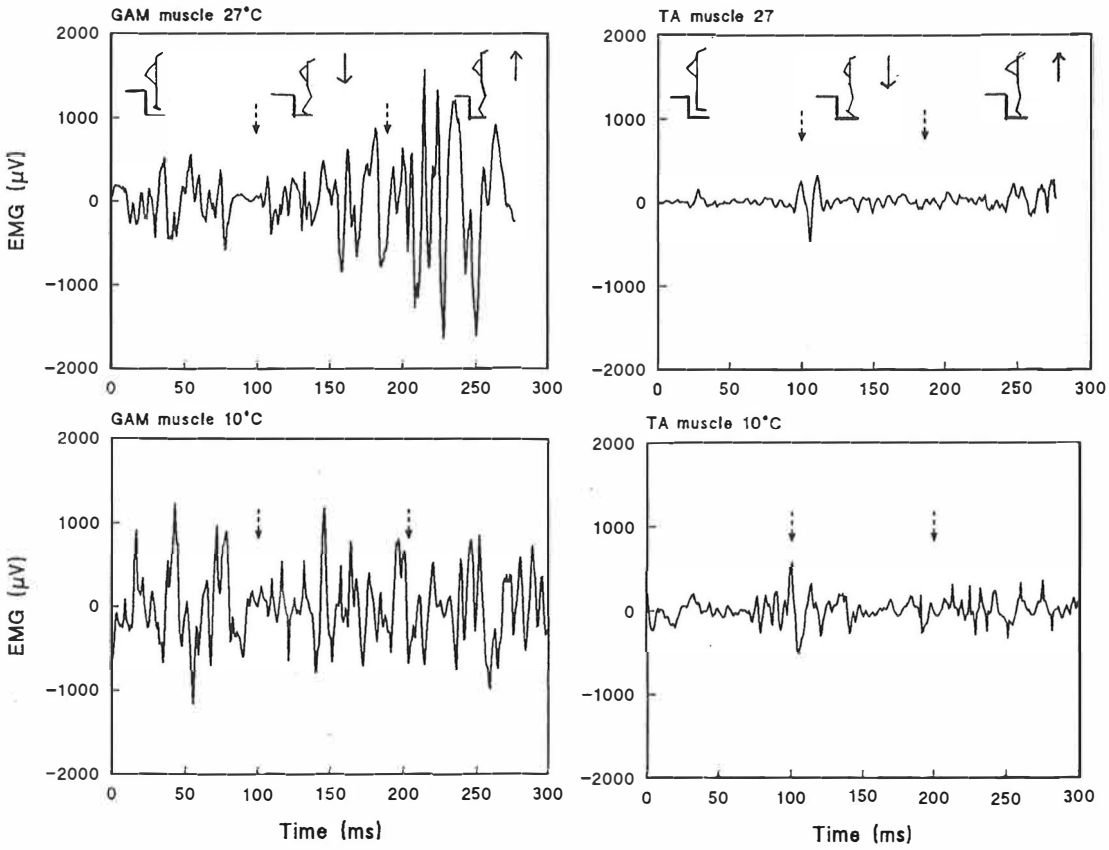

FIGURE 7. An example of the changes occurring in one subjects´ EMG-activity of the agonist (GAM muscle) and antagonist (TA muscle) muscle pair of the lower leg during different phases of stretch-shortening cycle. Dashed arrows indicate the turning points between preactivity and stretch phases and between stretch and shortening phases.

During the preactivity and stretch phases the aEMG of TS muscle increased with decreasing ambient exposure temperature, but along with rewarming exercise the activity returned to a thermoneutral level (Fig 8).

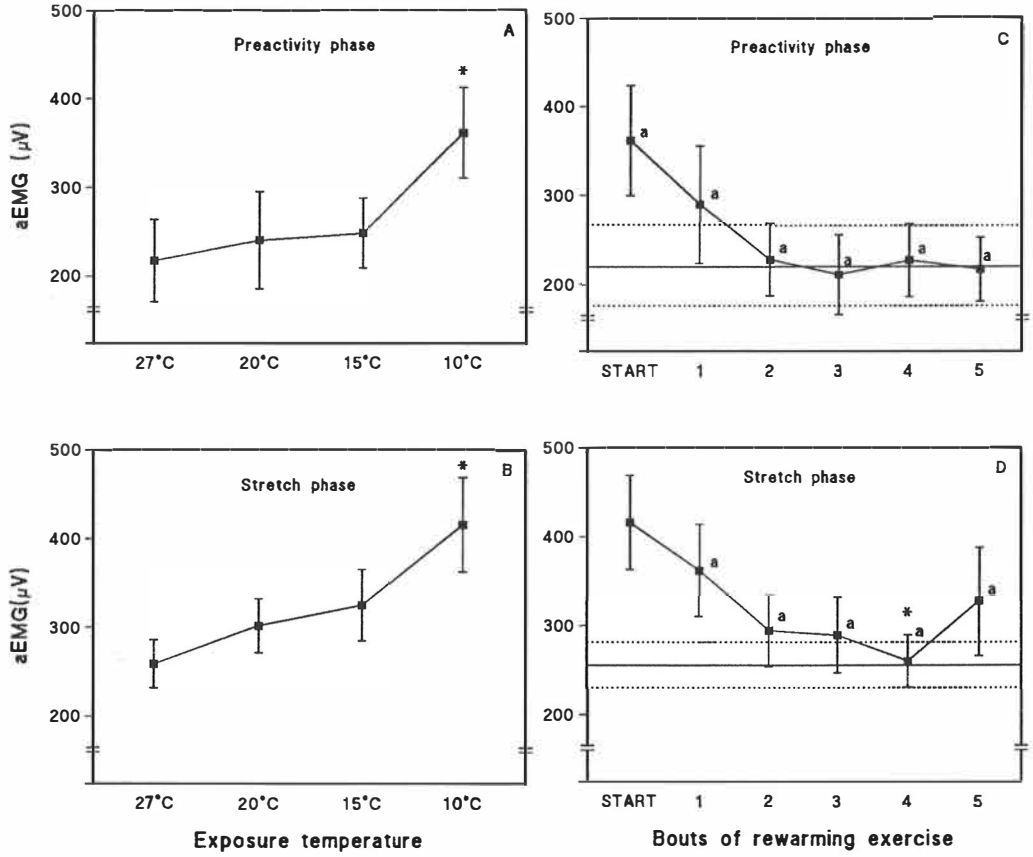

FIGURE 8. The aEMG of TS muscle during preactivity (A) and stretch (B) phases after exposures to 27°C, 20°C, 15°C and 10°C. The values are mean±SE of eight subjects. * = $p<0.05$ (**left panel**). The aEMG of TS muscle during preactivity (C) and stretch (D) phases after the exposure to 10°C and following five walking bouts (**right panel**). Explanations are as in Fig 5 B.

During the shortening phase, on the contrary, the aEMG of TS muscle decreased when ambient exposure temperature was lowered and returned along with rewarming exercise back to a thermoneutral level. The frequency component of EMG (MPF) reacted similarly during the shortening phase as did aEMG (Fig 9).

In contrast to the aEMG-activity of TS muscle the aEMG-activity of TA muscle increased during the shortening phase with decreasing ambient exposure temperature and once again along with rewarming exercise returned to a thermoneutral level (Fig 10).

In study VI keeping the lower legs warm resulted in similar EMG responses as in thermoneutrality. For example, in relation to 27°C the MPF decreased after the exposure to 10°C from 129±6 to 100±7 Hz ($p<0.05$). After the exposure to $10°C_{lw}$ MPF was 133±6 Hz.

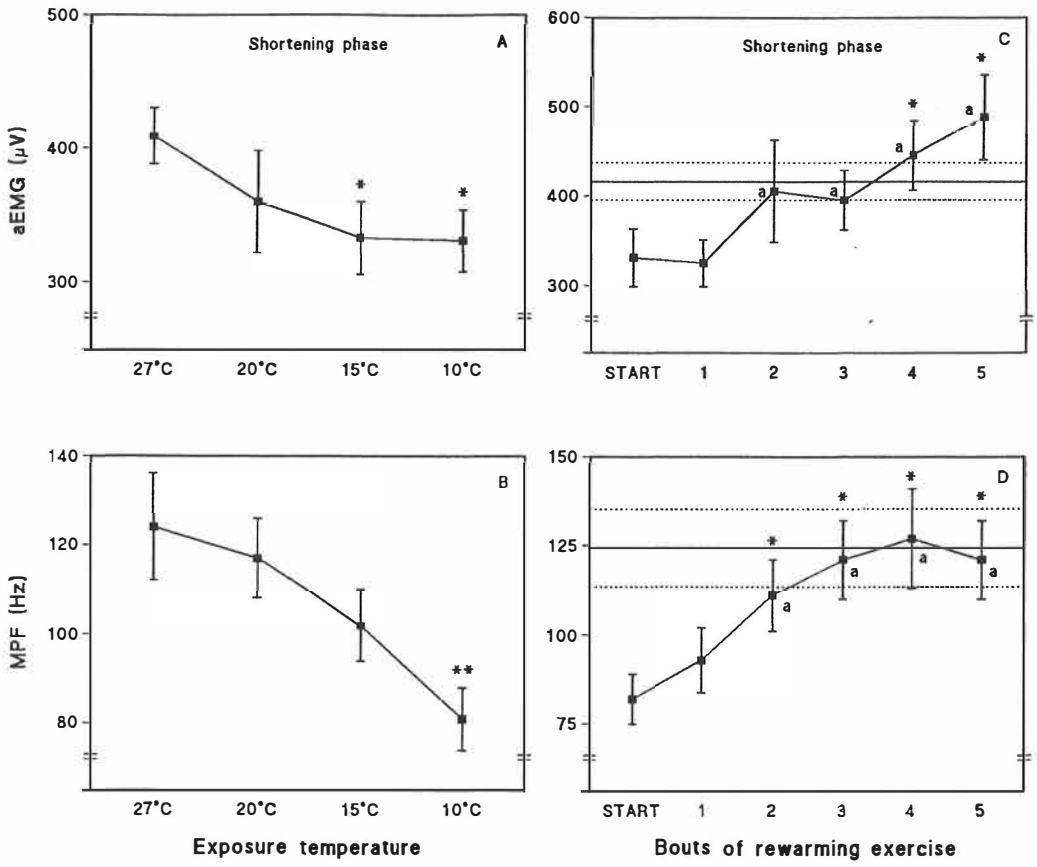

FIGURE 9. The aEMG (A) and MPF (B) of TS muscle during the shortening phase after the exposures to 27°C, 20°C, 15°C and 10°C. The values are mean±SE of eight subjects. * = $p<0.05$, ** = $p<0.01$ and *** = $p<0.001$. (**left panel**).The aEMG (C) and MPF (D) of TS muscle during shortening phase after the exposure to 10°C and following five walking bouts (**right panel**). Explanations are as in Fig 5 B.

The decreasing effect of the exposure to 10°C on the aEMG-activity of the three components of TS muscle were found to be similar during various phases of stretch-shortening cycle. However, there was a tendency that the aEMG-activity of GAM and GAL muscles were slightly more affected than the aEMG-activity of SOL muscle as illustrated in Table 8.

TABLE 8. The cooling (10°C) induced maximum percentage (%) increase (+) or decrease (-) in aEMG-activity of the three components of TS muscle in relation to corresponding aEMG-activity at 27°C during various phases of stretch-shortening cycle

	Preactivity	Stretch	Shortening
GAM muscle	+46	+31	-33
GAL muscle	+44	+31	-6
SOL muscle	+22	+7	-18

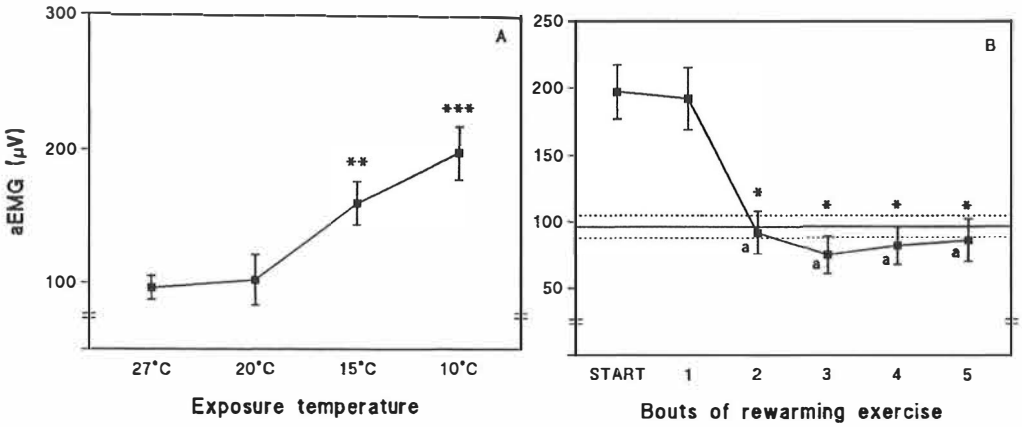

FIGURE 10. (A) The aEMG of TA muscle during the shortening phase after the exposures to 27°C, 20°C, 15°C and 10°C. The values are mean±SE of eight subjects. * = $p<0.05$, ** = $p<0.01$ and *** = $p<0.001$. (B) The aEMG of TA muscle during shortening phase after the exposure to 10°C and following five walking bouts. Explanations are as in Fig 5 B.

In addition, during the shortening phase after the exposure to 10°C the MPF of SOL muscle decreased 30±2 Hz, whereas the MPF of the two heads of gastrocnemius muscle decreased more; 46±4 (GAM, $p<0.001$ in relation to SOL muscle) and 42±8 (GAL, $p<0.01$ in relation to SOL muscle).

The time to reach the maximum aEMG-activity level of TB muscle during ball throwing at thermoneutral situation varied from 22 to 42 ms depending on the ball weight. After cooling the values were greater ($p<0.05$ - $p<0.001$) ranging from 37 to 61 ms. The duration of muscle action tended to be longer after cooling.

5.5.2 T- and H-reflexes

In study VI the thermal parameters were similarly altered due to standard cooling as in other studies. The use of electrical pillows around the lower legs ($10°C_{lw}$, downward from the knee) were able to keep the calf muscle and local skin temperatures (calf and shin) at a thermoneutral level (Table 9).

TABLE 9. Calf (T_c), shin (T_s) and GAM muscle temperature (T_m) at the end of exposures to 27°C, and 10°C and $10°C_{lw}$ (legs warm)

	T_c (°C)	T_s (°C)	T_m (°C)
27°C	31.4±0.1	31.7±0.2	32.4±0.6
10°C	23.7±0.3***	24.0±0.4***	28.8.0±0.7**
$10°C_{lw}$	32.5±0.5	32.6±0.5	32.3±0.5

The significance in relation to 27°C is denoted by ** = $p<0.01$ and *** = $p<0.001$.

The exposure to 10°C increased T-reflex duration and inter-spike-interval time but decreased the maximum amplitude significantly in TS muscle. Reflex latency remained unchanged. The exposure to $10°C_{lw}$ did not significantly alter the reflex responses in relation to 27°C (Table 10). Figure 11 gives an example of the cooling-induced changes in T-reflex responses in one subject. The results of the reflex responses did not deviate from each other in different parts of TS muscle complex thus, cooling affected similarly each of these muscles.

TABLE 10. The effect of different thermal exposures on tendon reflex latency (RL), reflex EMG duration (RD), maximum amplitude (T_{amp}) and inter spike interval (ISI) of TS muscle

	RL (ms)	RD (ms)	T_{amp} (μV)	ISI (ms)
27°C	37±3	22±2	580±51	6.4±0.5
10°C	38±2	34±5*	423±69*	8.4±0.3**
$10°C_{LW}$	38±2	26±3	544±59	6.7±1.1

The significance in relation to 27°C is denoted by * = $p<0.05$ and ** = $p<0.01$.

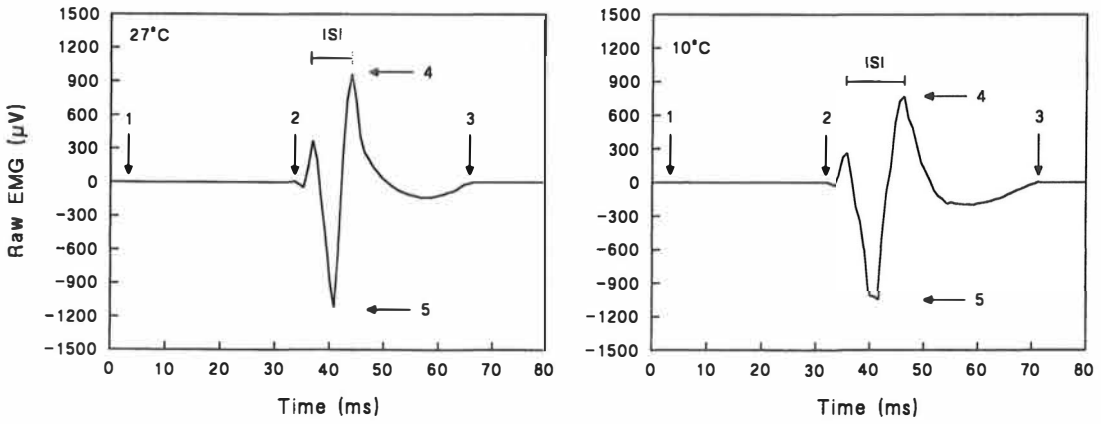

FIGURE 11. An example of the changes in tendon reflex responses between 27°C and 10°C in one subject. The tendon reflex latency is indicated by arrows 1 and 2, reflex duration by arrows 2 and 3 and maximum amplitude by arrows 4 and 5. The duration of ISI time is indicated by a bar.

The exposure to 10°C increased H- and M-amplitudes and H_{max}/M_{max}-ratio significantly but the exposure to $10°C_{lw}$ returned these changes back to thermoneutral level. The exposure to 10°C and $10°C_{lw}$ prolonged the H-reflex latency (Table 11). An example of the M-response and H-reflex at 27°C and 10°C in one subject is given in Figure 12.

TABLE 11. The effect of different thermal exposures on H-reflex latency (H_{lat}), H and M-amplitudes (H_{amp} and M_{amp}) and on the H_{max}/M_{max}-ratio

	H_{lat} (ms)	M_{amp} (mV)	H_{amp} (mV)	H_{max}/M_{max} (%)
27°C	29.3±0.4	26.5±2.2	11.6±1.3	47±7
10°C	30.3±0.4***	31.3±2.4*	15.2±1.9**	56±8*
$10°C_{lw}$	29.6±0.4*	24.2±2.1	10.2±0.9	47±6

The values are mean±SE of 5 subjects. The significance in relation to 27°C is denoted by * = $p<0.05$, ** = $p<0.01$ and *** = $p<0.001$.

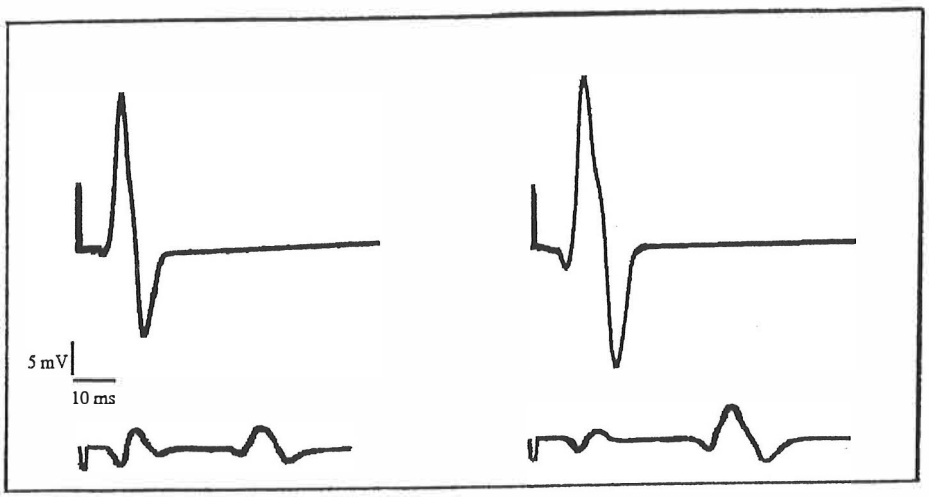

FIGURE 12. An example of the changes in M-response (upper figures) and H-reflex (lower figures) at 27°C (left) and 10°C (right) in one subject.

6 DISCUSSION

The main findings of this study were as follows:
1. During the preactivity and stretch phase muscle cooling increased the EMG-activity of TS muscle.
2. During the shortening phase the EMG-activity of TS muscle decreased whereas the activity of TA muscle increased i.e. the co-activation increased.
3. The above mentioned changes may be related to changes in T- and H-reflexes.
4. Very low level of muscle cooling was sufficient to significantly decrease muscular performance and its components and relatively little rewarming exercise was able to return the decreased performance back to thermoneutral level.
5. Very fast SSC exercise which prominently utilises elastic properties of the working muscles was most susceptible for cooling.

6.1. Neuromuscular function

Muscle cooling significantly altered the function of agonist-antagonist muscle pairs. These changes may, at least partly, be related to changes in T- and H-reflexes.

The results of this study showed that during preactivity and stretch phase the aEMG-activity of the agonist muscle (TS muscle) increased whereas during the shortening phase it decreased. Furthermore, the activity of the antagonist during shortening phase increased after muscle cooling. This cooling-induced "braking effect" of agonist-antagonist muscle pairs (increased co-activation) has not been reported earlier as a mechanism, which decreases muscular performance. Bergh and Ekblom (1979a) did not find any effect of muscle cooling on EMG-activity pattern of the agonist (*vastus lateralis* and *semitendinosus*) or of the

semitendinosus) or of the antagonist (*biceps femoris*) muscles of the thigh. However, in their study only one subject for the EMG measurements was used. Bawa et al. (1987) found that during light muscle work co-contraction of the antagonist muscle (*triceps brachii*) together with the agonist muscle (*biceps brachii*) appeared in cooled and shivering subjects but not in thermoneutral subjects. Thus, it may be assumed that the co-activation was due to shivering.

The tendon reflex responses have been shown to be weakened due to cooling (Petajan and Watts 1962; Bell and Lehmann 1987) and it has been attributed to decreased spindle activity (Eldred et al. 1960; Knutsson and Mattsson 1969; Chapman et al. 1979). The results of this study are in agreement with studies cited above showing decreased reflex amplitude referring to decreased spindle sensitivity. The decreased spindle sensitivity should cause disfacilitation of the agonist muscle (TS muscle) and disinhibition of the antagonist muscle (TA muscle) during exercise. This was actually seen during the shortening phase of the stretch-shortening cycle as decreased aEMG-activity of the TS muscle (agonist) and increased aEMG-activity of the TA muscle (antagonist). Therefore, muscle cooling-induced changes in the tendon reflex responses via decrement of muscle spindle activity seem to be responsible, at least partly, of the EMG-activity changes during the shortening phase. This assumption is further supported by the finding that the above mentioned changes vanished when the lower legs were kept warm.

The results showed also a significant decrease in nerve and muscle conduction velocity (H_{lat}) at 10°C. Decrement in conduction velocity quite likely increased the duration of muscle contraction (reported also elsewhere, e.g. Bigland-Ritchie et al. 1992) and the time to reach maximum aEMG-activity (II, III). Therefore, it can be argued that also due to these factors the instantaneous power of muscle contraction was less after cooling of the muscles.

When the lower legs were kept warm the MPF remained at the thermoneutral level. The inter spike interval (ISI) time during the tendon reflex behaved similarly as MPF: it increased after the exposure to 10°C and returned back to thermoneutral level when the lower legs were kept warm. It may be assumed that decreased nerve conduction velocity may also result in an increased ISI time reflecting slower rate of discharge or shift towards the usage of slower fibers. These changes, during muscle contraction, may consequently be seen as a shift of MPF towards lower frequencies. However, the fact that nerve conduction velocity did not recover totally when the lower legs were kept warm but ISI time and MPF did, may obscure this conclusion.

During preactivity and stretch phases an elevated aEMG-activity of TS muscle was observed after the exposure to 10°C. The exposure to 10°C increases thermoregulatory muscle tonus. It has been shown previously that after a 30 min exposure to 10°C thermoregulatory muscle tonus increases so that the resting aEMG-activity in peripheral muscles varies from 4 to 9 μV (Meigal et al. 1998). Since the increase in aEMG-activity during preactivity and stretch phases was considerably more than 4 - 9 μV (between 95 - 115 μV) the increased thermoregulatory muscle tonus can not explain these results.

For evaluation of the excitability of the motoneuron pool using H-reflex and M-response H_{max}/M_{max}-ratio has been applied (Funase et al. 1996). In this study we found an elevated H_{max}/M_{max}-ratio after muscle cooling indicating an increased level of excitability of the motoneuron pool. The elevated H_{max}/M_{max}-ratio disappeared when the lower legs were kept warm. At the same time we also found an increased aEMG-activity of the TS muscle during preactivity and stretch phases, which also disappeared when the lower legs were kept warm. This synchronous appearing and disappearing would imply that the increased level of excitability of the motoneuron pool is connected to the increased level of aEMG of TS muscle during preactivity and stretch phases.

Most likely, the increased excitability of the motoneuron pool is due to cooling. Whether it is caused by increased sensory input from the cutaneous afferents, by increased supraspinal drive from the central nervous system or their combination, remains uncertain. However, due to the lack of simultaneous increase in the EMG-activity of TA muscle it may be argued that this phenomenon is more centrally regulated. Since the sensory input from the antagonist skin area should be the same as from the agonist skin area, it is difficult to find a mechanism in the spinal level, without the interference of CNS, which could explain this selective increase in EMG-activity of only TS muscle.

The results of this study and the study of Sargeant (1987) show that cooling of the muscles decreases performance in fast exercises more efficiently than in slower ones. Muscle cooling also deteriorates the function of fast twitch fibers more efficiently (Faulkner et al 1991). The aEMG-activity and MPF of the GAM and GAL muscles (fast muscles) in this study were more decreased than SOL muscle (slow muscle) also implying that the function of fast twitch fibers are more easily hampered by cooling than slower ones.

6.1.1 Methodological considerations of EMG

The cooling of skin and muscles as well as electrodes may increase the possibility for methodological errors while using surface EMG. For example, the impedance of the skin or skin - electrode junction may change and cooled tissues may act as a low pass filter (Lindström et al. 1970). Decreased nerve conduction velocity and increased duration of action potentials (Buchthal et al. 1954) may also result in an increased amplitude of surface EMG (DeLuca 1985). However, since the EMG-activity of the agonist and antagonist muscles during the same contraction changed in an opposite manner the observed changes can not be explained by the methodological effects of cooling on EMG.

The use of mean power frequency as a parameter to describe changes in the frequency component of EMG during dynamic exercise has been criticised because of the possible unstationarity of the signal, which may be caused e.g. by the movements of the muscles during exercise. Signal is said to be stationary if there is no systematic trendlike change in the mean or in the variance of the signal (Chatfield 1989). To ensure that there were no systematic changes 20 ms samples of EMG were taken (mean) in the beginning and in the end of "steady state" activity during the shortening phase of the muscle contraction from every

subject during a drop jump in the sixth study. The mean activity of all subjects in the beginning and in the end varied less than 10 %, reflecting that the signal was rather stable. In addition, the short duration of the analysed period (stretch or shortening) probably sets restrictions that there simply is not enough time for trendlike changes in the signal level.

The test-retest reproducibility in EMG measurements expressed as reliability coefficients has been reported to be rather high: r = .94 for aEMG in jumping (Bosco 1982) and in isometric and dynamic actions with controlled movement velocity r = .88 (Komi and Buskirk 1970). To test the reproducibility of the EMG-measurements in cold two subjects were exposed to 10°C five times on consecutive days. They performed a drop jump from a 40 cm bench. Expressed as coefficient of variation of mean maximum EMG-activity (SD divided by mean) obtained from gastrocnemius medialis muscle the variation was less than 7 %.

6.2 Temperature dependence of muscular performance

A dose - dependent relationship between the degree of muscle cooling and decrease in muscular performance was found. Also, a similar dose - dependent relationship was found between rewarming exercise (muscle warming) and increase in muscular performance. In addition, all performance parameters as well as the changes in EMG-activity of the lower leg muscles changed in a dose - dependent manner. Surprisingly low level of muscle cooling was found to be sufficient to cause a significant decrease in muscular performance.

In most previous studies the decrease in rectal, muscle or mean skin temperature has been far more severe than in this study (Fischer and Solomon 1965; Bergh and Ekblom 1979b; Giesbrecht and Bristow 1992), especially when considering the exposure to 20°C. Still, a very marked decrease (17 % · $°C^{-1}$ in T_m) in muscular performance was found in this study already after the exposure to 20°C. Since we were not able to precisely identify where the threshold for decrease in performance is located (if one exists) two additional subjects were exposed also to 25°C for 60 min. The muscular performance of these two subjects did not, however, alter in comparison to 27°C and therefore (even though there were only two subjects) it may be speculated that the possible threshold is situated somewhere between 25°C and 20°C. Expressed as mean skin temperature values the threshold should be approximately between 28.1°C (mean value from 20°C) and 31.5°C (the mean value of the two subjects at 25°C). However, this temperature range for the threshold is valid only when similar exposures as in this study are being used.

Rewarming exercise was able to recover decreased performance back to thermoneutral level after three walking bouts in one subject, after four bouts in four subjects and after five bouts in three subjects. When considering the intensity and duration of the walking bouts, surprisingly little rewarming exercise is needed to enhance and finally to recover muscular performance. After cooling the rewarming exercise was able to recover decreased muscle temperature back

to thermoneutral level but local skin temperatures of shin and calf were only slightly elevated. In addition to the thermal effects of rewarming exercise (i.e. increasing temperature of the tissues thus enhancing e.g. nerve conduction velocity) it may also induce changes which are not directly related to temperature. For example, exercise increases demand for blood flow in the muscles which then can override the cold-induced vasoconstriction (Toner and McArdle 1988).

Like all performance parameters the changes in EMG-activity also seem to follow the ambient exposure temperature and rewarming exercise in a dose - dependent manner. This similarity between changes in performance parameters and EMG-activity along with decreasing ambient exposure temperatures and rewarming exercise strongly suggest that there exists a causal relationship between performance decrement and changes in muscular co-activation.

6.3 Muscular performance

In this study slow (throwing), fast (rebound jumping) and very fast (drop jump) strech - shortening cycle exercise types were used. The results show that faster exercises i.e. faster muscle contraction velocities (drop jump vs. other exercise types, throwing light vs. heavy balls) are more readily deteriorated by muscle cooling than slower exercises. This may imply that the function of fast twitch fibers are more susceptible to cooling. This is in part supported by the finding that MPF was shifted towards lower frequencies after muscle cooling (III). The observed shift in the frequency component functioned in a dose - dependent manner in relation to cooling (IV). Similar results concerning the effect of cooling on different contraction velocities have been obtained by Sargeant (1987), who found that muscular performance was more deteriorated when using fast (144 rpm) than slow pedalling rates (54 rpm).

The decrease in muscular performance was greatest in the very fast exercise in relation to fast and slow exercises (27 % vs. 11 % and 7 % or 6.8 vs. 4.8 % and 2.8 % $\cdot$ $^{\circ}C^{-1}$ in T_m.). This would seem to imply that the more efficiently the elastic properties of the muscles are being utilised the more muscular performance is decreased. This assumption, however, contradicts the results obtained by Asmussen et al. (1976) who found that cooling of the muscles may increase their capacity to utilise elastic properties during squatting jump with a drop from 40 cm bench. The possible difference may be due to different exercise types used, the squatting jump possibly utilising the elasticity to a lesser degree than drop jump.

6.3.1 The components of muscular performance

All the measurable components of muscular performance; power, force production and velocity decreased due to muscle cooling. Due to the decreased capacity to produce force the duration of the contact in 60 s jumping and shortening phase during drop jump exercise were prolonged. The increased dura-

tion of the stretch phase after cooling may be caused by increased stiffness of the calf muscles. When the same amount of external force is applied to the cooled muscles, longer time is needed to stretch them a given distance.

Power

During the recovery period after rebound jumping in cooled subjects oxygen consumption was less, blood lactate accumulation was less and heart rate was lower. These results indicate that the changes in overall anaerobic metabolism and biochemistry of muscle contraction are the primary reasons for the decreased jumping power. Changes in neuromuscular function may be of lesser importance in this kind of exercise.

In addition, chemical reactions are slower when tissues are cooled. This would simply imply that less energy can be produced and utilised and thus less work due to cooling can be done. This may be supported by the finding that the efficiency (relationship between mechanical work and oxygen consumption) of muscle work during rebound jumping exercise was found to be the same in thermoneutrality and after muscle cooling (unpublished result). However, there are also results which show that the efficiency of muscular work may decrease when exercising at cold ambient temperatures (Oksa et al. 1993).

Force and velocity

The changes observed in force - velocity relation in this study are in agreement with previous findings. To achieve the same velocity with cooled muscles as with thermoneutral ones requires more force: the force - velocity relation is shifted to the left (Bergh and Ekblom 1979a).

Muscular performance in throwing and drop jump as well as force production and velocity decreased due to muscle cooling. Due to the nature of these exercises, maximal single contraction, muscle cooling induced changes in neuromuscular function found in this study have a major role in causing these changes. There are, however, also several other mechanisms which may decrease force production and velocity of muscle contraction or movements: increased stiffness of the muscles and joints (Hunter et al. 1952), decreased ATP-hydrolysis (Edwards et al. 1971; Ferretti 1992), slowed Ca^{2+} release and uptake from the sarcoplasmic reticulum (Kössler et al. 1987) and decreased calcium sensitivity of the actomyosin (Hartshorne et al. 1972; Fuchs et al 1974; Wakabayashi et al. 1988; Sweitzer and Moss 1990). These changes may lead to an impaired cross-bridge formation and breakdown or decreased force per cross-bridge (Sweitzer and Moss 1990) thus decreasing force production and velocity.

7 MAIN FINDINGS AND CONCLUSIONS

The main findings and conclusions of the present study can be summarised as follows:

1. During preactivity and stretch phases of stretch-shortening cycle the aEMG-activity of the TS muscle (agonist) increased due to cooling. This may be due to increased excitability of the motoneuron pool caused by increased sensory input from the cutaneous afferents, by increased supraspinal drive from the central nervous system or their combination. Increased activity slows the velocity of the downward movement during ground contact after cooling.

2. During the shortening phase of stretch-shortening cycle the aEMG-activity of the TS muscle (agonist) decreased. Furthermore, the aEMG-activity of the TA muscle (antagonist) simultaneously increased due to cooling. This "braking effect" of agonist-antagonist muscle pair is probably caused by decreased sensitivity of muscle spindles as detected by increased T-reflex amplitude and inter spike interval time of reflex EMG. The "braking" effect of the agonist-antagonist muscles during shortening phase are related to decreased force production and muscular performance.

3. Unexpectedly low level of cooling (60 min at 20°C, average mean skin temperature 28.1±0.6°C) was sufficient to significantly decrease muscular performance and its components. Such a low level of cooling can very easily occur in work, sports or leisure time activities in cold and cool environments much more often than expected.

4. Decreased muscular performance can be recovered with rewarming exercise in cold. Rewarming exercise with rather low intensity and short duration, 5 min bouts of walking with the speed of 5 km · h^{-1}, from three to five times was sufficient to recover muscular performance. The significance of rewarming exercise should be emphasised.

5. The decrement or recovery of muscular performance followed the changes in muscle temperature. Therefore, muscle temperature may be considered as predominant factor causing decrease or increase in muscular performance.

6. Muscular performance decreased 7 %, 11 % and 27 % in slow, fast and very fast stretch-shortening cycle exercises, respectively. Therefore, an exercise type which is very fast and prominently utilises the elastic properties of the working muscles seems to be most susceptible for cooling.

8 TIIVISTELMÄ

Kylmän aiheuttamia laadullisia ja määrällisiä muutoksia ihmisen lihaksiston toimintakykyyn ei tunneta riittävästi. Lisääntyvän jäähtymisen ja suorituskyvyn heikentymisen sekä lämmittävän lihastyön ja suorituskyvyn parantumisen välistä annos - vaste suhdetta ei tunneta riittävästi. Tässä työssä tutkittiin vakiokylmäaltistuksen (10°C, 60 min) ja asteittain alenevan lämpötilan (20°C, 15°C ja 10°C) sekä vakioaltistuksen jälkeisen lämmittävän lihastyön vaikutusta suorituskykyyn, sen eri komponentteihin ja lihassupistukseen (EMG:n avulla).

Vakiokylmäaltistuksen aikana koehenkilöt istuivat 10°C:ssa 60 min pukeutuneina shortseihin ja lenkkitossuihin. Termoneutraalina vertailuarvona pidettiin samanlaista altistusta 27°C:een. Tutkittaessa eri tasoisten kylmäaltistusten vaikutusta, koehenkilöt altistettiin eri päivinä 27°C:een, 20°C:een, 15°C:een ja 10°C:een 60 minuutin ajan. 10°C:een altistuksen jälkeen koehenkilöt saivat ko. altistuslämpötilassa tehdä lämmittävää lihastyötä, kävelyä juoksumatolla nopeudella 5 km $\cdot$ h^{-1}.

Tutkimuksen tulokset osoittavat suorituskyvyn sekä sen eri komponenttien heikentymisen riippuvan kylmäaltistuksen tasosta. Mitä voimakkaampi altistus sitä alemmaksi laskee työskentelevien lihasten lämpötila ja sitä enemmän heikkenee suorituskyky ja sen komponentit. Heikentynyt suorituskyky voitiin palauttaa varsin vähäisellä määrällä matalaintensiteettistä lämmittävää lihastyötä (kävely nopeudella 5 km $\cdot$ h^{-1}). Pudotushyppy, työtyyppi jossa oleellisesti hyödynnetään lihaksiston elastisuusominaisuuksia, havaittiin olevan kaikkein herkin kylmän vaikutuksille.

Kylmä aiheutti lihassupistuksessa muutoksen, joka oli suorituskyvyn kannalta epäedullinen. Verrattuna termoneutraaliin lihassupistuksen EMG-aktiivisuuteen agonistilihaksen EMG-aktiivisuus lisääntyi jäähtymisen seurauksena esiaktiivisuus- ja lihaksen venymisvaiheessa (eksentrinen lihassupistus). Sensijaan lyhenemisvaiheessa (konsentrinen lihassupistus),

jolloin agonistilihaksen pitäisi työskennellä mahdollisimman tehokkaasti, sen EMG-aktiivisuus pieneni, jonka lisäksi vielä työtä vastustavan antagonistilihaksen aktiivisuus lisääntyi verrattuna termoneutraaliin. Nämä ns. "jarrutus" muutokset käyttäytyivät myös annos - vaste periaatteella: Mitä voimakkaampi kylmäaltistus (mitä alempi lihaslämpötila) sitä suurempi muutos kyseisissä EMG-aktiivisuuksissa. Niiden muutoksien perusteella, joita havaittiin H- ja T-reflekseissä, voitiin päätellä EMG-muutosten johtuvan, ainakin osittain, kylmän aiheuttamasta lisääntyneestä alfamotoneuronien ärsyyntyvyydestä ja alentuneesta lihassukkuloiden sensitiivisyydestä.

Hyvin vähäinen kylmäaltistus, 60 minuuttia 20°C:ssa, oli riittävä heikentämään suorituskykyä merkitsevästi. Tämän tasoinen jäähtyminen, keskimääräisen iholämpötilan putoaminen n. 4°C asteella, voi tapahtua hyvin helposti ulkotyön tai liikunnan tai kylmissä sisätiloissa tehtävän työn aikana.

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ORIGINAL PAPERS

I

Power output, fatigue and recovery in one minute jumping test in cooled and warmed men

by

Hannu Rintamäki, Juha Oksa, Sirkka Rissanen, Juhani Hassi and Heikki Rusko

(submitted)

II

Cooling-induced changes in muscular performance and EMG activity of agonist and antagonist muscles

by

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Aviation Space and Environmental Medicine 66, 26-31, 1995

Cooling-Induced Changes in Muscular Performance and EMG Activity of Agonist and Antagonist Muscles

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OKSA J, RINTAMÄKI H, MÄKINEN T, HASSI J, RUSKO H. *Cooling-induced changes in muscular performance and EMG activity of agonist and antagonist muscles.* Aviat. Space Environ. Med. 1995; 66: 26–31.

The effect of whole body cooling on the muscular performance and electromyographic (EMG) activity of agonist and antagonist muscles during dynamic exercise was studied. Eleven slightly clothed male subjects were exposed to ambient temperatures of 27°C and 10°C for 60 min. After the exposures the subjects performed an overhead ball throwing test. Five balls, weighing from 0.3 kg to 3.0 kg were thrown and the velocity of the balls was measured. The EMG activity of two agonist-antagonist muscle pairs (m. *triceps brachii* — m. *deltoideus* and m. *rectus abdominis* — m. *erector spinae*) were measured during throwing. Cooling decreased mean skin temperature by 6.3 ± 0.5°C (mean ± SE). The temperature of m. *triceps brachii* decreased by 4.0 ± 1.6°C and 1.8 ± 0.6°C from the depth of 20 and 30 mm underneath the skin surface, respectively. The corresponding values of m. deltoideus were 5.1 ± 0.4°C and 3.2 ± 0.8°C. The cooling-induced decrement in ball velocity varied from 9.4 ± 3.3% (0.3-kg ball) to 5.6 ± 2.8% (3.0-kg ball) ($p < 0.001$–0.01). After cooling, the time to reach the maximal level of integrated electromyographic (IEMG) activity in m. *triceps brachii* (agonist) was increased (30–42%, $p < 0.05$–0.001). Moreover, cooling decreased the mean IEMG activity m. *triceps brachii*, while the activity of m. *deltoideus* (antagonist) was increased. The alteration was significant ($p < 0.05$–0.001) with the three lightest balls. We conclude that cooling decreased muscular performance in dynamic upper body exercise, and the decrement was clearly pronounced with fast contraction velocities. Cooling also slowed the function of agonist muscle and decreased its IEMG activity, but increased the IEMG activity of the antagonist muscle.

COOLING HAS BEEN generally observed to cause a decrement in muscular performance (3,4,12). During short-term dynamic exercise, cooling decreases maximal force production and increases the time to reach the maximal force level (5,7). It also seems that the shortening velocity of the muscle is related to the decrement in force production. Sargeant (15) found that with a fast pedaling rate, the decrement in force production is enhanced, indicating a more pronounced effect of cooling on fast shortening velocities.

Little data are available about the coordination of cooled muscles. Faulkner et al. (9) suggested that decreased power production of agonist muscles and increased power absorption of antagonist muscles could occur after cooling, therefore affecting coordination. Bawa et al. (2) found that the antagonist muscle co-contracted together with the agonist muscle as detected by electromyography (EMG) in subjects who exercised and shivered at the same time, whereas in a thermoneutral situation it did not. However, Bergh and Ekblom (5) reported that during jumping exercise no changes in the relationship of agonist and antagonist EMG activity were observed in relation to changes in muscle temperature.

The majority of the studies concerning the effects of cooling on muscular performance have used cold water immersions to cool a restricted body area (mainly legs and lower arms). Consecutively, the exercise has focused on the same area, which means that only a relatively small amount of musculature has been used. Knowledge of the effects of whole body cooling on exercise (corresponding to outdoor activities in cold environments), where a large proportion of body muscle mass is involved, is scarce. Hence, the aim of this study was to evaluate: 1) the effects of whole body cooling on muscular performance using different contraction velocities; and 2) possible alterations in the EMG activity of two contracting agonist-antagonist muscle pairs during exercise.

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This manuscript was received for review in January 1994. It was revised and accepted for publication in March 1994.

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METHODS

Subjects

To ensure the homogeneity of the group of subjects participating in the study, only male subjects were used. Eleven sedentary and healthy men volunteered as test subjects. Their mean (± SD) age was 24.4 years, height 174 ± 7 cm, weight 70 ± 7 kg and body fat 12 ± 3%. Body fat was estimated according to skinfold thickness measurements (8). Before the tests, subjects were medically examined, the experimental protocol was explained, and their written consent was obtained. The experimental protocol was approved by the Ethics Committee of the Institute of Occupational Health.

Thermal Exposures and Temperature Measurements

Before the tests, the subjects sat motionless for 60 min in a climatic chamber either at 10°C (cool) or 27°C (control). The subjects were dressed in shorts and jogging shoes. During exposures the skin temperatures (forehead, shoulder, upper arm, lower back, abdomen, thigh, and calf) and rectal temperature (T_r, 10 cm depth) (YSI 400 series thermistors, Yellow Springs Instruments, Yellow Springs, OH) were recorded with 1-min intervals to a data logger (Squirrel 1200 Grant, Birmingham, UK). Mean skin temperature (T_{sk}) was calculated by weighing the 7 local skin temperatures by representative areas (11). On another occasion at the end of similar exposures, resting muscle temperature (T_m) of five subjects was measured from *m. triceps brachii* and anterior part of *m. deltoideus* with a needle electrode (YSI 511, Yellow Springs Instruments, Yellow Springs, OH) from the depth of 20 and 30 mm underneath the skin surface. T_m was measured from the same site as the EMG electrodes were placed.

Ball-Throwing Test

After the exposures the subjects performed a full effort overhead throwing test with both arms, where five balls, weighing 0.3, 0.6, 1.0, 2.0, and 3.0 kg, were thrown. The diameter of the balls ranged from 18 to 25 cm. Before the tests the subjects were allowed to get accustomed to the throwing exercise (they performed the test as many times as they wanted, at least three times) in order to avoid a training effect during the experiments. The flight times of the balls were measured with an accuracy of 0.001 s (Newtest 1500, Oulu, Finland) by using 10 infrared beams, located 10 cm apart from each other in a vertical line and a contact mat functioning with an on/off basis (Newtest, Oulu, Finland). Time measurement was activated when the ball reached the infrared beams and stopped when the ball hit the contact mat attached to the wall 250 cm behind the infrared beams. The throwing site was 100 cm in front of the beams. The test was performed in a standing position, legs slightly apart. The subjects held the ball with both arms and after a signal they lifted the ball above their head and threw it with full effort. During the test a recovery time of approximately 30 s was allowed between consecutive balls. The subjects were allowed to have one trial and the performance was accepted if the throwing site was not overstepped. The velocity (V) of each ball was calculated: $V = D / t$, where D = distance (m) and t = time (s). The test was randomized by exposure and ball weight.

EMG Measurements

Electromyographic signals from the skin above the working muscles were measured by a computerized system with a frequency of 1250 Hz (Mespec 4001, Mega Electronics, Kuopio, Finland). Four muscles, forming two agonist-antagonist muscle pairs, *m. triceps brachii* and anterior part of *m. deltoideus* (upper arm) and *m. rectus abdominis* and *m. erector spinae* (trunk) were measured. In the throwing exercise, *m. triceps brachii* and *m. rectus abdominis* generate force to cause the flexion of the upper arm and trunk (agonist muscles), whereas *m. deltoideus* and *m. erector spinae* are opposing the flexion (antagonist muscles). EMG signals were measured using prejelled bipolar surface electrodes (Medicotest, M-00-S, Olstykke, Denmark), which were placed in the middle part, over the belly of the muscle, except in the muscles of the trunk where the measuring height was approximately at the level of L_3 vertebra. The EMG electrodes were beside the thermistors measuring skin temperature. The spacing between recording contacts was 2 cm. Two ground electrodes were placed above inactive tissue. To ensure the accuracy of replacing the electrodes, their places were carefully marked on the skin with waterproof drawing ink. The markings were clearly visible throughout the experiments. The measured EMG-signal was amplified 2000 times (preamplificator situated 6 cm after the measuring electrodes) and signal band between 20 and 500 Hz was full wave rectified and integrated (IEMG) with a 13-ms time constant. Butterworth filtering was used in the measuring band. The technical qualities of the amplifier were: CMRR > 130 dB, noise < 0.5 μV rms, and input impedance 20^9 ohm.

The EMG measurement started simultaneously from the same signal that was given for the subjects to throw the ball. The EMG measurement was stopped automatically by a switch when the ball left the hands. The time to reach the maximal level of IEMG-activity (TMA) during contraction of *m. triceps brachii* and *m. deltoideus* was analyzed. The contraction was considered to have begun when 20% of the maximum level of activity was reached. Contraction duration (CD, started from 20% of the maximum and ended when the ball left the hands) of *m. triceps brachii* were analyzed. The IEMG analysis of all data covered the same period as the CD of *m. triceps brachii*. This period was also selected to represent the duration of muscular performance in this study. Comparisons of the IEMG results were done intraindividually between the exposures to 27°C and 10°C. The data were further normalized by dividing the individual mean IEMG values measured after the exposure to 10°C by the values measured after the exposure to 27°C.

Statistics

Paired *t*-test was used to test differences between temperatures and significance was accepted at the 0.05

level. The dependance between variables was analyzed by using Pearson's product moment correlation coefficient.

RESULTS

Thermal Exposures

Cooling decreased T_{sk} and T_{m}, whereas T_r was virtually unaffected (Table I). After cooling, the skin temperatures above the working muscles were significantly lower in the arm (21.9 ± 0.8°C, mean of shoulder and back of the upper arm) than in the trunk (25.5 ± 0.4°C, mean of lower back and abdomen, $p < 0.001$).

Ball-Throwing Test

Cooling significantly decreased the velocity of the balls of all weights (Fig. 1A). The average decrement was 0.88 $m \cdot s^{-1}$ (7%).

When the contraction velocity was fast (light balls) the decrement in ball velocity was more pronounced: 9.4% with a 0.3-kg ball in comparison to 5.6% with a 3.0-kg ball (Fig. 1B).

The reproducibility of the ball velocities were examined in thermoneutral conditions by allowing 4 subjects to perform the test 10 times using 3 balls (0.3, 1.0, and 3.0 kg). Coefficient of variation (CV = SD/mean • 100%) varied from 3.1% (0.3-kg ball) to 1.3% (3.0-kg ball).

EMG

The time-related IEMG analysis showed that cooling increased the TMA ($p < 0.001$–0.05) and tended to increase the CD (NS) of *m. triceps brachii* (Fig. 2).

The mean IEMG activity of agonist-antagonist muscle pair of the upper arm was altered after cooling: the agonist (*m. triceps brachii*) activity decreased, whereas the antagonist (*m. deltoideus*) activity increased. An example of individual alteration of one subject is shown in Fig. 3.

Individual levels of IEMG activity showed a large variation. Therefore, to be able to compare the mean IEMG values after exposures to 27°C and 10°C, the individual values were normalized (see *Methods* section). In group means, a similar change in the IEMG-activity of agonist and antagonist muscles were found. This was seen especially in the activity of *m. triceps brachii* and *m. deltoideus,* where significant differences were observed with the three lightest balls (Fig. 4).

A positive correlation was found between the decrease of *m. triceps brachii* IEMG-activity and the decrease in velocity of the lightest ball ($r = 0.748$, $p < 0.05$).

DISCUSSION

The cooling-induced decrement in muscular performance observed in this study is slightly less than in previous studies, where leg or lower arm (1,6,10) exercise was examined. This paper focuses on the cooling-induced time and intensity related changes in EMG of the working muscles.

The decrement in muscular performance varied from 9.4% (0.3-kg ball) to 5.6% (3.0-kg ball) in this study, indicating a pronounced effect of cooling on fast contraction velocities. This was further confirmed by the positive correlation found between the decrease of *m. triceps brachii* IEMG-activity and the decrease in velocity of the lightest ball. The power output during short-term dynamic exercises has been reported to decrease after cooling by approximately 4–8% • $°C^{-1}$ decrease in T_m (5,7,15). In the present study a smaller value was found: the decrement in muscular performance was 2.4% • $°C^{-1}$ decrease in T_m. The variation may be due to a more severe effect of local than whole body cooling or the exercise mode used in this study (using large muscle mass).

After exposure to 10°C the antagonist muscle (*m. deltoideus*) was more severely cooled than the agonist (*m. triceps brachii*). This may be explained by the posture of the subjects during the exposures. The subjects were seated with their arms freely in their laps. This posture may have caused *m. triceps brachii* to be in contact with the trunk, thus slowing the rate of its cooling.

The onset of *m. triceps brachii* activity as a starting point for EMG-analysis made the duration of the analysis period variable. This may have affected these results. However, the use of a fixed time period in the EMG-analysis would certainly have affected the results by leaving out some activity or including inactive moments. Though the use of an internal reference point causes variability in time periods of EMG-analysis, it is still essential to analyze the activity of the muscle during its specific work period. This was done in this study: the EMG-analysis covered the actual work period of *m. triceps brachii* regardless of ball weight and exposure, thus ensuring the reliability of the results.

Cooling could affect the amplitude of EMG by decreasing conduction velocity, modifying the shape of

TABLE I. THE EFFECT OF 60 MIN THERMAL EXPOSURES ON RECTAL TEMPERATURE (T_r), MEAN SKIN TEMPERATURE (T_{sk}) AND MUSCLE TEMPERATURES OF *m. triceps brachii* (T_{tri}) AND *m. deltoideus* (T_{delt}).

			30-mm depth		20-mm depth	
	T_r	T_{sk}	T_{tri}	T_{delt}	T_{tri}	T_{delt}
27°C	36.9 ± 0.1	31.4 ± 0.5	32.8 ± 0.8	34.0 ± 1.1	32.1 ± 0.8	33.4 ± 1.2
10°C	36.8 ± 0.2	25.1 ± 0.6	30.6 ± 1.4	30.8 ± 0.7	28.1 ± 1.5	28.3 ± 0.7
diff	0.2 ± 0.2	6.3 ± 0.5	1.8 ± 0.0	3.2 ± 0.8	4.0 ± 1.6	5.1 ± 0.4
	NS	***	NS	*	*	**

The values are mean ± SE of 11 subjects, except muscle temperature (n = 4–5). Difference between temperatures are denoted by (diff) and * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$, and NS = not significant.

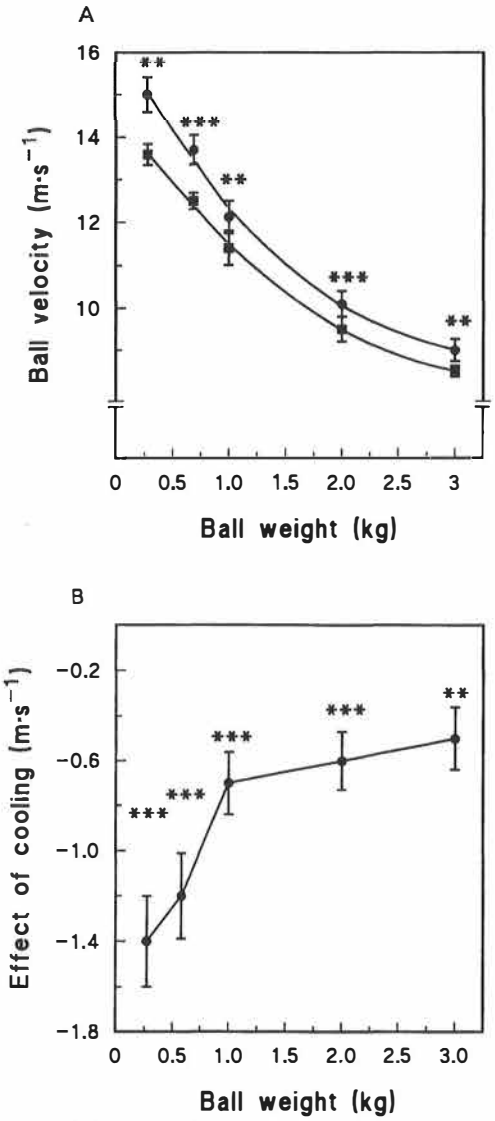

Fig. 1. Ball velocities after exposures to 27°C and 10°C (A) and the effect of cooling-induced decrement in ball velocities in relation to ball weight (B). Circles denote 27°C and squares 10°C. The values are mean ± SE, ** = $p < 0.01$ and *** = $p < 0.001$.

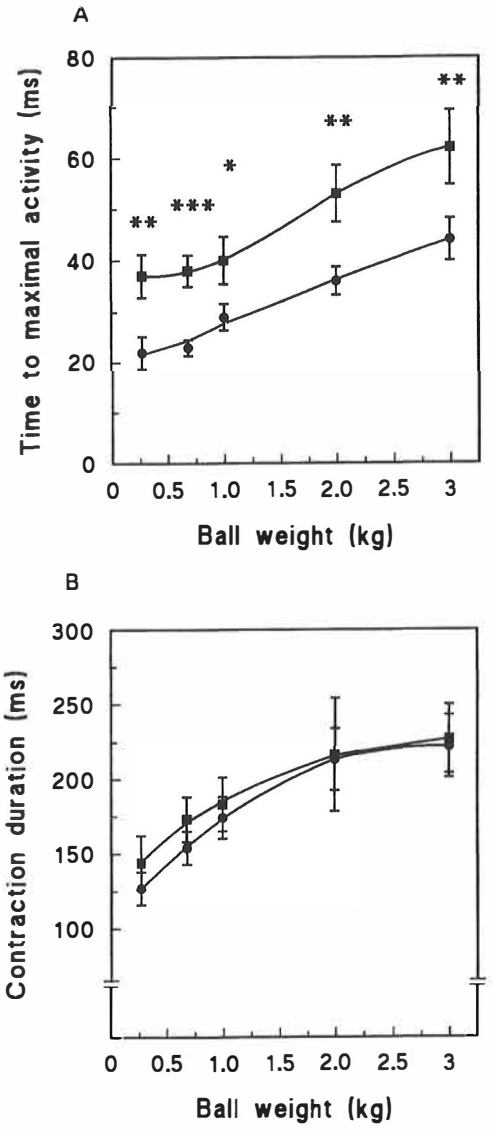

Fig. 2. The effect of cooling on the time to reach the maximal level of IEMG-activity, TMA (A) and contraction duration, CD (B) of m. *triceps brachii*. Circles denote 27°C and squares 10°C. Explanations as in Fig. 1.

the action potential, or changing the impedance of the skin. However, literature reports somewhat contradictory effects of cooling. Some authors have reported an increased activity (16), while others suggest a decreased activity (13,14). Since the EMG activity of the agonist and antagonist muscles in this study changed in an opposite manner after the exposure to 10°C, the observation cannot be explained solely by the methodological effects of cooling on EMG.

In cool muscles, a prolongation in the time to reach maximal force level has been observed (7). This, in turn, reflects a delay in the formation of cross bridges (1). It could be assumed that the delayed function of a cooled muscle should also be seen in its EMG-activity. This

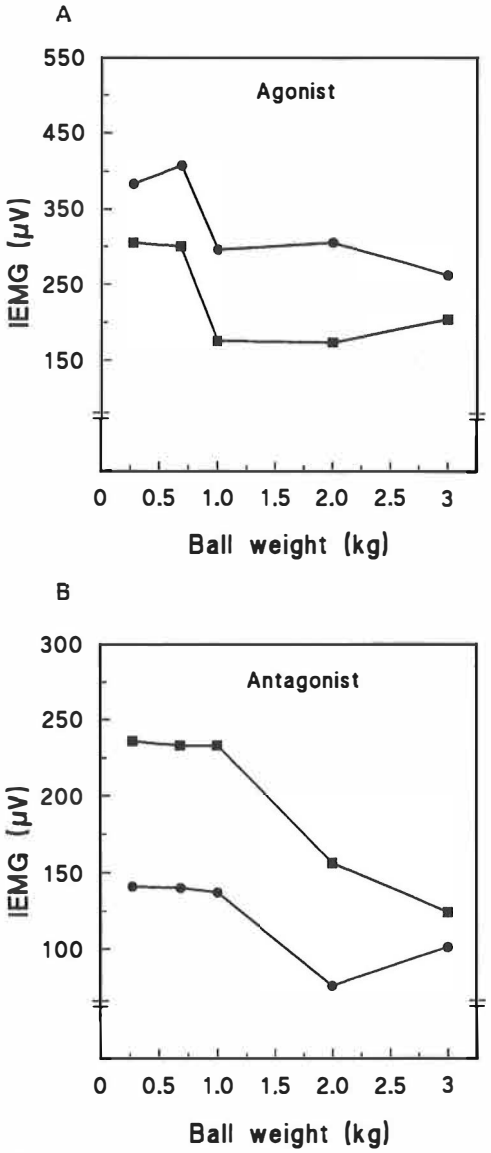

Fig. 3. An example of our subject's agonist (A, *m. triceps brachii*) and antagonist (B, *m. deltoideus*) IEMG-activity variation after exposures to 27°C (circles) and 10°C (squares).

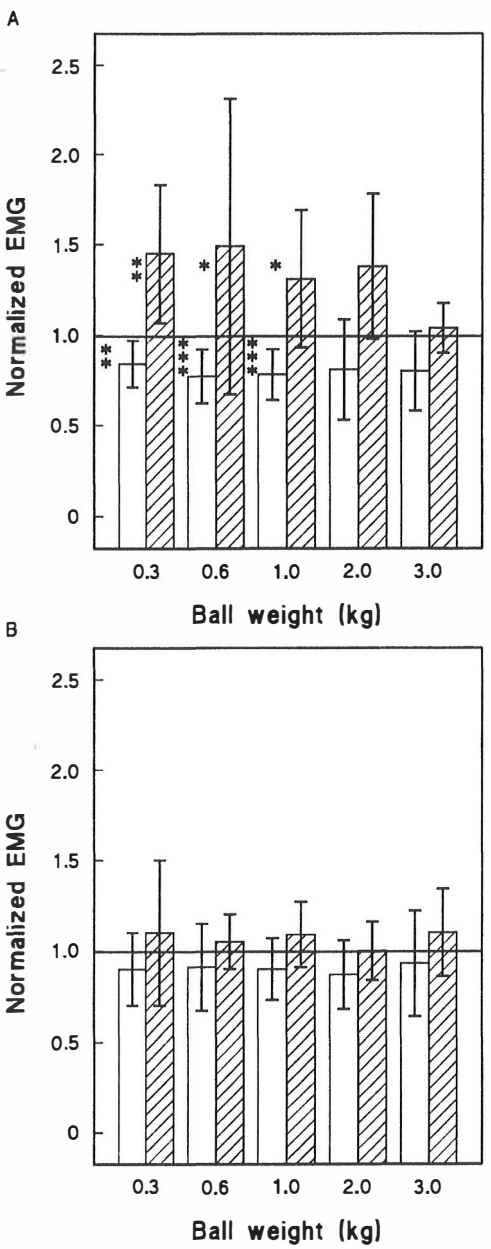

Fig. 4. The effect of cooling on the normalized IEMG-activity of agonist-antagonist muscle pairs of the upper arm (A) *m. triceps brachii* (solid bar) and *m. deltoideus* (dashed bar) and trunk (B) *m. rectus abdominis* (solid bar) and *m. erector spinae* (dashed bar). The line at 1.0 represents the activity at 27°C. Values above the line denote increased activity and values beneath the line decreased activity after exposure to 10°C. * = $p < 0.05$, ** = $p < 0.01$, and *** = $p < 0.001$.

assumption was confirmed in this study: a significant prolongation in the time to reach the maximal level of IEMG-activity during contraction (TMA) of the agonist muscle (*m. triceps brachii*) was observed after cooling. Additionally, the CD of *m. triceps brachii* also tended to increase after cooling (Fig. 2).

Recently, Faulkner et al. (9) stated that: "When muscle temperatures are decreased, many coordinated movements involve the decreased capacity for power of agonist muscle groups coupled with an increased power absorption by antagonistic muscle groups." The results of this study are consistent with those as proposed by Faulkner et al. (9). The throwing exercise is a very coordinated movement and the activity changes of the agonist and antagonist muscles were opposite: the IEMG activity of the agonist muscles was decreased, whereas that of the antagonist muscles was increased after cooling. The observed difference was significant in the agonist-antagonist muscle pair of the arm, but not of the trunk. This may be due to more severe cooling of the arm than the trunk.

A similar change in the EMG activity of antagonist muscle has been reported previously. Bawa et al. (2) found that during light muscular work (extension of the elbow) the antagonist (*m. biceps brachii*) cocontracted together with the agonist (*m. triceps brachii*, detected by EMG). This occurred when the subjects were shivering, but not when the subjects were thermoneutral. Therefore, it is possible that the co-contraction observed by Bawa et al. (2) was mainly due to shivering. In the present study bursts of shivering cannot be excluded, but continuous shivering was not observed. Due to the maximal effort during throwing, and therefore high levels of IEMG-activity and very short contraction durations, it is questionable if possible bursts of shivering could have substantially increased the IEMG-activity. Further, the IEMG-activity of agonist muscles were, in fact, decreased after cooling.

Only tentative suggestions can be made about the role of the observed changes in IEMG-activity of agonist and antagonist muscles. They may have a protective significance, by slowing the maximal velocity of movements and, therefore, guarding against tissue damage in a cooled situation. Nevertheless, both the decreased activity and increased TMA of agonist muscle (*m. triceps brachii*), as well as increased activity of antagonist muscle (*m. deltoideus*), could partly explain why muscular performance was decreased after cooling.

In the study of Bergh and Ekblom (5) where a jumping exercise was performed, no differences between the agonist and antagonist muscle EMG-activity in the leg were found after cooling. However, their results were based on data only from one test subject.

In conclusion, cooling decreased muscular performance, especially with fast contraction velocities. Along with the decrement in performance, cooling slows the function of the agonist muscle and decreases its IEMG-activity, but increases the IEMG-activity of the antagonist muscle.

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III

EMG-activity and muscular performance of lower leg during stretch-shortening cycle after cooling

by

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Acta Physiologica Scandinavica 157, 71-78, 1996

https://doi.org/10.1046/j.1365-201X.1996.452172000.x

IV

Muscle performance and electromyogram activity of the lower leg muscles with different levels of cold exposure

by

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European Journal of Applied Physiology 75, 484-490, 1997

V

Recovery of muscular performance by rewarming exercise in the cold

by

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Human Movement Science 15, 591-603, 1996

https://doi.org/10.1016/0167-9457(96)00024-3

ELSEVIER

Human Movement Science 15 (1996) 591–603

HUMAN MOVEMENT SCIENCE

Recovery of muscular performance by rewarming exercise in the cold

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Abstract

The effect of exercise on muscular performance and EMG-activity of the cooled lower leg muscles was studied. Eight voluntary male subjects (age 26 ± 4 years (mean $\pm$ SD), weight 72 ± 6 kg, height 175 ± 8 cm and body fat $15 \pm 3\%$) dressed in shorts and jogging shoes participated in the study. They were exposed to 27°C and 10°C for 60 min. After the 60 min exposure to 27°C the subjects performed two maximal drop-jumps (stretch–shortening cycle) from a 40 cm bench onto a force plate. The flight time was analysed and the jump with longer flight time was chosen as thermoneutral reference. Also the duration of the contact phase, force production, takeoff velocity and the electromyographic activity (EMG) of the working agonist–antagonist muscle pair (*m. triceps surae* and *m. tibialis anterior*) were measured. After the exposure to 10°C the subjects performed one drop-jump. To study the effect of exercise on the performance the subjects were thereafter allowed to walk on a treadmill at 10°C for 5 min with a speed of 1.4 $m \cdot s^{-1}$ (5 $km \cdot h^{-1}$) and after each walking bout they repeated the drop-jump. This cycle was repeated until the thermoneutral reference value in the flight time was achieved. One subject reached the thermoneutral flight time value after three walking bouts, four subjects after four bouts and three subjects after five bouts. Thermoneutral level in the duration of the contact phase, average force production during shortening and takeoff velocity were attained at the latest after the fifth walking bout. Muscle (*m. gastrocnemius medialis*) temperature was returned to thermoneutral level on the average after two walking bouts. Calf and shin skin temperatures rose significantly along the walking bouts but did not reach thermoneutral level. The walking bouts did not affect mean skin or rectal temperature. When compared to EMG-activity at 27°C during the shortening phase, cooling decreased the integrated EMG-activity (IEMG) and mean power frequency (MPF) of the agonist muscle but increased the IEMG-activity of the antagonist muscle.

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PII S0167-9457(96)00024-3

These changes vanished along with repetitive walking bouts and thermoneutral level of EMG-activity was gained. It is concluded that a relatively small amount of low intensity exercise is required to recover muscle temperature, muscular performance and EMG-activity of the cooled muscles. The reversible changes in EMG-activity of the agonist–antagonist muscle pair correlated with the increased force production and thus increase in the flight time.

PsycINFO classification: 2500; 2530; 2540

Keywords: Cooling; Rewarming; Muscular performance; EMG

1. Introduction

One of the main elements of the human body which enables work in cool and cold environments is the capacity of the muscles to produce heat while working. Due to the low mechanical efficiency (ca. 20%) muscular exercise is an effective method for heat production (Andersen et al., 1971). Its capacity is considerable: metabolic rate can be momentarily increased even up to 15 fold and for extended periods up to 10 fold in comparison to basal metabolic rate in well-trained persons (Åstrand and Rodahl, 1977).

Muscular exercise has been used as a method for rewarming the lowered superficial or deep body temperatures. Body heat balance can, at least partially, be recovered with exercise while the temperatures of the extremities are more difficult to recover. Rewarming of peripheral parts of the body is most efficient when the muscles of the extremities are working, thus producing heat and stimulating circulation (Rintamäki et al., 1992; Sawka et al., 1984).

A surprisingly low level of cooling is sufficient to decrease muscular performance and its various components. Already exposure to 20°C (wearing shorts) for one hour is sufficient to decrease muscular performance by 15% (Oksa et al., unpublished). Co-ordination of muscle contraction is as well altered due to cooling: the EMG-activity of the agonist muscle decreases and the EMG-activity of the antagonist muscle increases. These changes may be one possible reason for decreased performance (Oksa et al., 1995a).

Due to the low level of cooling, which is sufficient to decrease muscular performance, it is conceivable that performance is decreased more often than expected in many daily situations during work or leisure time. The recovery of performance is important when considering that frequent exposure to cooling may be a risk factor for musculoskeletal disorders (Rantanen and Lehtinen, 1992). In addition, cooling may occur in many athletic events as well as during leisure time activities, thus decreasing performance and also leading to an

increased risk of accidents. Though the disadvantages of cooling on muscular performance are well verified, the quantification of the effects of rewarming exercise to recover the performance and altered co-ordination is sparse.

This study was designed to evaluate:

1. How much exercise is needed to recover cooling-induced impairment in muscular performance and its components: force production, velocity and duration as well as the altered activity of the cooled agonist–antagonist muscle pair?
2. Is the recovery of performance dependent on the recovery of muscle temperature?
3. Can the changes in muscular performance be explained by changes in the EMG-activity of the working muscles?

2. Methods

2.1. Subjects

Eight sedentary and healthy men volunteered as test subjects. Their age was 26 ± 4 years (mean $\pm$ SD), height 175 ± 8 cm, weight 72 ± 6 kg and body fat $15 \pm 3\%$. Before the tests the subjects were medically examined, the experimental protocol was explained and their written consent was obtained. The experimental protocol has been approved by the Ethics Committee of the Institute of Occupational Health.

2.2. Thermal exposures and temperature measurements

Before the tests the subjects sat motionless for 60 min in a climatic chamber at 27°C (thermoneutral reference) and 10°C. The subjects were dressed in shorts and jogging shoes. During the exposures and following walking exercise bouts, skin temperatures (forehead, upper arm, forearm, chest, back, thigh, shin [T_s] and calf [T_c]), rectal temperature (T_r 10 cm depth) (Yellow Springs Instruments, YSI 400 series) and muscle temperature (T_m) with an elastic wire electrode from *m. gastrocnemius medialis* (3 cm depth underneath the skin surface, YSI 511) were recorded with one minute interval in a data logger (Squirrel 1200, Grant, UK). Mean skin temperature (T_{sk}) was calculated by weighting the eight local skin temperatures by representative areas (Mitchell and Wyndham, 1969). Body temperature (T_b) was calculated by the equation: $T_{sk} \cdot 0.35 + T_r \cdot 0.65$. Body heat content ($Q$) was calculated by the equation: $3.48 \cdot T_b \cdot$ body weight, where 3.48 = is the specific heat of the body ($kJ \cdot kg^{-1} \cdot °C^{-1}$) (Minard, 1970).

2.3. Drop-jump exercise (Stretch–shortening cycle)

After the thermal exposures the subjects performed a drop-jump exercise from a 40 cm bench. At 27°C the subjects dropped freely from the bench two times onto a force plate (Kistler 9287A, Switzerland) and performed an instantaneous maximal rebound jump (stretch–shortening cycle). The subjects were asked to perform the jump with as straight legs as possible (knee angle was between 150–170°) in order to fully utilize the capacity of *m. triceps surae* complex. Beforehand the subjects thoroughly practiced for the drop-jumps, which was to avoid any training effect during the measurements. The flight time of the jumps (depicting muscular performance) was achieved from the force plate data. The jump having longer flight time was considered as thermoneutral reference. After the 60 min exposure to 10°C the subjects performed one drop-jump. Thereafter, the subjects were allowed to walk on a treadmill at 10°C for 5 min with a speed of 1.4 $m \cdot s^{-1}$ (5 $km \cdot h^{-1}$) and after each walking bout they repeated the drop-jump. This cycle was repeated until the thermoneutral reference value in the flight time was achieved.

2.4. EMG measurements

The electromyographic activity (EMG) of the agonist and antagonist muscle pair of the lower leg was measured. The three parts of *m. triceps surae* complex (*m. gastrocnemius medialis*, *m. gastrocnemius lateralis* and *m. soleus*) and *m. tibialis anterior* were measured. In the drop-jump exercise *m. triceps surae* complex (agonist muscle) generates force to cause the extension of the ankle, whereas *m. tibialis anterior* flexes the ankle (antagonist muscle). EMG signals from the skin above the working muscles were measured by a computerized system which sampled with the frequency of 1250 Hz (Mespec 4001, Mega Electronics, Finland), using pregelled bipolar surface electrodes (Medicotest, M-OO-S, Denmark). The electrodes were placed in the middle part, over the belly of the muscle, except in *m. soleus* where the electrodes were placed in the descending lateral portion, approximately 15 cm above *lateralis malleolus*. The distance between recording contacts was 2 cm. Two ground electrodes were placed above inactive tissue. To ensure the accuracy of replacing the electrodes, their places were carefully marked on the skin with waterproof drawing ink. The markings were clearly visible throughout the experiments. The measured EMG signal was amplified 2000 times (preamplifier located 6 cm after the measuring electrodes) and the signal band between 20 and 500 Hz was full wave rectified and integrated (IEMG) with a 13 ms time constant. To assess the frequency

component of the EMG, the power spectrum was estimated by moving Fast Fourier Transform (FFT) window (256 points). From the power spectra, mean power frequency (MPF) and median frequency (MF) were calculated to describe changes in the frequency component. Butterworth filtering was used in the measuring band. The technical qualities of the amplifier were: common mode rejection ratio (CMRR) 130 dB, noise 0.5 μVrms and input impedance 20^9 ohm.

The EMG measurement started with a signal that was given to the subjects to perform the drop-jump. An on–off connector was attached to the sole of the shoe which gave a signal of the contact phase to the EMG measurement device. The EMG data were analysed during the preactivity phase (100 ms before the beginning of stretch) and stretch and shortening phases of the drop-jumps. Intraindividual comparisons of the mean IEMG, MPF and MF results were done between the exposures and walking bouts. The results of *m. triceps surae* complex are presented as an average of the three agonist muscles.

2.5. Force plate measurements

To measure ground reaction forces during the contact phase of the drop-jump a piezo-electric force plate system was used (Kistler 9287A, Switzerland). The measuring frequency of the force plate was 200 Hz and the obtained signal was amplified 50000 times (Kistler, charge amplifier 9865C). The linearity and hysteresis of the force plate were %FSO $< \pm 1$. From the data, average force production in the vertical plane (F_{conc}) during the shortening phase was calculated and also the flight times of the subjects were analysed.

2.6. Motion analysis

To determine the duration of the contact (t_c), stretch (t_{ecc}) and shortening (t_{conc}) phases and the takeoff velocity (Vel) (velocity of the *lateralis malleolus* at the moment of takeoff) of the subjects an infra-red optoelectronic motion analysis system was used (MacReflex, Qualisys, Sweden). The system consists of two cameras emitting infra-red light pulses each with a frequency of 50 Hz, with a 10 ms delay in relation to each other (total frequency 100 Hz), videoprocessors and a computer. Five infra-red light emitting markers attached to the hip, knee, ankle and tip of the shoe of the subjects were used. The fifth marker served as a reference and was attached to the side of the force plate. These markers remit infra-red light pulses which are sent from the camera-system, thus providing a two dimensional spatial co-ordinate information of the movements of the markers to the motion analysis system. The data were

delivered in a digital form to a computer and processed using a WingZ MacReflex-table calculation program. To minimize measuring errors the cameras were oriented perpendicularly to the measurement plane and were placed one above another.

2.7. Statistics

The results from the drop-jump with longer flight time at 27°C were considered as thermoneutral reference values. The performance and EMG parameters obtained after each walking bout were tested against the thermoneutral reference values and initial values after cooling (the values from the first drop-jump after the exposure to 10°C). SPSS statistical software was used. The dependent variables were tested using analysis of variance with repeated measures. When significant F-ratio was obtained, Duncan's post hoc test was applied. Pearson's product moment correlation coefficient was calculated between performance and EMG parameters. The significance was accepted at the 0.05 level.

3. Results

Calf and shin skin temperatures and muscle temperature increased already after one or two walking bouts (Table 1). The muscle temperature value did not differ significantly from the 27°C resting value after two walking bouts. In spite of the increase of lower leg skin temperatures the mean skin temperature was

Table 1
Rectal (T_r), mean skin (T_{sk}), calf skin (T_c), shin skin (T_s) and *m. gastrocnemius medialis* muscle temperature (T_m) after the exposure to 27°C (reference values), 10°C and after each walking bout

	T_r (°C)	T_{sk} (°C)	T_c (°C)	T_s (°C)	T_m (°C)
27°C	37.0±0.1	32.6±0.3	31.6±0.2	31.8±0.3	32.9±0.5
10°C	37.0±0.1 [a]	25.8±0.6	24.0±0.3	24.1±0.4	29.5±0.7
1 walk	36.8±0.1 [a]	24.8±0.5	23.7±0.7	24.9±0.6	30.5±1.0
2 walk	36.8±0.1 [a]	25.1±0.5	25.2±0.8	26.7±0.5 *	32.5±1.3 [a]
3 walk	36.9±0.2 [a]	25.0±0.5	25.6±0.3 *	28.0±0.3 *	33.0±1.2 *,[a]
4 walk	37.2±0.2 [a]	25.1±0.6	26.3±0.2 *	27.8±0.4 *	33.4±1.5 *,[a]
5 walk	37.0±0.2 [a]	24.9±0.4	27.1±0.3 *	28.2±0.2 *	33.8±0.7 *,[a]

The values are mean ± SE of 8 subjects, except at 4, $n = 7$ and at 5, $n = 3$. In T_m ($n = 6$), except at 4, $n = 5$ and at 5, $n = 1$. The significance in relation to 10°C is denoted by * = $p < 0.05$. Superscript [a] indicates the points where the data does not significantly differ from the 27°C thermoneutral values.

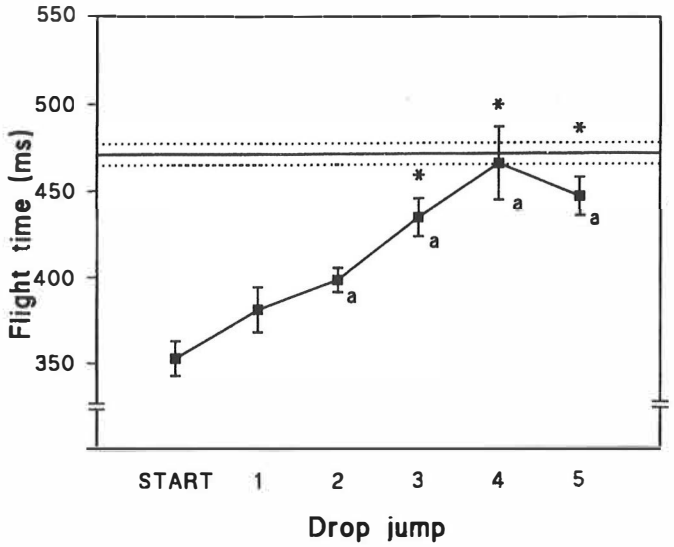

Fig. 1. The flight time after the exposure to 10°C (START) and after the following five walking bouts (1–5). The values are mean ± SE of eight subjects, except at 4, $n = 7$ and at 5, $n = 3$. The statistical difference in relation to START is denoted by * = $p < 0.05$. The solid and dotted lines indicate the thermoneutral value (mean ± SE). The letter 'a' indicates the points where the data does not significantly differ from the 27°C thermoneutral reference value.

not changed due to the slight decrease in upper body skin temperatures. Because rectal temperature remained unchanged by walking bouts the body temperature and body heat content were also unaffected.

Table 2
Changes in performance parameters after the exposure to 10°C and after each walking bout. 27°C are reference values

	t_{ecc} (ms)	t_{conc} (ms)	t_c (ms)	F_{conc} (N)	Vel ($m \cdot s^{-1}$)
27°C	104 ± 9	103 ± 7	206 ± 9	2429 ± 111	2.57 ± 0.09
10°C	133 ± 6	146 ± 9	277 ± 13	1268 ± 100	2.10 ± 0.06
1 walk	136 ± 11	141 ± 10	277 ± 17	1612 ± 105	2.19 ± 0.06
2 walk	117 ± 8 [a]	120 ± 8 [a]	237 ± 12 [a]	1886 ± 144 *	2.30 ± 0.08 [a]
3 walk	116 ± 8 *,[a]	110 ± 8 [a]	226 ± 13 [a]	2104 ± 94 *[a]	2.37 ± 0.1 *,[a]
4 walk	107 ± 7 *,[a]	107 ± 6 *,[a]	214 ± 7 *,[a]	2305 ± 133 *[a]	2.52 ± 0.1 *,[a]
5 walk	110 ± 7 *,[a]	90 ± 5 *,[a]	203 ± 5 *,[a]	2520 ± 38 *[a]	2.35 ± 0.06 *,[a]

Duration of stretch phase (t_{ecc}), shortening phase (t_{conc}) contact phase (t_c), force production during shortening phase (F_{conc}) and takeoff velocity (Vel). The values are mean ± SE of 8 subjects, except at 4, $n = 7$ and at 5, $n = 3$. The significance in relation to 10°C is denoted by * = $p < 0.05$. Superscript [a] indicates the points where the data does not significantly differ from the 27°C thermoneutral values.

Table 3
The correlation coefficient (r) of performance and EMG-parameters in relation to flight time and average force production during shortening phase (F_{conc})

	Increase in flight time	F_{conc}
F_{conc}	0.477 * *	–
Vel	0.800 * * *	0.342 NS
T_s	0.692 * * *	0.165 NS
T_m	0.590 * *	0.233 NS
IEMG *m. ts*	0.318 *	0.465 * *
MPF *m. ts*	0.674 * * *	0.523 * *
IEMG *m. ta*	−0.607 * * *	−0.550 * * *

Takeoff velocity (Vel), shin (T_s) and muscle (T_m) temperature, IEMG and MPF of *m. triceps surae* (*m. ts*), IEMG of *m. tibialis anterior* (*m. ta*) during shortening phase. The significance of correlation is denoted by * = $p < 0.05$, * * = $p < 0.01$ and * * * = $p < 0.001$.

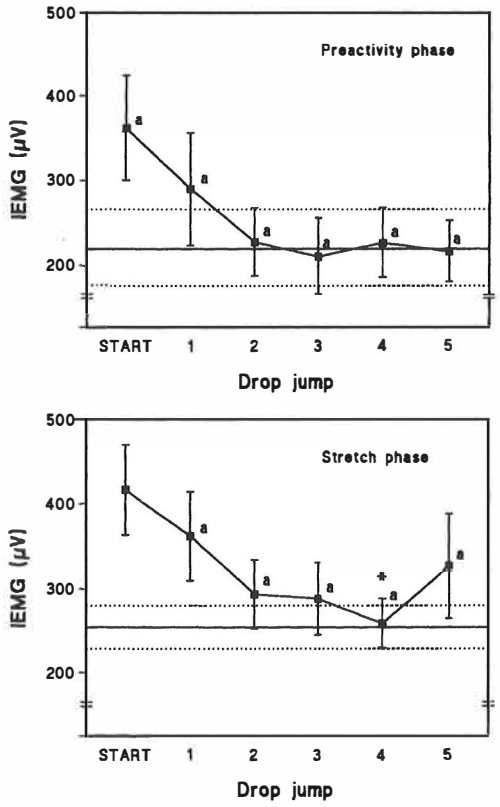

Fig. 2. The IEMG of *m. triceps surae* during preactivity and stretch phases after the exposure to 10°C and following five walking bouts. Explanations are as in Fig. 1.

Muscular performance was considered to have been recovered when thermoneutral (27°C) reference value in the flight time was attained. Recovery occurred after three walking bouts in one subject, after four bouts in four subjects and after five bouts in three subjects. The flight time started to increase already after the first walking bout and was significantly higher (in relation to first drop-jump at 10°C) after three walking bouts (Fig. 1). After two walking bouts the flight time did not differ siginificantly from the 27°C reference value.

The duration of the contact phase, t_c (both stretch, t_{ecc} and shortening, t_{conc}) started to decrease after two walking bouts. The average force production was significantly higher after two and takeoff velocity after three walking bouts in relation to 10°C (Table 2). At the latest after the third walking bout the results did not differ significantly from the 27°C reference values.

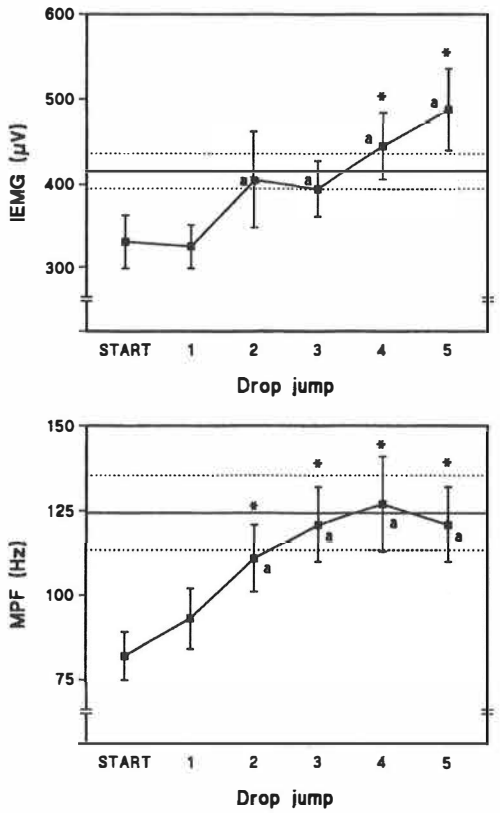

Fig. 3. The IEMG and MPF of *m. triceps surae* during shortening phase after the exposure to 10°C and following five walking bouts. Explanations are as in Fig. 1.

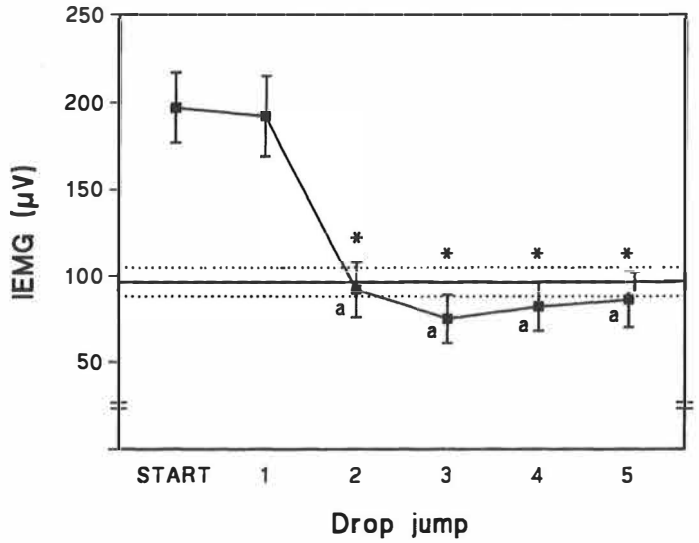

Fig. 4. The IEMG of *m. tibialis anterior* during shortening phase after the exposure to 10°C and following five walking bouts. Explanations are as in Fig. 1.

The EMG results concerning frequency changes were similar in MPF and MF. MPF was chosen to represent the frequency component of the EMG results.

Cooling increased the IEMG-activity of the agonist muscle (*m. triceps surae*) during preactivity and stretch phases in relation to 27°C reference values (Fig. 2). However, cooling decreased the IEMG-activity and MPF of the agonist muscle during the shortening phase (Fig. 3), whereas it increased the IEMG-activity of the antagonist muscle (*m. tibialis anterior*) (Fig. 4). when compared to 27°C reference values. These changes vanished along with repetitive walking bouts and the not significant difference in relation to the 27°C reference values was reached at the latest after the second walking bout.

The increase in the flight time correlated significantly with F_{conc}, Vel, EMG-activity during the shortening phase and with shin skin and muscle temperatures. Both the increasing IEMG-activity and MPF of the agonist muscle as well as the decreasing IEMG-activity of the antagonist muscle correlated significantly with F_{conc} (Table 3).

4. Discussion

The results of this study clearly show that decreased muscular performance can be recovered even with small amounts of low intensity exercise. The

exercise also increased the local muscle or skin temperatures without affecting the whole body heat content. Increase in muscular performance, depicted by the increase in flight time, was accompanied by restorative changes in force production and EMG-activity.

The measure of the recovery of muscular performance (flight time) started to increase already after the first walking bout and reached statistical significance after the second bout. Thermoneutral level of performance in all subjects was reached within five bouts. When considering the intensity and duration of walking surprisingly little exercise is needed first to enhance muscular performance and finally to recover it.

Rintamäki et al. (1992) found that leg skin temperatures of cooled subjects were rewarmed most efficiently by stepping, in comparison to arm cranking or bicycling exercise with equal metabolic rate. In this study the amount of walking exercise was sufficient to warm the working muscles but not the overlying skin in the lower leg to a thermoneutral level. Average muscle temperature increased by 5.3°C to 33.8°C, rising above the 27°C reference T_m (32.9°C) already after three walking bouts. Calf skin temperature increased by 3.1°C to 27.1°C but did not reach the thermoneutral level of 31.6°C. Shin skin temperature increased by 4.1°C to 28.2°C bit did not reach thermoneutral level of 31.8°C, either. Muscle and shin skin temperature correlated significantly with the length of the flight time but calf skin temperature did not. These results show that thermoneutral skin temperature level is not a prerequisite to recover muscular performance, more important is the temperature of the muscle tissue.

Other factors which correlated significantly with the increase in the flight time were average force production, takeoff velocity and changes in the EMG-activity of the agonist–antagonist muscle pair during the shortening phase. Takeoff velocity had the strongest correlation ($r = 0.800$, $p < 0.001$) to the increase in flight time. However, takeoff velocity can be regarded as a function of the speed of force production: the more force one is able to produce at a given time the faster is the takeoff velocity (Häkkinen and Komi, 1986). The increase in the IEMG and MPF of *m. triceps surae* and decrease in the IEMG of *m. tibialis anterior* during the shortening phase all correlated significantly with the increase in force production during shortening (Table 3). This implies that the changes in agonist–antagonist muscle pair EMG-activity, apparently caused by increased muscle temperature, could be responsible for the increase in force production and thus the increase in flight time.

In this study similar changes due to cooling were found as in previous studies (Oksa et al., 1995a; Oksa et al., 1995b). Cooling increased the activity of the agonist muscle during preactivity and stretch phases, but decreased it during the

shortening phase. The activity of the antagonist muscle during preactivity and stretch phases remained unchanged but increased during the shortening phase. These changes in the EMG-activity of the agonist–antagonist muscle pair have been shown to be in connection with decreasing ambient exposure temperature and consequently decreasing muscle temperature (Oksa et al., unpublished). The present results indicate that along with the increasing muscle temperature and without totally recovered skin temperatures the above mentioned changes in EMG-activity are recovering and finally they correspond to thermoneutral activity. These results reflect the reversible nature of the phenomenon and suggest its dependence on the temperature of the muscle tissue.

The observed changes in EMG-activity may be caused either by central or peripheral regulation. The preactivity and stretch phase activity may be due to an anticipatory effect arising from the evolving impact of the ground contact (Komi, 1983). This effect has previously been observed to be elevated due to cooling (Oksa et al., 1995b). The results of this study show that the activity of the agonist muscle during preactivity and stretch phases is decreasing when the muscle is becoming warmer (decreased anticipatory effect) (Fig. 2). This may be possible due to decreased supraspinal drive from the central nervous system.

The activity arising during the shortening phase may be partly regulated by stretch reflex (T-reflex) which is initiated during the stretch phase. The reflex is activated by stretching the muscle spindles which in turn facilitates the following contraction of the agonist muscle and inhibits the contraction of the antagonist muscle (Matthews, 1964). Previously it has been found that cooling decreases the activity of muscle spindles (Bell and Lehmann, 1987; Eldred et al., 1960) and consequently the amplitude of EMG and the force produced by the agonist muscle (Knutsson and Mattsson, 1969; Petajan and Watts, 1962). However, it is not known if rewarming has a similar but contradictory effect on this phenomenon. Moreover, the function of the antagonist muscle has not been well studied. There are only implications that with spastic patients the effectiveness of antagonist contraction would be increased after local cooling (Knutsson, 1970). Nevertheless, if the facilitation of the function of the agonist muscle is decreased due to cooling or increased due to warming, it may be assumed that also the degree of inhibition of the antagonist muscle is temperature dependent. If this temperature dependence exists (as seems to be the case concerning the EMG-activity) it could be one explanation for the observed contradictory changes in the EMG-activity of the agonist–antagonist muscle pair during shortening.

In conclusion, bouts of low intensity exercise were sufficient to increase muscle temperature (without affecting the whole body heat content) and recover

muscular performance of the leg muscles to a thermoneutral level. The simultaneous reversible changes in the force of contraction (measured as EMG-activity) of the agonist–antagonist muscle pair could be responsible for the increased force production and thus an increase in the flight time.

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VI

Leg T- and H-reflexes during whole body cooling and local warming

by

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