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Muscle Thixotropy

Implications for Human Motor Control

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Abstract

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Human skeletal muscles possess thixotropic, i.e. history-dependent mechanical properties. This means that the degree of passive muscle stiffness and resting tension is dependent on the immediately preceding history of contractions and length changes. Athletes, for instance, reduce passive muscle stiffness by various types of 'limbering-up' procedures, whereas muscle stiffness gradually increases during inactivity.

Passive resistance of antagonistic muscles may significantly add to the total load during voluntary muscle contractions. This resistance may vary from one moment to another, depending on immediately preceding events. This research was conducted to determine whether history-dependent variations in passive muscular forces influence motor control of voluntary joint movements and steady maintenance of joint positions in healthy subjects.

In study I, the EMG signal revealed motor compensations for history-dependent variations in passive stiffness of the antagonists during slow voluntary wrist joint movements. Studies II and III demonstrated that the voluntary muscle activity required to maintain a certain wrist joint position was highly influenced by previous changes in forearm muscle length and contractions. Study IV showed that rapid voluntary movements varied in speed and onset time depending on the prevailing degree of muscle resistance, and in addition that the central nervous reaction time required to execute rapid movements was highly influenced by immediately preceding muscle-conditioning procedures.

History-dependent variations in passive muscular forces seem to be effectively compensated by the motor control system. Presumably, voluntary motor commands to the muscles are automatically adjusted in strength to history-dependent changes in passive muscular forces. Such adjustments occur within the central nervous system, which receives information about the mechanical state of the muscles. Several issues in connection with muscle thixotropy remain unaddressed. For instance, do alterations in the normal thixotropic mechanical behaviour of the muscles impose a particular problem in patients with certain neuromuscular diseases?

Keywords: Motor control, Skeletal muscle, Thixotropy, History-dependent, EMG, Voluntary contractions

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To Anna, Emil & Fredrik

List of papers

This thesis is based on the following papers. They will be referred to in the text by their Roman numerals:

- I **H. Axelson**, K-E Hagbarth, Human motor control consequences of thixotropic changes in muscular short-range stiffness. *J Physiol*. 2001 Aug 15; 535:279-88.
- II **H. Axelson**, K-E Hagbarth, Human motor compensations for thixotropy-dependent changes in resting wrist joint position after large joint movements. *Acta Physiol Scand*. 2003 Dec; 179(4):389-98.
- III **H. Axelson**, Human motor compensations for thixotropy-dependent changes in muscular resting tension after moderate joint movements. *Acta Physiol Scand*. 2004 Nov; 182(3):295-304.
- IV **H. Axelson**, Signs of muscle thixotropy during human ballistic wrist joint movements. Submitted for publication.

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Contents

Introduction.....	9
History-dependent properties of the skeletal muscles.....	10
Basic mechanics	10
Thixotropy	11
Viscoelasticity	12
Implications	13
Motor consequences of muscle thixotropy.....	14
Previous observations.....	14
General aims of the studies.....	15
Specific aims of the studies.....	16
Methods	17
Subjects	17
Experimental set-up.....	17
Passive force and joint angle measurements	18
EMG recordings	19
Muscle-conditioning procedures	19
Experimental designs: in general	20
Data acquisition and statistics	21
Results and specific experimental designs.....	22
Study I.....	22
Experimental design	22
Short-range stiffness in passive wrist joint torque.....	23
Short-range stiffness in passive flexor tension	24
Motor adjustments for short-range stiffness.....	25
Passive and voluntary movements generated by constant forces: the effect of stirring procedures.....	26
Conclusion	27
Study II.....	28
Experimental design	28
Thixotropic changes in resting wrist joint position	28
Motor compensations for changes in resting wrist joint position.....	29
Motor adjustments during steady maintenance of a wrist position.....	30
Conclusion	31

Study III	33
Experimental design	33
After-effects in passive torque during stepwise movements	34
Motor adjustments	35
The effect of intervening ‘stirring’ co-contractions	37
Conclusion	39
Study IV	40
Experimental design	40
Passive twitch movements and voluntary ballistic movements	41
Repetitive passive twitch movements and voluntary ballistic movements	43
Premotor reaction time	43
Conclusion	43
General discussion	45
Mechanisms	45
History-dependent properties of the skeletal muscles	46
Motor control	47
Methods	48
EMG	48
Force measurements	49
Implications	49
Future perspectives	50
Sammanfattning på svenska (Summary in Swedish)	51
Acknowledgements	53
References	55

Abbreviations

EMG	Electromyography
IEMG	Integrated surface electromyography
sMRT	Stimulus motor reaction time
SREC	Short-range elastic component
FRT	Filamentary resting tension
MRT	Motor reaction time
MT	Movement time
PMRT	Premotor reaction time
EXT	Isometric wrist extensor contractions
FLEX	Isometric wrist flexor contractions
OSC	Oscillating wrist joint movements
REST	Resting period

Introduction

Many daily motor activities involve precise voluntary movements and steady maintenance of joint positions. Such motor tasks are executed and regulated by the central motor nervous system. A crucial task for the motor system is to compensate for changes in load that may otherwise disturb intended movements or positioning of the extremities.

It is well known that load-compensatory mechanisms rely on proprioceptive information from peripheral receptors such as the muscle spindles and Golgi tendon organs, which detect changes in skeletal muscle length and tension. This afferent information concerning changes in load converges in the central nervous system and modifies the motor commands to the force-generating skeletal muscles.

Changes in load for voluntary contractions may, for instance, be due to increased passive muscular resistance, which can sometimes be perceived as a certain degree of ‘muscle stiffness’. It is empirically well known that muscle stiffness can be reduced by ‘limbering-up’ procedures such as ‘stretching’ and that the stiffness gradually increases during inactivity. Apparently, the passively opposing load for voluntary contractions varies depending on what has happened to the muscles in the preceding time period. In fact, this is a manifestation of the ‘history-dependent’ properties of the skeletal muscles, also referred to as thixotropic effects. Understandably, history-dependent effects in the skeletal muscles have attracted considerable attention among researchers in fields such as biomechanics and sport science.

The main hypothesis put forward in this thesis is that history-dependent variations in passive skeletal muscle resistance have motor control consequences for human voluntary contractions. Despite the vast amount of information available concerning human motor control mechanisms and the passive properties of the skeletal muscles, this particular issue has been largely unexplored. The reason for this might be a common misconception that relaxed muscles act like simple springs during elongation and that their passive resistance may seem easily predictable and may not appear to impose a particular problem for the motor control system to handle.

In this thesis, four studies (I-IV) on healthy subjects are presented, concerning different types of thixotropic changes in the mechanical behaviour of the relaxed wrist muscle-joint apparatus. In consistence with the underlying hypothesis, it was of particular interest to investigate whether such changes had any implications for ordinary voluntary motor tasks such as wrist extensor and flexor muscle contractions.

The results of study I-III provide strong evidence that muscle thixotropy gives rise to motor adjustments as judged by the surface EMG. Unpublished observations by the author have confirmed that similar types of motor adjustments occur in other muscle groups. Findings in study IV clearly indicate that muscle thixotropy also influences the performance of rapid, so-called ballistic, voluntary contractions. In short, these studies add new information about fine motor control, which is often affected in neuromuscular disease. Also, the obtained results should be of interest to those working in the fields of physiotherapy and sports.

In the following, the passive mechanical behaviour of the skeletal muscles of particular relevance to this thesis will be briefly reviewed. Also presented are some earlier studies from this laboratory which have been sources of inspiration for further, more comprehensive investigations on muscle thixotropy and its consequences for human motor control. The following sections will begin with a short explanation of some of the terms and concepts encountered in this thesis and elsewhere in the literature.

History-dependent properties of the skeletal muscles

Basic mechanics

Relaxed skeletal muscles *in situ* typically possess a certain degree of *resting tension* at fixed lengths. During elongation they exhibit *passive stiffness*, i.e. a tension increase during extension. In this thesis, the term *passive resistance* refers to such forces (resting tension and passive stiffness) that oppose voluntary contractions or externally imposed forces. In addition to passive muscular resistance, activation of the antagonists (co-contractions) and gravitational force may also contribute to the total load encountered by the contracting, load-bearing muscles.

In whole joint experiments, forces are typically measured and expressed as *torque* (the rotational force, i.e. force multiplied by the length of the moment arm). The measured joint torque (net torque) is the algebraic sum of all individual forces (torques) acting on the joint.

According to the so-called *equilibrium point hypothesis*, the motor control system regulates joint positions by recruiting reciprocally antagonistic muscles to various degrees (Loeb & Ghez, 2000). The limbs will move to a mechanically defined equilibrium point where antagonistic torques are equally strong.

The implementation of the equilibrium point hypothesis in a motor control context seems to be afflicted with oversimplifications. For instance, it is commonly assumed that co-contractions are essential parts of voluntary joint movements and position maintenance. This is certainly the case if joint stability is imperative, as when preparing to catch a heavy object. In contrast, tasks such as smooth elevation of a limb primarily involve activation of the prime movers against loads formed by gravitation and passive muscle resistance.

It is perhaps not a critical issue to what extent antagonistic forces are generated by contractions and to what extent they are passively developed in the muscles. More importantly, by disregarding the contribution of passive forces in the equilibrium point hypothesis no considerations are paid to the complex *thixotropic* mechanical behaviour of the skeletal muscles.

Thixotropy

Thixotropy, from the Greek *thixis* and *-tropy*, means “transformation by touch”. It is a physical property of substances that appear as semi-solid fluids under resting conditions, but temporarily become more fluid after exposure to ‘*stirring*’ forces. This transformation is caused by detachments of weak bonds between polymeric molecules, bonds which gradually reform during a resting period. There are many inorganic and organic substances that share this physical property (e.g. certain paints, ketchup, synovial fluid, mucus).

About 50 years ago, Buchthal & Kaiser (1951) introduced the term thixotropy in connection with skeletal muscle physiology. In their extensive investigations of the passive mechanical properties of amphibian skeletal muscle fibres, they found that muscle fibre stiffness was temporarily reduced by one single stretch movement and that the stiffness returned during a resting interval. The thixotropic behaviour of the muscles was explained in terms of disruption and reformation of ‘entanglements’ within the muscle fibres.

Presumably, Buchthal & Kaiser (1951) were examining a phenomenon that was later termed the *short-range stiffness* of skeletal muscles. In response to stretch, extrafusal muscle fibres typically display an initial steep linear tension increase - up to a certain *elastic limit* - after which the tension levels off during the remainder of the stretch (Hill, 1968; Rack & Westbury, 1974). This phenomenon was originally designated by D.K. Hill (1968) as the *short-range elastic component* (SREC). Hill proposed that the SREC and a fraction of the muscle fibre resting tension - the filamentary resting tension (FRT) - were generated by ‘stable’ cross-bridges between actin and myosin filaments. This hypothesis has since been supported by many others (Lännergren, 1971; Haugen & Sten-Knudsen, 1981; Hufschmidt & Schwaller, 1987; Campbell & Lakie, 1998). Hufschmidt & Schwaller (1987) suggested that their observation of a steep initial rise in passive human ankle

joint torque in response to imposed movements were the *in situ* manifestation of the SREC.

The skeletal muscles (or the extrafusal muscle fibres) are not fixed in this relatively stiffened state. As concluded by Buchthal & Kaiser (1951), the short-range stiffness is temporarily eliminated or reduced by a stretch movement. In addition, other types of ‘mechanical agitation’ such as that caused by electrical stimulation or voluntary contraction of the muscles concerned have similar after-effects (Hufschmidt & Schwaller, 1987; Wiegner, 1987; Lakie *et al.*, 1984; Lakie & Robson, 1988a; Walsh & Wright, 1988; Warner & Wiegner, 1990).

The apparent thixotropic behaviour of the short-range stiffness has commonly been explained by cross-bridge interactions (Proske *et al.*, 1993). For clarity, it must be pointed out that this has nothing to do with the ‘sliding filament theory’ in connection with muscle fibre activation (Huxley, 1969). On the contrary and in conformity with the original hypothesis proposed by D.K Hill (1968), it is believed that those cross-bridges that are formed in the resting muscle disrupt during stretch and only reform if the muscles are left undisturbed, i.e. relaxed. Further, the cross-bridge reformation is not an instantaneous process but may continue for several seconds or even longer (Hufschmidt & Schwaller, 1987; Lakie & Robson, 1988b; Campbell & Lakie, 1998). During this time interval the resting tension of the muscle fibres gradually increases as a result of the build-up of tension by newly formed cross-bridges.

Viscoelasticity

The skeletal muscles are perhaps more commonly described as having viscoelastic properties. The elastic, spring-like mechanical behaviour provides the muscle with a tension response that is length-dependent, whereas muscle viscosity means that the tension response depends on the rate of length change. In contrast to the ‘ideal spring’, viscoelastic structures produce different types of characteristic tension responses during and after a change in length (Buchthal & Kaiser, 1951; Lännergren, 1971). For instance, *hysteresis* occurs during a passive stretch-release cycle, i.e. the tension is higher during lengthening than during shortening. If skeletal muscle fibres are stretched and then kept at the new length, the resistance gradually declines to a steady level (*stress relaxation*). In contrast, if the muscles are shortened and then held at the shorter length, the resistance gradually increases (*stress recovery*). These phenomena are illustrated schematically in Figure 1A and B.

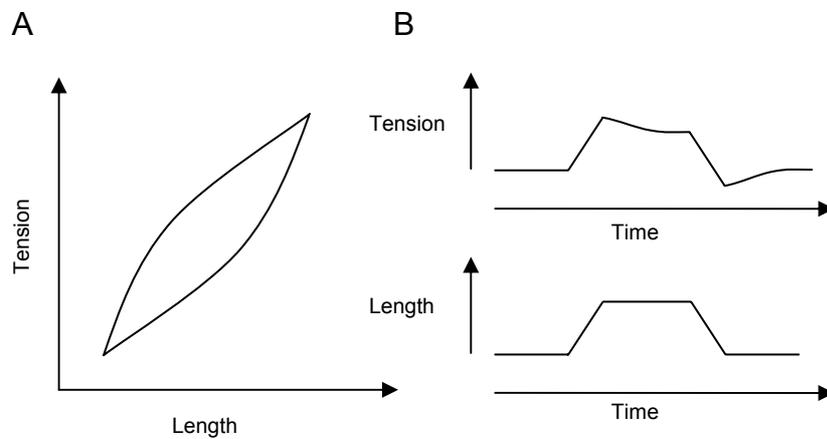


Fig 1. A, hysteresis curve. B, stress relaxation and recovery. Muscle tension may vary at a given length and over time as a result of hysteresis and stress relaxation/recovery .

It may seem doubtful as to whether it is appropriate to use the term thixotropy in connection with all types of after-effects in skeletal muscle mechanics. As exemplified in Figure 1, pure viscoelasticity produces history-dependent after-effects, and many different mechanisms (besides cross-bridge interactions) and structures (besides the muscles) seem to be involved (Butler *et al.*, 1978; Finlay, 1978; Minajeva *et al.*, 2001). Nevertheless, the author has adhered to the term thixotropy and uses it interchangeably with ‘history-dependency’. The reason for doing so will be brought up in the General discussion.

Implications

Irrespective of the underlying mechanisms, skeletal muscle thixotropy implies that muscle stiffness and passive resting tension of the muscles are critically dependent on immediately preceding movements and contractions. It is now generally believed that the short-range stiffness of the muscle provides the musculoskeletal system with an inherent postural stability against minor perturbations of the limbs. Once broken up, the short-range stiffness remains at a relatively low degree as long as the muscles are kept in motion. It is easy to imagine the significance of such stiffness reduction not only in many sports but also in ordinary life.

It is also important to consider thixotropic changes in muscle resting tension. For instance, the relaxed angular position of the joints may to a large extent be determined by passive pulling forces from reciprocally antagonistic muscles. For example, if one muscle group has been exposed to a ‘loosen-

ing-up' procedure, the resting tension will become decreased in those muscles. As a consequence, the passive pulling force of that muscle group will be reduced. This will lead to a shift in the angular resting position (i.e. equilibrium point) of the joint and seems to explain why, for instance, the resting position of the fingers differs according to whether the finger flexors have previously been held flexed or extended (Hagbarth *et al.*, 1985).

If passive forces and in particular their history-dependent properties are incorporated into the equilibrium point hypothesis (as described above) there is clearly no simple relationship between the level of skeletal muscle activity and the joint angle. It seems reasonable to assume that in order to maintain a steady joint position or to produce a given type of movement, the motor control system has to compensate continuously for thixotropic changes in the muscles. Only a limited number of studies (Hagbarth *et al.*, 1995; Nordin & Hagbarth, 1996) have been devoted to this particular issue.

Motor consequences of muscle thixotropy

Previous observations

Thixotropic changes do not occur only in extrafusal muscle fibres, but also in the intrafusal fibres of the muscle spindles. It is now generally accepted that intrafusal thixotropy can affect spindle afferent activity and thereby contribute to history-dependent changes in the strength of the stretch reflex (for a review see Proske *et al.*, 1993).

It has previously been reported from EMG studies that voluntary finger flexor activity required to maintain the index finger in a slightly flexed position against gravity varied in strength depending on whether the finger had been held in a flexed or extended position beforehand (Hagbarth *et al.*, 1995; Nordin & Hagbarth, 1996). Such variations were interpreted as motor adjustments for extrafusal muscle thixotropy in unrecruited parts of the muscles.

It has also been shown (Tal'nov *et al.*, 1997) that the mean EMG activity in elbow flexors engaged in maintaining a desired elbow joint position differed depending on whether the joint position was reached by a joint flexion or extension movement. In this case, however, the alterations in the motor commands to the position-holding muscles were primarily considered to be consequences of history-dependent changes in the force-producing capacity of *recruited* muscle fibres (Abbott & Aubert, 1952; Herzog, 2004).

The above reports provided motivation for further investigations on history-dependent adjustments in the motor commands to muscles. If muscle thixotropy significantly influences the fine tuning of voluntary contractions, it certainly deserves more attention in studies of human neuromuscular control.

General aims of the studies

The principal aim of these studies was to investigate whether and to what extent voluntary contractions in human muscle are influenced by thixotropic changes in passive forces formed by the skeletal muscles.

Experiments were conducted to demonstrate how muscle thixotropy is expressed in the passive mechanical behaviour of the wrist joint. The main focus of this research, however, lies in voluntary contraction experiments. These experiments involved slow and rapid voluntary wrist joint movements (studies I and IV) and steady maintenance of the wrist joint position (studies II and III). The voluntary contraction experiments systematically addressed the motor control consequences of muscle thixotropy (as this was demonstrated in the relaxed wrist joint) for ordinary voluntary motor tasks.

Somewhat outside the main scope of this work, the studies also aimed at providing further information about the thixotropic behaviour of the muscles. Thixotropic after-effects were produced by so-called muscle-conditioning procedures which are commonly used, for instance, by physiotherapists and athletes (e.g. muscle stretching, massage and vibration), but which also occur in everyday life (e.g. joint movements *per se*).

The studies provide information about the duration of such thixotropic after-effects and their possible beneficial consequences for motor performance. In addition, the importance of considering thixotropic effects in other experimental settings is emphasised.

In studies I-III surface EMG was used to reflect thixotropy-dependent motor adjustments. In study IV, thixotropy-dependent changes were examined in movement characteristics of voluntary ballistic contractions.

Specific aims of the studies

The studies were carried out with the following specific aims:

- 1) To investigate whether the motor control of slow voluntary wrist movements is influenced by thixotropic changes in short-range muscle stiffness, and to examine the 'loosening-up' effect of different muscle-conditioning procedures on the short-range stiffness (Study I).
- 2) To elucidate motor compensations for thixotropic changes in resting wrist joint positions (Study II).
- 3) To determine how muscle thixotropy influences the motor control of steady maintenance of wrist joint positions (Studies II and III).
- 4) To find out whether post-movement slowly subsiding passive and motor after-effects are influenced by mechanical agitation of the forearm muscles (Studies II and III).
- 5) To seek evidence that muscle thixotropy also influences the performance of rapid, so-called ballistic, wrist flexor contractions (Study IV).

Methods

Subjects

The experiments in studies I-IV were carried out in 25 healthy students and laboratory staff. These volunteers generally participated in several studies. There were almost equal numbers of men and woman. Their mean age was 31 years. Informed written consent was obtained and the project was approved by the Ethics Committee of the Faculty of Medicine at Uppsala University (Dnr 99114) in accordance with the Declaration of Helsinki.

Experimental set-up

The experimental set-up is illustrated in Figure 2. With only minor modifications, the basic mechanical arrangements were the same in all studies. The ambient room temperature was 18-20° C. All trials were carried out on the left wrist joint. The subjects were seated in a comfortable chair, the height of which could be adjusted so that the shoulder joint was abducted 30-40 degrees while the relaxed forearm was supported by a table. The upper arm rested in a support and was held fixed with Velcro straps, which restrained movements along the axis of the forearm (see Fig. 2). The elbow was extended approximately 100°.

The distal forearm was placed in a U-shaped wooden block mounted on the table. With this arrangement, the wrist joint axis for extension and flexion movements was kept in the vertical plane (i.e. wrist joint flexion/extension movements were made in the horizontal plane). The groove between the two tendons of the extensor pollicis longus and brevis muscles at the wrist was used as an anatomical landmark to align the wrist joint to the rotational axis of a torque motor (TQ 34W -22 Aeroflex, Hauppauge, NY, USA).

In some experiments, a small rubber pad connected to a vertical steel bar was used to support the volar aspect of the radial styloid process, in order to avoid a tendency to pronation of the relaxed forearm (not shown in the figure).

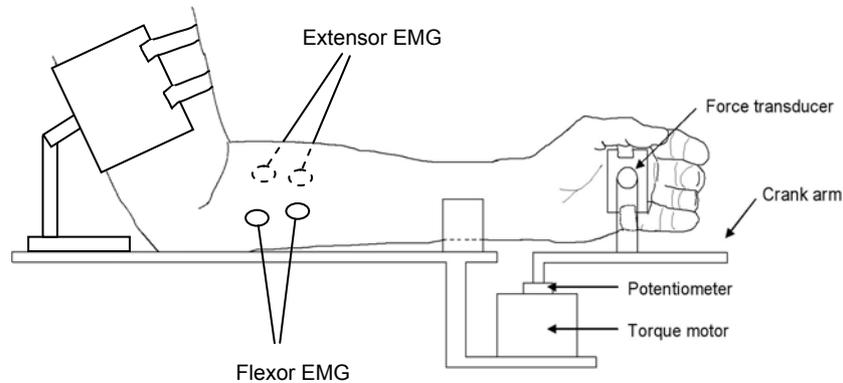


Figure 2. Schematic drawing of the mechanical set-up from a horizontal view

Passive force and joint angle measurements

Passive wrist joint torque was measured in studies I-III. A force transducer was mounted on the torque motor crank arm and placed 9 cm from the rotation axis. A rectangular wooden plate was connected to the transducer and firmly strapped against the palm. With this arrangement, passive pulling or pushing forces of the hand were sensed by the transducer via the wooden plate. Ulnar and radial deviations of the wrist joint were restricted by the straps that attached the wooden plate to the hand. The sensitivity of the torque signal was commonly tested by gently moving the crank arm a few degrees in the direction of either wrist dorsiflexion or volar flexion. Such imposed forces were expected to produce changes in the torque signal.

A different force transducer was used in studies I and III. This was fixed to a rigid external support and connected to a small rectangular wooden plate (9 x 33 mm) which was firmly pressed against the flexor tendons in the distal part of the forearm (e.g. the flexor carpi radialis tendon). The purpose of this arrangement was to measure the compliance of the flexor tendons during passive as well as active wrist dorsiflexions. It was considered that the force signal obtained would indirectly reflect the tension developed in the flexor muscles.

Angular displacements of the wrist joint were measured with a high-precision potentiometer connected to the axis of the torque motor. The motor was switched off in several tests and the crank arm could then be freely rotated within the physiological range of the joint.

EMG recordings

In all studies, surface EMG was recorded from wrist flexor and extensor muscles in order to quantify muscle contraction strengths. The EMG was also used to confirm relaxation, when so desired, by visual inspection of the EMG displayed on an oscilloscope screen and through feedback from loudspeakers. Two pairs of disposable surface EMG electrodes (Blue sensor, type F-10-VS, Medicotest, Ølstykke, Denmark) were placed over the bellies of the flexor carpi radialis and extensor carpi muscles. The electrodes were placed 5-6 cm apart in a proximal-distal direction for bipolar recordings. The proximal electrodes were placed ~5 cm distal to the lateral and medial epicondyles of the humerus (for extensor and flexor EMG recordings, respectively). The skin was rinsed with ethyl alcohol to lower impedance. A ground electrode was placed on the upper left arm.

Integrated surface EMG (IEMG) was recorded in studies I-III. The EMG signals were amplified (usually about $\times 2000$) and rectified. The rectified EMG was integrated with a time constant of 200 ms. In study IV, the signals were not integrated and the rectified EMG was primarily used to trigger the oscilloscope to obtain average signals of the EMG during repeated ballistic flexor contractions. The EMG signals were high-pass filtered (30 Hz). Amplification, rectification, integration and filtering of the signals were performed by analogous hardware previously designed for EMG measurements. Calibration was made with a function generator (300 Hz).

Muscle-conditioning procedures

Most of the experiments were typically preceded by a standardised preconditioning procedure. This consisted of high-amplitude flapping hand movements for a few seconds followed by total relaxation for at least 20 s. The angular wrist joint position after preconditioning was denoted as the '*original resting position*'. The purpose of the preconditioning procedure was to eliminate any possible mechanical after-effects of previous uncontrolled muscle activities.

The original resting position of the hand was commonly about 5-10° volarflexed relative to the angular position at which the dorsum of the hand was in line with the dorsum of the distal forearm.

Several different types of *specific* muscle-conditioning procedures were carried out throughout the studies. The majority of these procedures or *manoeuvres* have previously been used by others (Lakie & Robson, 1988a; Proske *et al.*, 1993) in investigations of skeletal muscle thixotropy. In agreement with findings in those studies, they were considered to produce thixotropic after-effects on passive muscle fibre stiffness and/or resting tension. Further, in agreement with the nomenclature used in connection with

thixotropy, they were sometimes denoted as ‘stirring’ procedures, giving rise to ‘mechanical agitation’ of the muscle fibres. The terms ‘loosening-up’ or ‘limbering-up’ manoeuvres were also used in this context. Thus, for example in study I, conditioning procedures in the form of flapping hand movements, isometric co-contractions, mechanical high-frequency vibration or manual massage of the muscles were considered to produce thixotropic ‘stirring’ of the muscles.

In study II, there were two types of muscle-conditioning procedures that were considered particularly effective in producing after-effects on resting tension of the extensor/flexor muscles. The wrist joint was displaced from its resting position to either a highly dorsiflexed or volar flexed position ($\sim 60^\circ$), which was maintained for about 5 s while the subject co-contracted the forearm muscles. Depending on the direction of the conditioning movements, they were denoted as either dorsiflexion or volar flexion conditioning. These procedures can also be regarded as forceful stretching manoeuvres of the flexors and the extensors, respectively.

Study III showed that much more moderate joint displacements (without forceful co-contractions) were sufficient to produce similar types of after-effects on muscular resting tension. This was the reason why the conditioning procedures in study IV, carried out for the same purpose, only involved a brief period of voluntary maintenance (without co-contractions) of a moderately flexed or extended wrist joint position.

Experimental designs: in general

The experiments were designed to investigate, as a first step, thixotropic changes in the passive mechanical behaviour of the wrist joint during imposed movements and steady maintenance of joint positions. Passive thixotropic changes were measured from the torque, flexor tension and goniometer signals recorded from relaxed subjects.

During the passive trials, joint movements and position holding were performed either by the torque motor or manual manoeuvring of the torque motor crank arm by the experimenter. The motor was driven by a power amplifier (BBC Axodyn 05LV09) which was fed by a function generator (HP331A). The output signal of the function generator could be set so that the motor generated a desired type of externally imposed torque. No servo control mechanism was available for automatic regulation of joint positions and the experimenter was thus required to follow the goniometer trace on the oscilloscope while holding the torque motor crank arm.

As a next step, the subjects were typically asked to execute movements or hold positions that corresponded to a certain passive test. Since only muscle activation of wrist flexors or extensors was required to perform a certain task, the subjects were specifically instructed to avoid finger movements and

to keep other arm muscles relaxed. Nevertheless, there were sometimes unexpected bursts of EMG activity, that could not be explained solely by wrist extension or flexion activity. In such cases, it was usually apparent that the electrodes picked up activity from nearby muscles. For example, unintentional activation of the brachioradial, finger extensor and flexor muscles sometimes occurred. Usually such disturbances could be avoided by giving the subjects more thorough instructions.

In order to avoid co-contractions, the subjects were asked to use minimal efforts. Except in study IV, all movements (including passive ones) were relatively slow ($0.5\text{-}7^\circ/\text{s}$) in order to avoid stretch reflexes. The subjects were usually given visual feedback of joint positions from the oscilloscope screen. As previously mentioned, surface EMG was used in studies I-III to observe whether changes in the EMG signal could be explained by motor compensations or adjustments for the passive thixotropic effects that had been demonstrated beforehand in the passive experiments.

Study IV differed from the other investigations (I-III) in that the aim was not to demonstrate motor *adjustments* for muscle thixotropy, but rather to determine how and to what extent muscle conditioning influences movement characteristics of ballistic contractions. The subjects were instructed to perform ballistic volar flexion contractions as fast as possible immediately following a certain muscle-conditioning procedure and a command 'go' signal. In the next chapter (Results including specific experimental designs), more detailed information is provided regarding the experimental protocols in each study.

Data acquisition and statistics

All recorded signals were displayed on a 4-channel digital oscilloscope (Le-croy 9314M, upgraded with 93XX-MWPM hardware and software v.08.2.2). The signals were either printed out directly by the oscilloscope screen dump unit, stored on floppy discs or transferred to a PC hard drive for offline analysis. Software tools integrated in the oscilloscope were used for signal measurements.

Non-parametric and parametric statistical evaluations were made and typically involved 'before-and-after' paired comparisons. Wilcoxon paired-sample test was used in studies I-III and paired-sample t-test in study IV. $P < 0.05$ was chosen as significance level.

Results and specific experimental designs

The main results from studies I-IV are presented below. For each study, the results are preceded by specific details about the experiments and are followed by concluding remarks. A thorough presentation of the results, together with statistical evaluations and illustrated details, are given in the original papers.

Study I

Experimental design

In the first experiments, a series of slow repetitive dorsiflexion movements was imposed on the relaxed wrist joint. Passive wrist joint torque or flexor tension was recorded during the trials. A detailed analysis of the observed short-range stiffness component was made from the torque/tension records. Particular attention was paid to the question of whether the short-range stiffness component varied in amplitude during the movements.

In corresponding voluntary contraction trials, the subjects were instructed to perform dorsiflexion movements similar to those in the passive tests. The initial rise of the IEMG from the movement generating extensors was analysed to see whether the shape of the IEMG signal varied similarly to the passive short-range stiffness.

Tests were also made to see whether the previously described (p. 19) 'stirring' manoeuvres were able to 'loosen up' the passive muscular resistance of the wrist joint. The torque motor delivered weak repetitive torque pulses towards dorsiflexion which produced small passive dorsiflexion movements. An intervening muscle-conditioning procedure was carried out by the subject and post-conditioning changes in movement amplitudes were studied. In corresponding contraction experiments, the subjects were instructed to produce repetitive wrist extensor contractions of constant IEMG strengths. The question was examined whether the voluntarily generated movements differed in amplitude after muscle conditioning.

In all passive trials, IEMG was recorded to verify that the wrist flexor/extensor muscles were relaxed.

Short-range stiffness in passive wrist joint torque

In relaxed subjects, passive wrist joint torque responses to imposed repetitive wrist dorsiflexions of equivalent slow speed and amplitude ($\sim 15^\circ$) typically exhibited a marked stiffness component for the first movement compared to succeeding movements (Fig. 3). The first torque response had a characteristically biphasic shape with a deflection point indicating the so-called elastic limit. This limit was commonly reached at a dorsiflexed position of 3-4°.

The passive torque resistance was evaluated at a dorsiflexed position of 2° and was significantly higher ($p=0.01$) for the first than for the second movement. The stiffness recovered during a 15 s resting period and there was no difference ($p=0.73$) in passive torque resistance at 2° between the first and the fourth post-rest movement.

As also seen in Figure 3, the torque signal crossed the zero torque level during the return movements. This indicated that after each imposed dorsiflexion, a force of opposite direction was required to bring the wrist back fully to its initial resting position. During the 15 s rest period this force gradually subsided.

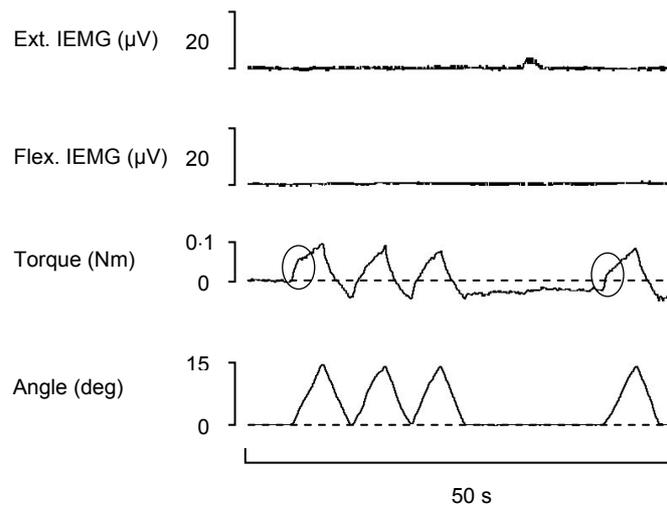


Figure 3. Passive torque resistance to imposed repetitive wrist dorsiflexions exceeding the limit for short-range stiffness. The dashed line in the torque trace indicates the zero torque level. There is a particularly steep rise in passive torque during the initial part of the first and fourth dorsiflexion movements (encircled). The level of resting torque gradually approaches the zero torque level during the resting interval. The subject is relaxed, as shown in the integrated EMG (IEMG) traces.

Short-range stiffness in passive flexor tension

A strain gauge was firmly pressed against the flexor tendons in the distal part of the forearm (see Methods on p. 18). The purpose of this arrangement was to confirm that the short-range stiffness demonstrated in the torque recordings mainly originated from the stretched relaxed wrist flexor muscles. It was found that during repetitive passive wrist dorsiflexions with an amplitude exceeding the limit for short-range stiffness, the flexor tension responses exhibited a marked similarity to the previously described torque responses, i.e. the tension responses varied in shape depending on whether the movement in question was or was not preceded by a period of rest (Fig. 4). Furthermore, the flexor tension responses exhibited another similarity with the torque responses: there was a decrease in resting tension when the hand was returned to the resting position after a preceding dorsiflexion, and when the consecutive movements ended the resting tension gradually recovered.

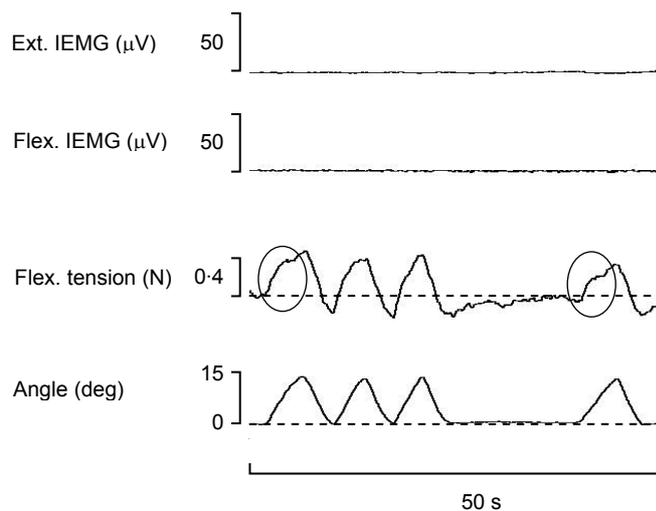


Figure 4. Passive resistance in the flexors to imposed repetitive wrist dorsiflexions. The short-range stiffness components in the first and fourth movements are encircled. The IEMG traces confirm that the forearm muscles are completely relaxed.

Motor adjustments for short-range stiffness

In analogous voluntary contraction experiments, the subjects were guided by the goniometer signal on the oscilloscope screen and instructed to make repetitive voluntary wrist dorsiflexion movements. As shown in Figure 5, the first voluntary movement in the series was accompanied by an initial increase in extensor EMG activity that was steeper than that in the two succeeding movements. The extensor IEMG amplitude was evaluated at 2° of dorsiflexion and was significantly higher ($p=0.01$) for the first than for the second movement.

It was also observed that in the same way as the short-range stiffness reappeared after a rest period of about 15 s, so did the initial increase in extensor EMG activity reappear after approximately the same rest interval. There was no difference ($p=0.94$) in extensor IEMG amplitude at 2° between the first and the fourth post-rest movement.

Figure 5 also shows that brief wrist flexor contractions were needed to restore the wrist completely to its original position and a more prolonged flexor contraction was needed to maintain the resting position after the final third dorsiflexion.

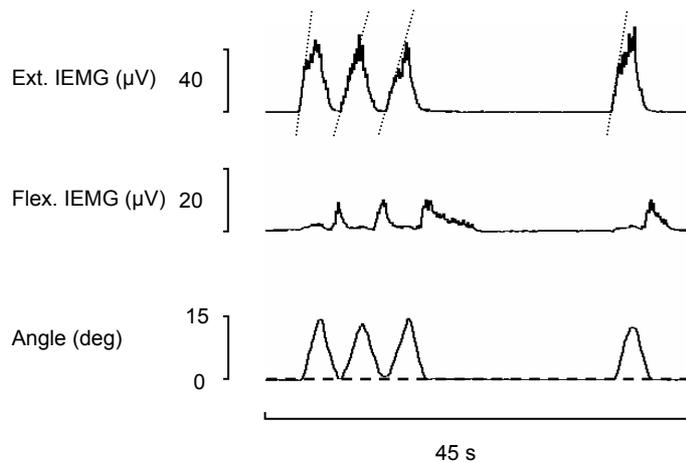


Figure 5. Muscle activation patterns during active repetitive wrist dorsiflexions. Dotted lines in the wrist extensor IEMG trace are drawn to demonstrate that the initial rise in EMG activity depends on whether the dorsiflexion movement is made after a period of rest or immediately after a similar dorsiflexion movement.

The EMG was also recorded simultaneously with the flexor tension signal. It was shown that the wrist extensor IEMG during repetitive voluntary wrist dorsiflexions varied in parallel with the simultaneously recorded flexor tension responses. The stronger the flexor short-range stiffness components, the more prominent were the initial steep up-going deflections in the extensor IEMG traces.

To sum up, one single wrist dorsiflexion movement exceeding the elastic limit of 3-4° was sufficient to reduce the passively opposing short-range stiffness of the flexor muscles. Consequently, less activation of the wrist extensors was required to produce succeeding dorsiflexion movements. The short-range stiffness recovered during a period of inactivity and this was compensated by stronger extensor contractions.

Apparently, a single dorsiflexion movement acted as a thixotropic 'stirring' event in the flexor muscles, 'loosening up' the inherent short-range stiffness.

The question was then addressed as to how other 'stirring' procedures affected the short-range stiffness of the muscles and whether they also had motor consequences.

Passive and voluntary movements generated by constant forces: the effect of stirring procedures

In relaxed subjects, constant repetitive torque pulses towards wrist dorsiflexion generated larger passive dorsiflexion movements after a brief period of flapping hand movements (120%). There was also a post-conditioning increase in movement amplitudes after vibration of the flexors (60%) and co-contractions of the forearm muscles (110%).

In a subsequent experiment, the subjects were allowed to watch the wrist extensor EMG trace on the oscilloscope screen. They were instructed to keep all movement-generating contractions (extensor IEMG amplitudes) equally strong before and after muscle conditioning. The experiments revealed that the voluntary dorsiflexion movements were larger after flapping hand movements (140%), vibration (45%) and co-contractions (170%). Examples of the results are shown in Figure 6.

It was observed that the chosen strengths of the torque pulses and contractions were critical. If the pre-conditioning movements were near or exceeded the limit for the short-range stiffness, gradual loosening-up effects sometimes occurred even before the actual stirring manoeuvres were carried out. Care was therefore taken to see that the pre-conditioning movements were kept below the elastic limit of 3-4° so that the movements themselves did not cause a reduction in short-range stiffness.

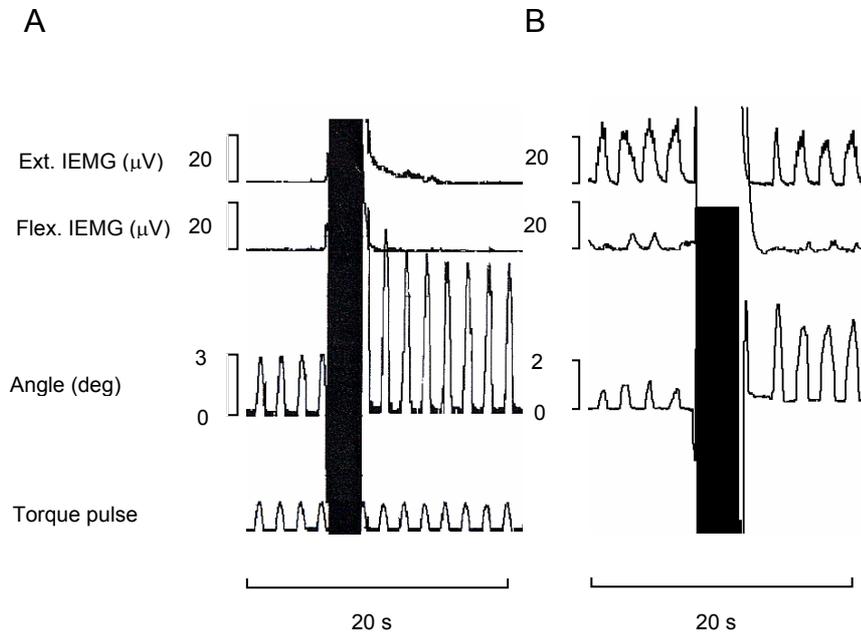


Figure 6. A, passive wrist dorsiflexion movements induced by weak repetitive torque pulses. B, dorsiflexion movements actively generated by repetitive weak extensor contractions of constant strengths. Note that the movement amplitudes are increased after high-amplitude rapid hand movements (A and B). The flapping movements overloaded the goniometer and are displayed in the figures as solid black rectangles.

Conclusion

The above-described experiments clearly indicated that the movement-generating capacity of voluntary wrist extensor contractions varies with the prevailing degree of passive short-range stiffness of unrecruited antagonistic flexors. It was shown that not only a single stretch movement, but also other types of ‘stirring’ muscle-conditioning procedures reduce the muscular short-range stiffness.

It was also demonstrated that the flexor resting tension was reduced after a single dorsiflexion movement, indicating a post-stretch reduction in passive restoring force from the flexors. This seemed to explain why external forces (Fig. 3) or voluntary flexor contractions (Fig. 5) were needed to restore and maintain the original position. Apparently, history-dependent after-effects may also be manifested as shifts in the resting joint position. Study II dealt with such after-effects and their motor compensations.

Study II

Experimental design

In this study, the muscle-conditioning procedures consisted of brief forceful co-contractions of the forearm muscles while the wrist joint was held near maximally flexed or extended (*volar flexion* and *dorsiflexion* conditioning, respectively).

As the subjects became fully relaxed after muscle conditioning, it was observed how the angular resting wrist joint position varied depending on the type of conditioning manoeuvre. The extent to which ‘stirring’ procedures (flapping hand movements or co-contractions) influenced post-conditioning shifts in the resting wrist joint position was also investigated.

The subjects were not allowed to watch the position of the hand or the goniometer trace on the oscilloscope screen in the above-described trials.

In subsequent experiments, the subjects were instructed to bring the hand fully back to, and maintain a predetermined intermediate wrist joint position after the conditioning manoeuvres. They were now guided by the goniometer trace on the oscilloscope screen. Analyses were made to see how the two types of conditioning manoeuvres affected the strength and time course of the wrist muscle activations required to voluntarily maintain an intermediate position.

Thixotropic changes in resting wrist joint position

The resting position of the unrestrained hand was shifted towards a relatively dorsiflexed position (median 11.2°) after ‘dorsiflexion conditioning’ and towards volar flexion (median 21.6°) after ‘volar flexion conditioning’. These were consistent findings in all subjects and are exemplified in Figure 7A and B. The subjects, having no visual feedback, were not aware of the fact that the resting position was different after, compared to before, the conditioning manoeuvres.

Brief periods of mechanical agitation induced by either forceful co-contraction of the forearm muscles (Fig. 7A) or vigorous hand movements (Fig. 7B) abruptly brought the hand back to its original resting position. On average, the original resting position was restored by 87 % immediately after a flapping manoeuvre and 35 % after co-contraction.

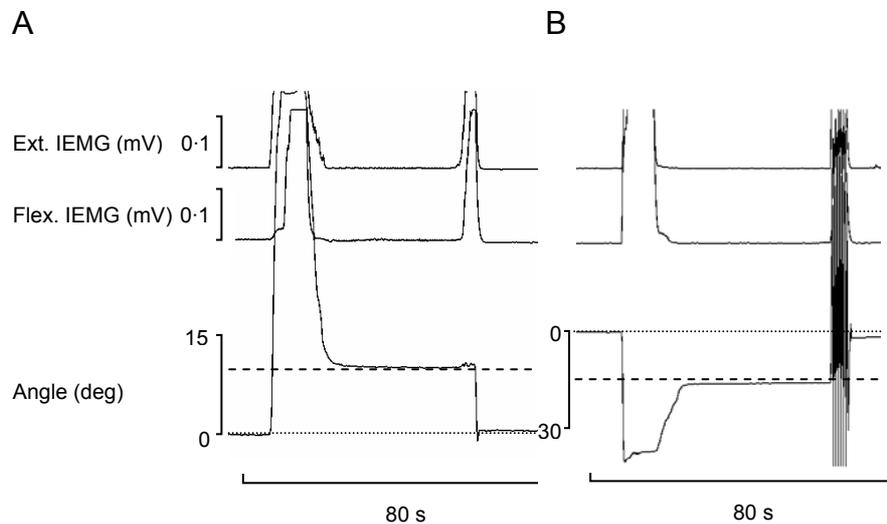


Figure 7. Shifts in resting joint position after dorsiflexion (A) and volar flexion (B) conditioning. Newly assumed resting positions after the conditioning procedures are indicated by the horizontal dashed lines. The original resting positions are indicated by the dotted lines. Strong co-contractions of the wrist muscles (A) or high-amplitude flapping hand movements (B) abruptly restored the original position.

Motor compensations for changes in resting wrist joint position

In a subsequent test, the subjects were instructed to voluntarily restore the wrist to the original resting position after the two types of conditioning procedures while being given visual guidance of the joint position from the oscilloscope screen. It was noted that the final part of the return movement after dorsiflexion conditioning was achieved by voluntary flexor muscle activation, which gradually declined while the original position was steadily held (Fig. 8A). Volar flexion conditioning had the opposite effect in the sense that extensor muscle contractions were required to return the hand to and maintain the original position (Fig 8B).

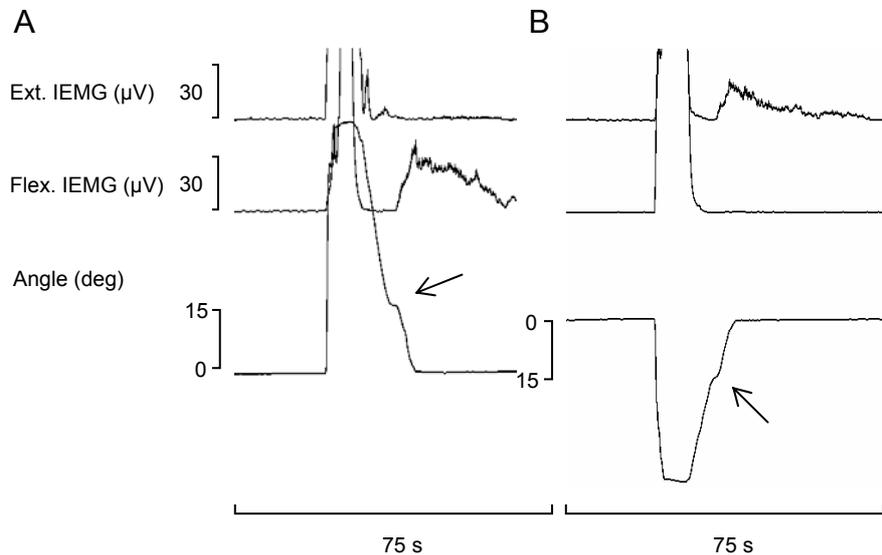


Figure 8. Flexor (A) and extensor (B) muscle activity is required to restore and maintain the original position after the dorsi- (A) and volar flexion (B) conditioning procedures. The small notches in the goniometer traces (arrows) represent a short pause at the newly obtained resting position preceding the voluntary return movements.

Motor adjustments during steady maintenance of a wrist position

In these experiments the subjects were instructed to 1) voluntarily and under visual guidance slowly move the hand from the original (unconditioned) resting position to a moderate (15°) dorsiflexed position, 2) maintain this position for about 20-30 s and then perform a dorsiflexion or a volar flexion conditioning procedure and finally 3) return the hand to the 15° dorsiflexed test position.

The typical EMG findings from the wrist extensors were as follows: The extensor IEMG activity slowly declined during a 20-30 s period prior to the conditioning procedure (Figs. 9A and B). When the test position was voluntarily restored after dorsiflexion conditioning, the IEMG level was reduced ($p < 0.01$) compared to the immediately pre-conditioning situation, and then slowly rose ($p < 0.01$) towards a steady level (Fig. 9A).

By contrast, when the test position was voluntarily restored after volar flexion conditioning, the extensor IEMG started from a comparatively higher ($p = 0.02$) level and declined. Examples of the latter findings are shown in Figure 9B.

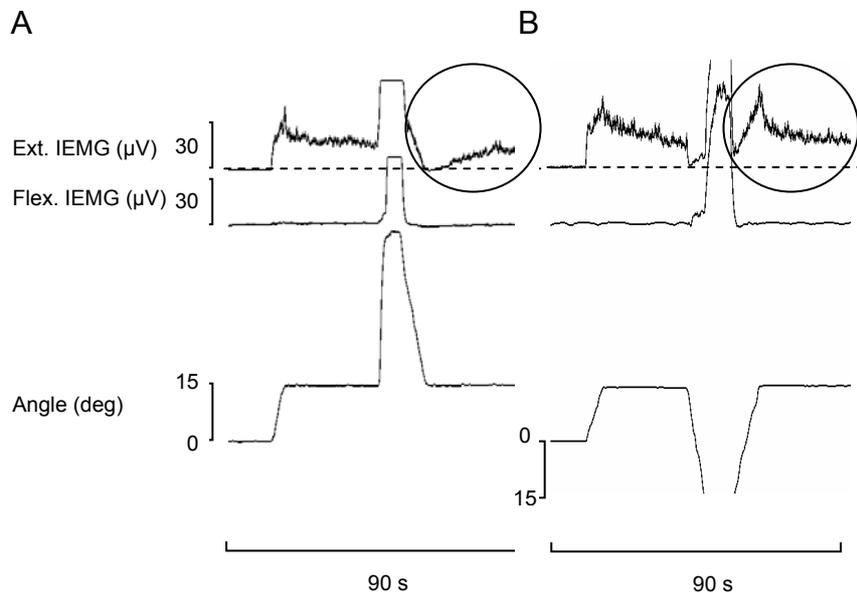


Figure 9. Extensor muscle activity recorded before and after dorsi- (A) and volar flexion (B) conditioning. For comparisons, post-conditioning differences in the strength of wrist extensor contractions required to maintain a 15° dorsiflexed position are encircled. Note that after dorsiflexion conditioning (A), no extensor muscle activity was initially required to maintain the test position in this particular case, indicating a shift in the resting position, which happened to correspond to the test position. Both figures show a gradual decrease in IEMG activity before conditioning.

Conclusion

The main outcome of this study was that the dorsi- and volar flexion conditioning manoeuvres had pronounced after-effects not only on the resting position of the wrist joint but also on the motor activity required to maintain a desired wrist joint position. The history-dependent shifts in the resting joint position appeared to be relatively long-lasting. However, the original position (or the original equilibrium point) of the wrist joint was quickly restored by mechanically agitating manoeuvres (co-contractions and flapping hand movements).

Attention was also drawn to the slowly declining extensor activity *preceding* the conditioning procedures while the wrist joint was held at a 15° dorsiflexed position (Fig. 9A and B). Naturally, the flexors exerted a passive internal load against the extensor contractions. A reasonable explanation for

the pre-conditioning IEMG decline was that the extensor activity was gradually adjusting to ongoing stress-relaxation in the stretched flexors. Apparently, a 15° dorsiflexion movement was sufficient to produce after-effects in the extensor IEMG very similar to those observed after the large dorsiflexion return movement made after volar flexion conditioning (Fig. 9B).

Study III concerned the question of whether, in general, such after-effects in passive muscular resistance and motor activity occur not only after rather extreme conditioning joint excursions (as shown in study II) but also after only moderate joint movements.

Study III

Experimental design

The majority of the experiments involved imposed or voluntary consecutive stepwise (7.5°) dorsiflexion movements followed by symmetrical return movements back to the starting position. Every single ramp-and-hold movement consisted of a relatively slow transitional movement followed by a 7-10 s period of steady position maintenance. Extensor and flexor IEMG were recorded in all experiments as well as torque or flexor tension in passive trials.

As demonstrated in study II, a brief period of forceful co-contractions removed passive mechanical after-effects manifested as changes in resting joint positioning. In a final type of experiment in study III, it was tested whether after-effects in passive torque and extensor IEMG produced by moderate changes in joint position were also influenced by co-contractions.

After-effects in passive torque during stepwise movements

Figure 10A displays the typical passive wrist joint torque response, in a relaxed subject, to imposed stepwise changes in wrist joint position within the dorsiflexion range. At any given newly obtained steady joint position, the torque signal slowly declined if the hand was previously held in a less dorsiflexed position. In contrast, the torque increased if the hand had previously been held in a more dorsiflexed position.

The torque signal crossed the zero baseline during the last return movements. In agreement with previous findings (study I and II), this implies that the resting wrist joint position would have been shifted towards dorsiflexion if no external forces had been imposed to counteract this shift.

As shown in Figure 10B, there was clear hysteresis in the torque signal, that is, at any given steady joint position the torque signal rose from a relatively lower level after a return movement.

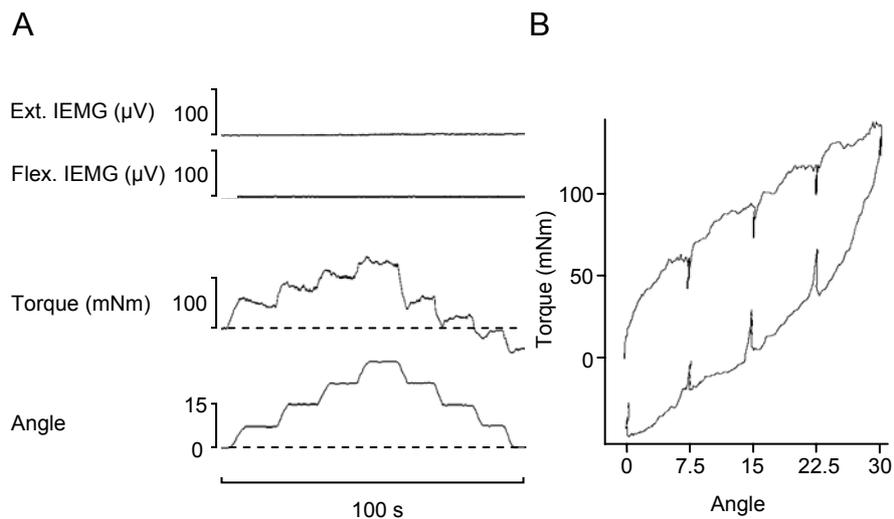


Figure 10. Passive wrist joint torque during stepwise movements. The dashed horizontal line in the torque trace represents the zero torque level. A, there is a time-dependent decrease and increase in joint torque depending on the previous position of the joint. B, torque vs. joint position hysteresis curve. Steep upward and downward deflections represent changes in passive joint torque at different steady joint positions.

Motor adjustments

After undergoing the passive tests, the subjects found it fairly easy, after some practice, to execute similar types of stepwise changes in joint positions. As shown in Figure 11A, the extensor IEMG amplitude was relatively higher and declined when a given steady position was reached from a less dorsiflexed position compared to that when the same position was reached by a movement from a more dorsiflexed position, whereafter the extensor IEMG was initially relatively lower and then increased.

Also illustrated in Figure 11A is the observation that flexor muscle contractions were required to finally return the hand to the starting position. The latter types of motor compensations were obviously necessary to overcome the passive opposing resistance encountered when the original resting position was restored.

Figure 11B displays a hysteresis curve which further depicts changes in extensor IEMG at steady positions.

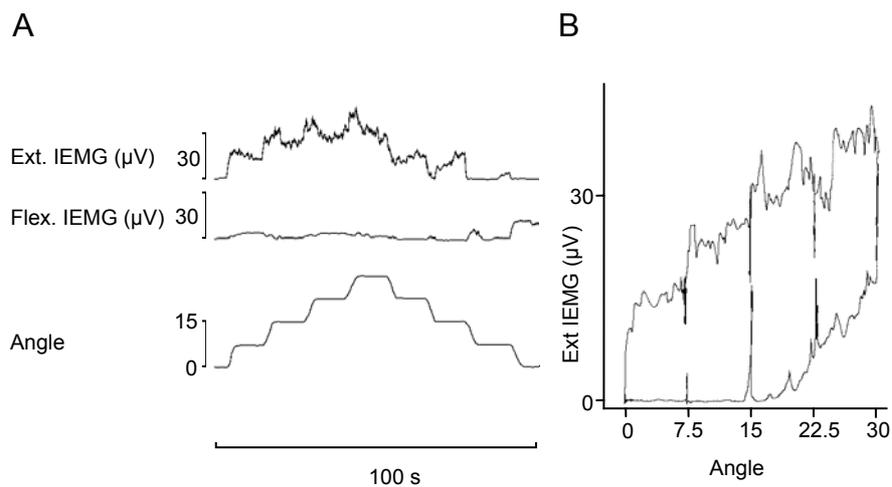


Figure 11. A, Variations in the extensor IEMG amplitude during stepwise changes in joint position. B, extensor IEMG vs. joint position hysteresis curve. The same general characteristics are seen as for the passive torque hysteresis curve (Fig. 10B), with steep upward and downward deflections representing changes in IEMG amplitude at steady positions.

Simultaneous recording of EMG and wrist flexor tension

It seemed reasonable to assume that the relaxed flexor muscles, which are reciprocally stretched or shortened by stepwise movements, contributed to the observed history-dependent changes in the wrist joint torque. To support this view, tests were made to determine the extent to which history-dependent changes in the isometric extensor IEMG correlated to simultaneously recorded changes in the antagonistic passive flexor tension. During these trials, the compliance of the flexor tendons was recorded from the distal forearm during stepwise changes in joint position (same method as in study I and described on p. 18).

As exemplified in Figure 12, the tension signal exhibited the same general characteristics as those found in corresponding torque measurements. Further, it was evident that gradual changes in isometric extensor IEMG occurred in parallel with changes in passive flexor tension during voluntary steady maintenance of a joint position. There was a strong correlation between extensor IEMG amplitude and flexor tension (Spearman's rank correlation, $r_s=0.91$, $p<0.01$).

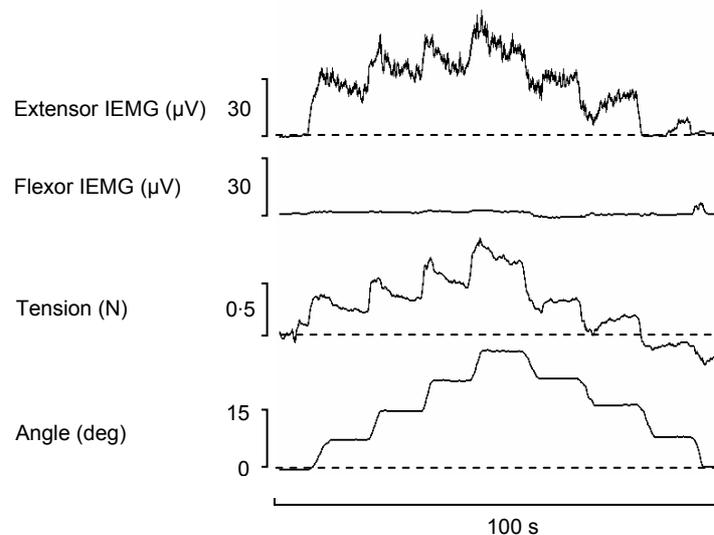


Figure 12. Simultaneous recordings of extensor IEMG and flexor muscle tension during the stepwise movements.

The effect of intervening ‘stirring’ co-contractions

On after-effects in passive torque

The wrist joint was held by the experimenter for about 40 s in either a moderately dorsiflexed or volar flexed position of 15° (final ~ 10 s shown in figures). After this conditioning period, an imposed transition movement was made to an intermediate test position.

The passive tests showed (in agreement with the previous findings) that the torque signal either declined or rose during a 40 s observation time depending on the previous position of the hand (Fig. 13A).

Figure 13B displays superimposed records of a test in which a subject is instructed to briefly (~ 5 s) and forcefully co-contrast the forearm muscles for a few seconds after the test position is reached. As shown, the torque signals remained fairly constant independent of the direction of the transition movements after the co-contractions, that is, history-dependent effects were no longer evident after the forceful co-contractions.

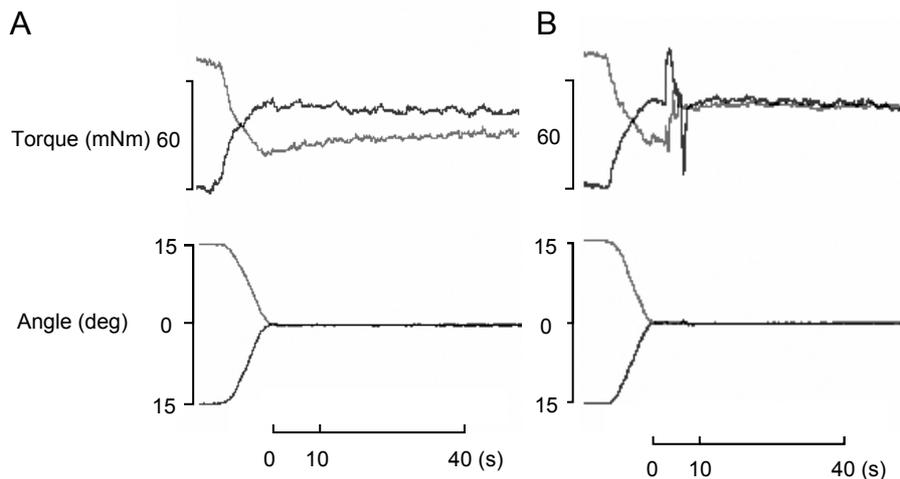


Figure 13. Post-movement changes in passive torque: the effect of intervening co-contractions. A, superimposed torque and goniometer signals (EMG traces omitted). Note the relative difference in passive wrist joint torque depending on whether the test position was reached by a dorsiflexion (black) or a volar flexion (grey) movement. B, intervening co-contractions after the test position is reached. Brief deflections in the torque signals occurred during the contractions. The torque level remains steady irrespective of the type of conditioning.

On after-effects in extensor IEMG

In analogous active tests, the torque motor was used to produce a sustained external volar torque, which had to be counteracted by extensor contractions in all relevant positions. The wrist joint position was voluntarily maintained in a dorsiflexed or a volar flexed position for ~40 s and then voluntarily changed to the intermediate test position. It was thus possible to study whether gradual motor adjustments in the extensor IEMG recordings were also influenced by co-contractions. Without intervening co-contractions, the extensor IEMG either slowly decreased or slowly increased in amplitude during steady maintenance of the test position, depending on how the test position was reached (Fig. 14A). However, after a brief period of co-contraction at the newly obtained test position this difference was no longer clearly seen (Fig. 14B).

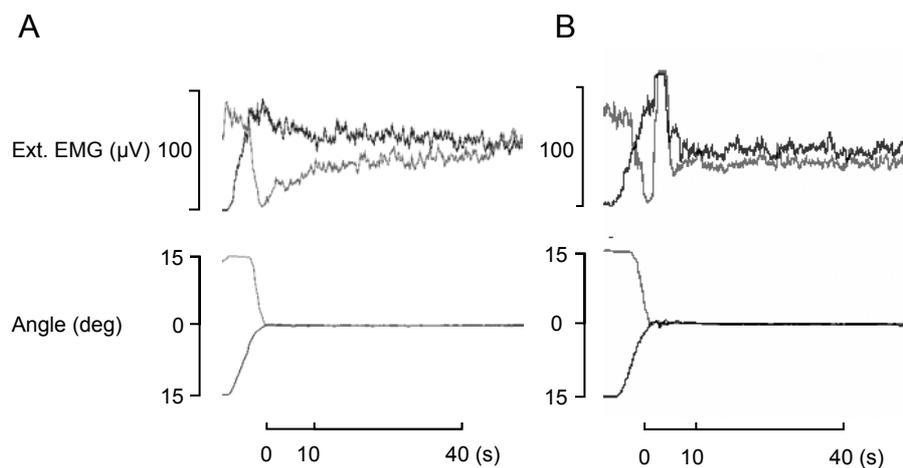


Figure 14. Post-movement passive changes in extensor IEMG: the effect of intervening co-contractions. A, superimposed extensor IEMG and goniometer signals. After a dorsiflexion movement (black) there was a gradual decrease ($p < 0.05$) in extensor IEMG and after volar flexion (grey) there was a gradual rise ($p < 0.05$). B, the co-contractions are shown as high-amplitude bursts in the EMG (truncated). After the co-contractions, there was no change in IEMG ($p = 0.02$ and $p = 0.86$ post-dorsiflexion and volar flexion, respectively).

Conclusion

Study III was regarded as a continuation of study II and provided evidence that moderate transitional joint movements are sufficient to produce history-dependent motor adjustments. Further, it was demonstrated that the slowly subsiding passive and motor after-effects were effectively eliminated by a brief period of forceful co-contractions.

Studies I-III concerned motor adjustments for thixotropic changes in passive forces acting on the wrist joint. The studies dealt specifically with relatively slow transitional movements and maintenance of wrist joint positions. With the intention of covering a greater range of commonly occurring motor activities, a final study (IV) was carried out to determine whether muscle thixotropy influenced the performance of rapid movements.

Study IV

Experimental design

In this study, comparisons were made between movements produced by nerve stimulation and movements generated by ballistic contractions after different types of muscle-conditioning routines.

In passive trials, the median nerve was stimulated by single shocks at the elbow in order to produce twitch flexion movements. The nerve was stimulated immediately after a muscle-conditioning manoeuvre. The twitch movements were usually about 50 times faster than, for instance, the passive movements in study I.

In the ballistic contraction trials, the subjects were instructed to hit a small metal plate by wrist volar flexion as quickly as possible in response to an imperative auditory 'go' signal. The metal plate (covering a shock-absorbing rubber pad) was placed so that the volar flexion movement was about 45° in amplitude relative to the starting position. The 'go' signal was preceded by a muscle-conditioning procedure.

Four different conditioning procedures similar to those performed in the previous studies (I-III) were carried out:

- 1) 40 s rest (REST).
- 2) 10 s of voluntary oscillating wrist joint movements (OSC).
- 3) Isometric 25° wrist extensor contractions (EXT).
- 4) Isometric 25° flexor contractions (FLEX).

The OSC procedure highly resembled the 'flapping' procedure performed in the previous studies (I-II). Differences in movement parameters were compared between OSC and REST and between FLEX and EXT. In addition, the so-called premotor reaction time (PMRT) was measured in voluntary contraction experiments.

The test movements were opposed by a small background torque. All movements (both passive and active) started from the same intermediate joint position.

Passive twitch movements and voluntary ballistic movements

OSC vs. REST

Figure 15A shows two superimposed records from the same individual in which the twitch movements were preceded either by oscillating hand movements (OSC) or by a resting period (REST). As illustrated (Fig. 15A), there was no difference ($p=0.20$) in the time delay between nerve stimulation and movement initiation (stimulus motor reaction time, sMRT) between the two conditioning procedures. Movement amplitude and peak velocity were higher ($p<0.01$) after OSC.

Figure 15B shows two superimposed voluntary ballistic movements after the same conditioning routines. There was no difference ($p=0.83$) in the time delay between flexor EMG onset and movement initiation (motor reaction time, MRT). Also in agreement with the passive tests, movement peak velocity was significantly higher ($p=0.02$) after the OSC procedure. The time that elapsed during the movement (movement time, MT) clearly tended to be reduced ($p=0.06$).

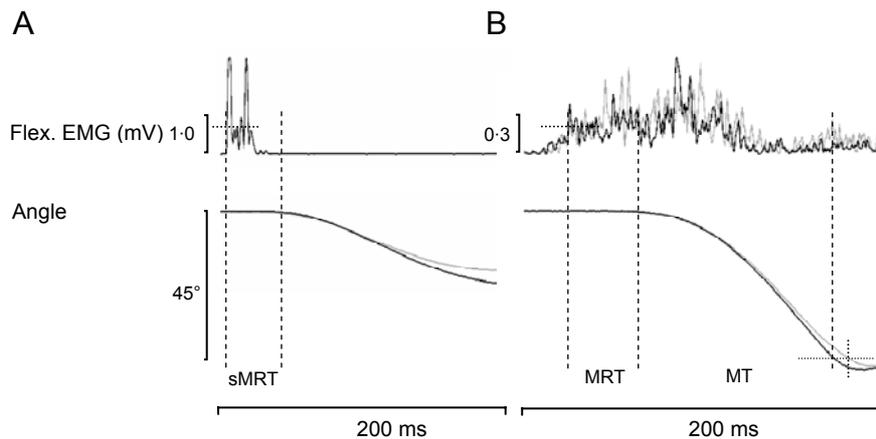


Figure 15. Superimposed signals after oscillating wrist movements (OSC; dark) and after a resting period (REST; grey). Vertical dashed lines indicate limits for OSC variables. Horizontal dotted lines in flexor EMG traces represent oscilloscope trigger levels. A, passive volar flexion movements in one subject after OSC and REST. There is no difference in movement initiation (stimulus motor reaction time, sMRT), but after OSC the peak amplitude is higher. B, ballistic contractions in one subject after OSC and REST. Dotted horizontal line in goniometer trace indicates stop position. The goniometer 'overshoot' is due to elasticity of the rubber pad. There is no difference in motor reaction time (MRT). The stop position is reached earlier after OSC. Difference in movement time (MT) is indicated with a vertical dotted line.

FLEX vs. EXT

Comparisons were also made between passive movements elicited by nerve stimulation after the wrist joint had been voluntarily held in either a flexed (FLEX) or extended (EXT) position of $\sim 25^\circ$ (Fig. 16A). After FLEX, sMRT was significantly shorter ($p < 0.01$) and the movement amplitude and peak velocity were significantly higher ($p < 0.01$, for both).

Figure 16B shows two superimposed voluntary ballistic movements after the same types of conditioning routines. Movement onset (MRT) was earlier ($p = 0.01$) after FLEX but in contrast to the passive test, peak velocity was reduced ($p = 0.02$) after FLEX. There was no significant difference in MT ($p = 0.20$).

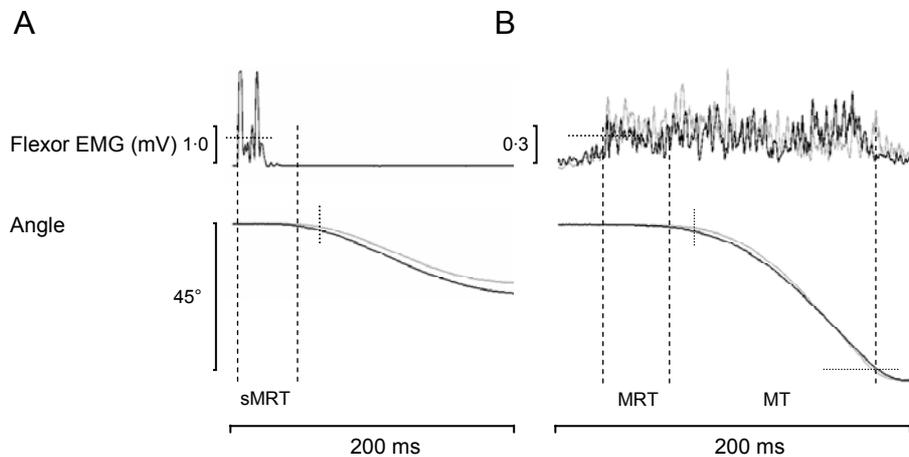


Figure 16. Superimposed records after isometric wrist flexor contractions (FLEX; dark) and extensions (EXT; grey). Vertical dashed lines indicate limits for FLEX variables. The horizontal dotted lines in the flexor EMG traces represent oscilloscope trigger levels. Vertical dotted lines in the goniometer traces indicate differences in sMRT (A) and MRT (B). A, passive volar flexion movements from one subject after FLEX and EXT. After FLEX, sMRT is shorter and peak amplitude is higher. B, ballistic contractions in one subject after FLEX and EXT. After FLEX, MRT is shorter.

Repetitive passive twitch movements and voluntary ballistic movements

Study I had demonstrated that passive opposing muscle stiffness was higher in the first than in the second movement in a series of repetitive wrist dorsiflexion movements. In study IV a similar protocol was used.

The mean amplitude and peak velocity for the first, compared to the second, in a series of three consecutive passive twitch movements were relatively lower (amplitude, $p < 0.01$; peak velocity - not significant). Data indicated a 'loosening-up' effect of the first passive movement on the next one in a series.

In corresponding active experiments the subjects were given three repetitive 'go' commands for ballistic contractions. The peak velocity was lower ($p < 0.01$) in the first ballistic movement than in the second. Apparently, a single ballistic movement made after a period of muscle relaxation is sufficient to produce a faster immediately succeeding movement.

Premotor reaction time

The premotor reaction time was calculated by measuring the time delay between the 'go' signal and the onset of the first extensor EMG burst. In addition, the total time taken to reach the stop position after the 'go' signal (GO-STOP) was calculated by adding the PMRT to the 'peripheral' parameters MRT and MT.

The GO-STOP interval was 31 ms shorter after OSC than after REST ($p = 0.01$), and PMRT was 25 ms shorter ($p = 0.01$). In relative terms, 80% of the reduction in GO-STOP latency after OSC was due to reduction in PMRT and the remaining 20% was due to a 6 ms shorter MT.

The GO-STOP interval was 32 ms shorter after FLEX than after EXT ($p = 0.04$). The PMRT was 31 ms shorter ($p = 0.04$), indicating that most (if not all) of the reduction in GO-STOP latency after FLEX was caused by reduction in PMRT.

Conclusion

Study IV demonstrated history-dependent variations in movement characteristics of voluntary ballistic contractions very similar to those observed after nerve stimulation. The experiments indicated that the movement-generating capacity of voluntary ballistic contractions is enhanced by thixotropic stiffness reduction (as judged by the OSC vs. REST comparisons and the repetitive ballistic movement trials).

The experiments on relaxed subjects showed that twitch movements had an earlier onset if the muscles had been conditioned beforehand by volar flexion compared to dorsiflexion (FLEX vs. EXT). Similar observations

were made in corresponding ballistic contraction trials. These findings indicate that history-dependent changes in the resting tension of the muscles influence the so-called electromechanical delay, i.e. the time delay between muscle activation and the following mechanical response. However, as regards movement speed the results were discordant, which was difficult to explain.

Somewhat unexpectedly, there was a comparatively pronounced difference in central nervous reaction time depending on the type of muscle conditioning procedure. Alternating hand movements (OSC vs. REST) and volar flexion conditioning (FLEX vs. EXT) caused a reduction in PMRT. A decrease in PMRT seemed to be more important than history-dependent changes in the peripheral resistance for the overall performance of ballistic contractions.

General discussion

The results of studies I-IV support the underlying assumption put forward in this thesis, i.e. that history-dependent changes in passive forces acting on the joints have consequences for human voluntary muscle contractions.

Studies I-III revealed motor adjustments during voluntary wrist joint movements and position maintenance that corresponded to history-dependent alterations in passive stiffness and resting tension of the forearm muscles. Study IV provided evidence that passive history-dependent changes in the muscles also influenced the performance of ballistic contractions.

Studies II and III confirmed some previous reports about history-dependent motor adjustment during isometric contractions (Hagbarth *et al.*, 1995; Nordin & Hagbarth, 1996; Tal'nov *et al.*, 1997; Welter & Bobbert, 2001). Some of these studies (e.g. Hagbarth *et al.*, 1995) suggested that post-conditioning motor adjustments were the consequence of passive thixotropic changes in extrafusal muscle fibres. In contrast, other authors (e.g. Welter & Bobbert, 2001) point out that history-dependent changes in *recruited* muscle fibres are especially relevant in this context. The latter deductions were made from earlier observations that the force generating capacity of contracting muscle fibres at a given length depends on whether the fibres were shortened or lengthened beforehand (Abbott & Aubert, 1952).

It cannot be ruled out that history-dependent variations in recruited fibres also to some extent gave rise to motor compensations in the present studies (II-III). Nevertheless, the voluntary contractions performed by the subjects required only minor motor efforts (except in study IV). These types of contractions presumably lead to recruitment of only a small fraction of available motor units in the muscles, suggesting that history-dependent changes in passive parts of the muscles were particularly important for the observed motor adjustments.

Mechanisms

The present studies were not designed to unravel the cellular and molecular mechanisms underlying the history-dependent behaviour of the muscles. Further, it remains unclear how the motor control system modified central motor commands according to the immediate mechanical state of the muscles.

The objectives of the passive experiments and the muscle-conditioning procedures were primarily to produce different types of after-effects in passive muscle mechanics and then to attempt systematically to determine whether the motor system adjusted to post-conditioning changes in the passive structures. Results from the passive experiments corroborate previous observations and interpretations made by others regarding history-dependent mechanisms of the muscles.

History-dependent properties of the skeletal muscles

Although it could not be excluded that other tissues besides the muscles may have contributed to the observed passive after-effects, there were clear indications that many of the conditioning procedures primarily involved the skeletal muscles. For instance, the short-range stiffness response (study I, Figs. 3&4) - unique for skeletal muscles - was loosened up by conditioning procedures which mainly affected the muscles (vibration, isometric co-contractions and massage).

It is generally accepted that muscular passive forces largely emanate from the muscle sarcomeres. Within the sarcomeres, weakly attached actin-myosin cross-bridges and titin filaments seem to be the main sources of passive force production and responsible for the history-dependent behaviour of the muscles (Whitehead *et al.*, 2001).

Cross-bridges

As originally proposed by Hill (1968), weak but 'long-lasting' bonds between actin and myosin filaments are spontaneously formed within the sarcomeres under resting conditions. In simplified terms, formations of stable cross-bridges in relaxed muscles imply that the muscle fibres tend to be 'stuck' at a particular length and 'released' by imposed forces detaching the cross-bridges.

Such a mechanism explains why a wrist joint movement exceeding a certain angular limit breaks up the short-range stiffness and furthermore, why other types of thixotropic agitating procedures release the muscles from their stiffened state. In contrast, if imposed movements are made within the short-range limit, this particular stiffness remains intact (Fig. 6A in Study I; Lakie & Robson, 1988a).

A cross-bridge mechanism can also explain why the resting position of the wrist joint was shifted after the high amplitude conditioning procedures performed in study II. For example, while reaching a highly dorsiflexed position, cross-bridges detach in the stretched flexors. Forceful co-contractions at the highly dorsiflexed position further detach any remaining bonds that were not detached by the movement. As the subject became relaxed in the highly dorsiflexed position, new cross-bridges were allowed to form in the muscles. As a consequence, the muscles became 'fixed' in the dorsiflexed

position and pure elastic restoring forces in the flexors were not sufficiently strong to return the wrist joint to its original starting position. However, a brief period of co-contractions or flapping hand movements quickly detached the newly formed bonds and the original position was restored (Fig. 7 in Study II; Hagbarth *et al.*, 1985).

Study III showed that when a new joint position was reached, the muscles gradually became adapted to the newly assumed position, i.e. stress relaxation occurred in stretched muscles and stress-recovery in those muscles that were shortened. It is unclear to what extent such processes can be attributed to cross-bridge interactions, but it was of interest to note that the passive adaptation processes were accelerated by co-contractions (Fig. 13). As previously mentioned, such procedures effectively detach cross-bridges and perhaps this may explain the sudden change in passive resistance.

Titin

As more information rapidly accumulates about the giant sarcomere protein titin, the role of cross-bridges in connection with passive muscle force may well need to be re-evaluated. Titin has been declared to be the major contributor to the elastic behaviour of the skeletal muscles (Linke, 2000). The titin protein spans over half of the sarcomere, connecting the Z band to the myosin filaments.

Of particular interest are those domains of the titin protein that unfold during stretch and recoil during shortening. Titin extracted from rodent muscle exhibits many of the mechanical phenomena that can be demonstrated in the whole muscle (Minajeva *et al.*, 2001; Tskhovrebova & Trinick, 2002). Based on a study on muscle-tendon preparations from the cat, Whitehead *et al.* (2001) proposed that the history-dependent fraction of muscular resting tension is partly explicable in terms of cross-bridge formations, but that the mechanical properties of titin filaments play an important role particularly at longer muscle lengths (beyond the optimal lengths for active tension).

The term thixotropy does not seem to be used, for example, by those working on the mechanical properties of titin. Presumably, this only reflects the fact that different terms are used depending on the particular author's view of the mechanism underlying the obtained results. As pointed out previously (Introduction), in studies I-IV the term thixotropy was used in connection with all types of history-dependent after-effects irrespective of their possible origin. The cross-bridge explanation given above served as a valuable 'working model' and thixotropy as a 'working term' in the design of the experiments and the interpretation of the results.

Motor control

In studies I-III, the subjects were given visual feedback about the prevailing wrist joint position while they performed movements and held steady joint

positions. Although the motor tasks were naturally executed by voluntary commands to the muscles, this does not mean that these commands were also responsible for the history-dependent adjustments. The motor adjustments were considered, rather, to be reflexogenic, as they occurred without the subjects being consciously aware of them.

Most likely, muscle spindles provide the central motor system with information about ongoing history-dependent changes in the muscles. Many previous studies have demonstrated how the spindles signal such information through peripheral sensory nerve afferents (Matthews, 1972; Gregory *et al.*, 1986; Hagbarth, 1993; Haftel *et al.*, 2004). Additional information may also be given by the Golgi-tendon organs, which sense the prevailing degree of tension in the muscles.

As regards the ballistic movements (study IV), it was uncertain whether the contractions were modified by proprioceptive feedback concerning history-dependent variations in passive resistance. It was of interest to note that the reaction time required to initiate a ballistic movement was greatly influenced by the type of muscle-conditioning procedure. This may indicate that centrally arriving afferent activity influences the central preparation of ballistic contractions.

Methods

EMG

Integrated surface EMG is a well-established method of measuring the strength of voluntary muscle activity (Bigland-Ritchie, 1981). The amplitude of the surface IEMG signal is determined by various factors, such as the number of recruited extrafusal muscle fibres, their size and firing rate and the impedance between the recording electrodes and the muscle. All of the above mentioned factors may explain the noticeable differences in integrated EMG amplitudes among the subjects. Further, the differences in IEMG amplitudes may reflect inter-individual differences in passive opposing muscle resistance. The latter is presumably influenced by for instance, age, gender and physical training.

It is well known that the isometric IEMG amplitude increases during fatiguing submaximal voluntary contractions owing to progressive failure in the excitation-contraction coupling process (Gandevia *et al.*, 1995; Axelson *et al.*, 2002). The rise in extensor IEMG amplitude observed after a dorsiflexion conditioning procedure (Fig. 9A) could perhaps in some other types of experimental settings be misinterpreted as a sign of muscle fatigue, but as shown (Fig. 9B), the time-dependent change of the IEMG was completely the opposite after a volar flexion conditioning procedure, that is, the amplitude decreased.

Force measurements

The way in which the passive joint torque was measured in studies I-III (methods p. 18) proved to be sensitive in detecting very small changes in joint torque.

Regarding *in situ* passive muscle tension, it seems difficult to obtain such information without surgical exposure of the tissues. In order to obtain indirect information about the prevailing degree of muscle tension, a specific method was devised for this purpose (methods p. 18). There were no indications that the force transducer that was pressed against the tendons hampered their normal sliding movements, and the joint mobility seemed to be unchanged. Naturally, passive tensile forces of the skin and underlying connective tissue are also sensed by the force transducer and may add to the observed history-dependent changes.

Unfortunately, it was not possible to record tension responses from the wrist extensor tendons. The tendons are located so closely over underlying bone that in contrast to the wrist flexor tendons they do not yield in the same way under the pressure exerted by the force transducer, but are compressed against the bone. It is highly reasonable to believe, however, that passive muscle fibres in the two antagonistic muscles have similar mechanical behaviour.

Implications

Thixotropic stiffening of the muscles provides the limbs with an inherent resistance against movements. The relaxed muscles thus act to preserve the prevailing joint position. This mechanism is certainly favourable for postural maintenance in the sense that passive forces lower the demand for muscle activation. Indeed, it has previously been shown that postural stability requires a surprisingly small amount of muscle activity (Joseph & Williams, 1957).

When joint positions are continuously changing, for example during rhythmical movements, the short-range stiffness is not able to recur. Only the first movement requires some extra muscle activation (Fig. 5) but for the succeeding movements, less muscle activity is required.

Muscle thixotropy in connection with postural maintenance and continuous joint movements seems to reduce the overall energy expenditure for the 'organism'. It is therefore easy to understand why the motor control system has developed strategies to handle thixotropy-dependent changes in the peripheral resistance. As demonstrated in this thesis, the motor control system effectively adjusts for such types of changes.

The muscle-conditioning procedures also require some comment. They were of the types commonly performed in sports and in physiotherapy. A number of mechanisms have been proposed to explain their beneficial effects (Bishop, 2003), and muscle thixotropy is certainly relevant in this context. However, an after-effect of muscle conditioning in the form of thixotropic stiffness reduction appears to be short in duration, ranging from seconds to some minutes (Figs. 3&4 in study I; Lakie *et al.*, 1984; Lakie & Robson, 1988a). It is therefore difficult to attribute any long-term reduction in muscular stiffness to this mechanism, an effect which would be particularly desirable from a clinical point of view (e.g., in muscle pain, contractures, etc.).

Muscle conditioning is perhaps even more interesting with respect to its immediate effect on motor performance. In study IV it was demonstrated that the speed of ballistic movements is increased following a brief period of thixotropic ‘stirring’ hand movements. This may explain why many athletes keep their muscles in motion in their preparation for ballistic actions (see also Castellote *et al.*, 2004).

Finally, for those working with other types of experiments involving passive muscular forces, it is important to be aware of the history-dependent behaviour of the muscles. In the present studies, most trials were preceded by a ‘pre-test’ conditioning procedure consisting of alternating hand movements in order to diminish any history-dependent after-effects unknown to the experimenter.

Future perspectives

Many issues remain to be further explored. For instance:

- It is unclear in what way muscle pathology influences the thixotropic behaviour of the muscles. Are there any specific clinical problems related to muscle thixotropy?
- It is well known that in patients with disturbed proprioception (e.g. peripheral nerve disease), appropriate information from the end organs is not adequately received by the central nervous system, which may lead to difficulties in maintaining stable joint positions. Does the lack of proprioceptive information about history-dependent changes in the muscles impose a particular problem for these patients?
- Muscle thixotropy seems to be influenced by muscle temperature (Lakie *et al.*, 1986). Passive muscle stiffness increases with lower temperature. In what way and to what extent is motor performance affected?

Sammanfattning på svenska (Summary in Swedish)

Skelettmuskulatur hos djur och människa uppvisar s.k. tixotropa egenskaper. I förenklade termer innebär detta att musklernas passiva motstånd mot sträckning varierar beroende på vad som omedelbart tidigare skett med musklerna. I exempelvis idrottssammanhang används olika 'uppmjukningsmetoder' för att minska musklernas passiva motstånd mot rörelser. Omvänt är det en vanlig erfarenhet att muskulaturen spontant blir 'stelare' under inaktivitet.

I många situationer utgör passivt muskelmotstånd en kraft som måste övervinnas under viljemässiga muskelkontraktioner. Detta motstånd är inte konstant vid en given ledposition utan kan p.g.a. muskeltixotropi variera i styrka från ett tillfälle till ett annat. Detta är känt sedan tidigare.

Det är däremot till stora delar okänt om tixotropa förändringar i passivt muskelmotstånd har någon betydelse för motoriska kontroll hos människa. Om man exempelvis avser att lyfta armen till en viss exakt position är det rimligt att anta att den mängd muskelaktivitet som krävs för en sådan enkel uppgift måste anpassas till tixotropa förändringar i passivt muskelmotstånd. Avhandlingen avsåg att belysa om en sådan finmotorisk reglering av muskelaktiviteten verkligen sker hos människa.

I avhandlingen ingår fyra arbeten (I-IV) som ger en systematisk beskrivning av det passiva muskelmotstånd som verkar runt handleden och hur detta motstånd kan variera beroende på vad som omedelbart tidigare skett med underarmsmuskulaturen. Förändringar i passivt muskelmotstånd mättes under långsamma och snabba handrörelser (I och IV) och under konstant positionshållning av handen (II och III).

Erhållna resultat från friska försökspersoner (Arbeten I-III) visade att det ständigt krävs en finreglering av muskelaktiviteten för att anpassa styrkan av viljemässiga kontraktioner till tixotropa förändringar i passivt muskelmotstånd. Denna reglering är omedveten och sker sannolikt i det centrala nervsystemet där viljemässiga motoriska kommandon från 'högre centra' modifieras av inkommande information från perifera receptorer. Arbetet IV visade att vår förmåga att utföra maximalt snabba rörelser påverkas av variationer i passivt muskelmotstånd men att förändringar i centralnervös reaktionstid kanske är en viktigare faktor.

Det är ännu okänt om det motoriska kontrollsystemet lika effektivt kan kompensera för de mekaniska förändringar som uppstår i muskulaturen i samband med vissa neuromuskulära sjukdomar, temperaturförändringar i muskeln, immobilisering eller normalt åldrande.

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