



NEUROMUSCULAR ASPECTS OF SPORT PERFORMANCE

VOLUME XVII OF THE ENCYCLOPEDIA OF SPORTS MEDICINE

AN IOC MEDICAL COMMISSION PUBLICATION



EDITED BY

PAAVO V. KOMI PhD

 **WILEY-BLACKWELL**

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Contents

- List of Contributors*, vii
- Foreword*, ix
- Preface*, x
- 1 From Isolated Actions to True Muscle Function, 1
PAAVO V. KOMI
 - 2 Stretch-Shortening Cycle of Muscle Function, 15
PAAVO V. KOMI AND CAROLINE NICOL
 - 3 Utilization of Stretch-Shortening Cycles in Cross-Country Skiing, 32
ROBERT NORMAN, VESA LINNAMO, AND PAAVO V. KOMI
 - 4 Neuromechanics of the Cycling Task, 52
ROBERT J. GREGOR AND W. LEE CHILDERS
 - 5 Kinetics and Muscular Function in Alpine Skiing, 78
ERICH MÜLLER, JOSEF KROELL, CHRISTIAN SCHIEFERMÜLLER, AND JAMES M. WAKELING
 - 6 Kinetics and Muscular Function in Ski Jumping, 91
MIKKO VIRMAVIRTA AND PAAVO V. KOMI
 - 7 Mechanical Efficiency of SSC Exercise, 103
HEIKKI KYRÖLÄINEN AND PAAVO V. KOMI
 - 8 Transcranial Magnetic Stimulation as a Tool to Study the Role of the Motor Cortex in Human Muscle Function, 115
JANNE AVELA AND MARKUS GRUBER
 - 9 Contribution of In-Vivo Human Tendon Force Measurements for Understanding Tendomuscular Loading During SSC, 135
PAAVO V. KOMI
 - 10 Ultrasound as a Tool to Record Muscle Function During Normal Locomotion, 150
MASAKI ISHIKAWA AND PAAVO V. KOMI
 - 11 Neuromechanical Loading of Biological Tissues, 164
GERT-PETER BRÜGGEMANN
 - 12 Stretch-Shortening Cycle Fatigue, 183
CAROLINE NICOL AND PAAVO V. KOMI
 - 13 Training Adaptation of the Neuromuscular System, 216
JACQUES DUCHATEAU AND STÉPHANE BAUDRY

- | | |
|--|---|
| <p>14 Control and Training of Posture
and Balance, 254
WOLFGANG TAUBE AND ALBERT GOLLHOFFER</p> <p>15 Sport Performance in Master Athletes:
Age-Associated Changes and Underlying
Neuromuscular Factors, 270
HARRI SUOMINEN AND MARKO KORHONEN</p> | <p>16 Rehabilitation of Overuse Tendon Injuries and
Ligament Failures, 283
HEINZ LOHRER, WILFRIED W. ALT, AND
ALBERT GOLLHOFFER</p> <p>Index, 299</p> |
|--|---|

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Foreword

All physical activity of the human organism is the result of the interaction between the body's nervous system and the system of skeletal muscles that link and move the skeletal framework. Performance of the complex movements involved in competitive sport depends upon the production of force and power in highly coordinated movements. This involves interaction among the brain, spinal cord, peripheral nerves, and over 300 skeletal muscles.

This volume of the *Encyclopaedia of Sports Medicine* has focused on the complex interactions that have been identified as "neuromuscular." Prof. Komi and the many contributors to this volume have carried out their research in both the laboratory setting and the sport venue. Following sections of background science and basic mechanics,

the chapters deal with fatigue, effects of training, disuse, and ageing. The knowledge and understandings gleaned from the scientific investigations of these interactions is of great importance to sports medicine physicians, sports scientists, and coaches who work with athletes of all ages and skill levels to improve conditioning, enhance performance, and prevent injuries.

It is a pleasure to welcome this volume to the IOC Medical Commission series, *Encyclopaedia of Sports Medicine*.



Dr Jacques Rogge
IOC President

Preface

It is always a challenge to produce a volume that includes a large amount of research material that was produced in one's own laboratory and/or in collaboration with others in the field. I have been in the fortunate position of developing a laboratory related to both biomechanics and exercise physiology called, since 1994, the Neuromuscular Research Center in the Department of Biology of Physical Activity at the University of Jyväskylä in Finland. This has been a 35-year journey with many exciting scientific attempts to try to better understand the secrets of the function of the neuromuscular system during exercise and sport. The travel has been characterized by not only many disappointments, but also many rewarding experiences. The frequent visits and research collaboration of numerous foreign colleagues and students have secured a continuous flow of publications and new approaches to explore this focus of our research. The contribution of our own Finnish students to the overall development of the research strategy has been fundamental, and I am proud that many of them have been able to work with me to produce several chapters for this particular volume.

Production of the "Neuromuscular Aspects of Sports Performance" as a special volume of the Encyclopedia of Sports Medicine is important for the following basic reason: In international perspective, the number of research laboratories in the area of neuromuscular function has been continuously increasing and the research problems have become more and more "natural." Consequently, advancement of the sophisticated instruments,

often developed and applied to isolated neuromuscular models, are today utilized for answering many research questions related to normal locomotion and exercise. An important example of this development is the so-called "Stretch-Shortening Cycle" (SSC) of muscle function, the mechanistic exploration of which started with isolated muscle preparations. Today, we are able to revisit these "isolated problems" by means of many methods and at many levels of exercise intensities. This includes also the examination of the interaction of supraspinal and spinal controls with muscle and tendon function during exercise.

The principal objective of the present publication is to present in the form of an Encyclopedia Volume the latest information on neuromuscular function in sport and exercise. Many chapters are written in a very comprehensive way, thus combining the basic mechanistic knowledge with true applications. The neuromuscular system is, therefore, often treated both as a control "organ" and as a system that can itself operate and function very efficiently in demanding sport activities. Starting with the introduction of basic neuromuscular function, as studied traditionally with isolated muscle functions, the chapters progress to explain the present state of the art in SSC and how it is applied in various sport activities. Many specific research tools, some of which require invasive approaches are explained in detail. These in-vivo techniques are needed to understand the true nature of tensile and ligament forces during intensive exercise, such as running, jumping, skiing, and cycling.

Special chapters are also devoted to the use of two more recently developed research methodologies during natural locomotion: high speed ultrasonography (US) and transmagnetic electrical stimulation (TMES). The US methodology has developed enormously over the last 20 years and can now be applied to study the function and interaction of fascicles and tendons even during outdoor activities such as cross-country skiing. TMES methodology, although still limited to recordings in the laboratory, has clarified what role the central nervous system can play in different exercise and fatigue situations.

A volume of this kind would not be comprehensive enough unless it contained specific chapters on neuromuscular fatigue, neuromuscular training, and musculoskeletal loading. The chapters of these particular topics are quite lengthy and thus very comprehensive and helpful in the understanding of the problems often faced in sports and, also, in activities of rehabilitation. This volume has in total 16 separate chapters, thus covering many aspects of "Neuromuscular Aspects of Sports Performance." The way the material has been presented varies slightly among chapters. In some cases, considerable depth and detail were necessary while, on the other hand, a few chapters have been written in a more readable and overview type format. Whatever the writing style, the material should be accessible to the readers who have a background in the biological aspects of sport science. As already indicated, the volume includes both basic knowledge and practical applications, the latter being, however, slightly more emphasized. The target audience consists, therefore, of those groups who have been involved in gathering this newest information: biomechanists, sports medicine specialists, sport scientists, and graduate

students in all these areas. It is also expected that advanced level coaches and sport physiotherapists will find this volume very useful. The volume was not, however, intended to make the sport activities as objects of descriptive analysis only. The purpose for the production of this collection of chapters was much more challenging: the understanding of both the "whys" and "hows" of neuromuscular function during sport activities.

As editor and contributing author of this present volume, I am extremely grateful to my colleagues who have contributed to the various chapters of the book. Special thanks go naturally to the IOC Medical Commission and its Coordinator of Scientific Publications, Professor Howard Knuttgen, whose assistance in the production of the volume has been instrumental. Through his vast experience, many of the problems related both to the volume content and organization were solved smoothly.

I am also very grateful to the Ministry of Education (in Finland), which gave me partial support for the production of the book. With regard to the possibility to concentrate on this particular project well, I am very grateful to the following two individuals: to my able successor as the Head of the Department of Biology of Physical Activity, Professor Keijo Häkkinen, and to the Director of the LIKES Research Institute of Sport and Health Sciences, Dr. Eino Havas, provided me with excellent working conditions as an emeritus professor. Finally, it was also rewarding to receive competent technical assistance from Ms. Martta Mäkilä, communication assistant at the LIKES—Foundation for Sport and Health Sciences.

Paavo V. Komi
Jyväskylä, Finland

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Chapter 1

From Isolated Actions to True Muscle Function

PAAVO V. KOMI

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Introduction

In order to function properly for force and power production in locomotion, the skeletal muscle possesses structural elements, which make it very responsive to commands from the nervous system, both centrally and peripherally. It is not intended to go into the details of the nervous control of the muscle contraction in this chapter. However, certain important aspects are needed to clarify the role the nervous system plays in muscle function and movement control. For more specifics, the reader is referred to general textbooks of neurophysiology and motor control. In the simplest form of force generation, the central nervous system (CNS) triggers the chain of activation along the direct pathway (pyramidal or corticospinal tract) as shown in Figure 1.1. The signal can also come from the different brainstem motor areas along the indirect pathways labeled as extrapyramidal tract. The activation signal from the motor areas passes then through several stages before reaching the muscle fibers to activate events which include Ca^{2+} release from the sarcoplasmic reticulum (SR) and subsequent contraction (excitation–contraction coupling). In addition, and very importantly in motor control, the signal can be initiated in the various muscle, joint, skin receptors, etc. The nervous system operates as a controller being able to initiate action potentials, receive and integrate feedbacks from the

higher brain centers and from the spinal cord levels. Consequently, even when the activating signal travels along the simplest and most direct pathway (corticospinal tract, CST) it is under the influence

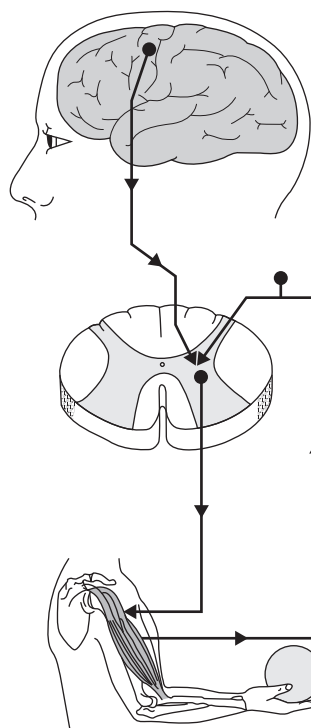


Figure 1.1 Control of muscle by the nervous system. Voluntary strength performance is determined not only by the quantity and quality of the involved muscle mass, the “engine”, but also by the ability of the nervous system, the engine controller, to effectively activate the muscles. (According to Sale 1992, with permission.)

of inputs from different sources and levels of the nervous system. All this information, regardless of the source, is received and integrated by the alpha motor neuron, which is called the “final common pathway of the motor system.” The type of muscle contraction is then determined by the frequency of action potentials coming from the alpha motor neuron. The alpha motor neuron can itself have different properties, as will be discussed shortly in connection with “fast” and “slow” motor units. Skeletal muscle contains all of the elements needed for force and movement production, but without the nervous control, the muscle is not capable of any force production above the passive tension. The passive tension (or resting tension) maybe due to the muscle’s structural elements, which offer resistance to stretch. Nonetheless, the muscle can be activated by impulses coming along the final common pathway, the alpha motor neuron. Upon activation, it then has a special ability to generate force, resulting in either shortening (concentric action) or resisting external loads (lengthening contraction or eccentric action). A complex integrative process involving the three components, the nervous system, skeletal muscle, and the external load, determines the final direction of movement as well as its velocity (or rate) and magnitude. It is the purpose of this chapter to characterize those factors that are important in understanding the basic interaction between the elements mentioned earlier. Greater emphasis will, however, be given to the important concepts of muscle mechanics as well as to the interaction between the contractile structures and tensile elements in the process of force production under varying movement conditions.

The Motor Unit and its Functional Significance

It is usually believed that human skeletal muscle fibers are innervated by only one motor neuron branch, but this branch maybe one from 10 to 1000 similar branches all having the same axon. Therefore, one axon innervates a number of muscle fibers and this functional unit is called a “motor unit.” Consequently, a motor unit is defined as a combination of an alpha motor neuron and all the

muscle fibers innervated by that neuron. Motor unit size (muscle fibers per alpha motor neuron) varies within a muscle, and the number of motor units varies between muscles. As illustrated in Figure 1.2, the motor units have different structural and functional characteristics, which result in their differences with regard to rate of force development (RFD), peak force production, and maintenance of force level without loss of tension (fatigue). The fast, fatigable (FF) type unit develops tension quickly, but is also very easily fatigued. At the opposite end, the S-unit has a slow rate of force production but can produce the same tension (force) repeatedly for longer periods of time without signs of fatigue. It is therefore also called a “fatigue-resistant motor unit.” In addition to those events described in Figure 1.2, there are also other functional differences between motor unit types. One particular feature that illustrates such differences is the response of the motor units to tetanic stimulation. The FF type unit requires a high stimulation frequency to reach a state of tetanus. In contrast, the slower unit requires a much lower fusion frequency. When subjected to repetitive tetanic stimulation, the resulting difference in mechanical response between the two extreme types of motor units is remarkable. It is most probably the size of the alpha motor neuron that determines the specific type of the motor unit. Motor neurons supplying the faster motor units have larger cell bodies and axons. They also innervate larger number of individual muscle fibers. One important feature must be emphasized here: the type of alpha motor neuron determines the histochemical profile and biochemical performance of the individual muscle fibers in a motor unit. Consequently, all the fibers in the same unit have similar chemical profile. It is well known in the literature that muscles differ in their fiber composition (and thus in their motor unit profiles), and that there can be great variation among athletes with regard to the fiber structure in a specific muscle. For example, in the vastus lateralis (VL) muscle, sprinters may have a motor unit make up that causes most of the fibers in that muscle to be of a fast type, and thus capable of producing force at a high rate, but with low fatigue resistance. Endurance runners, on the other hand,

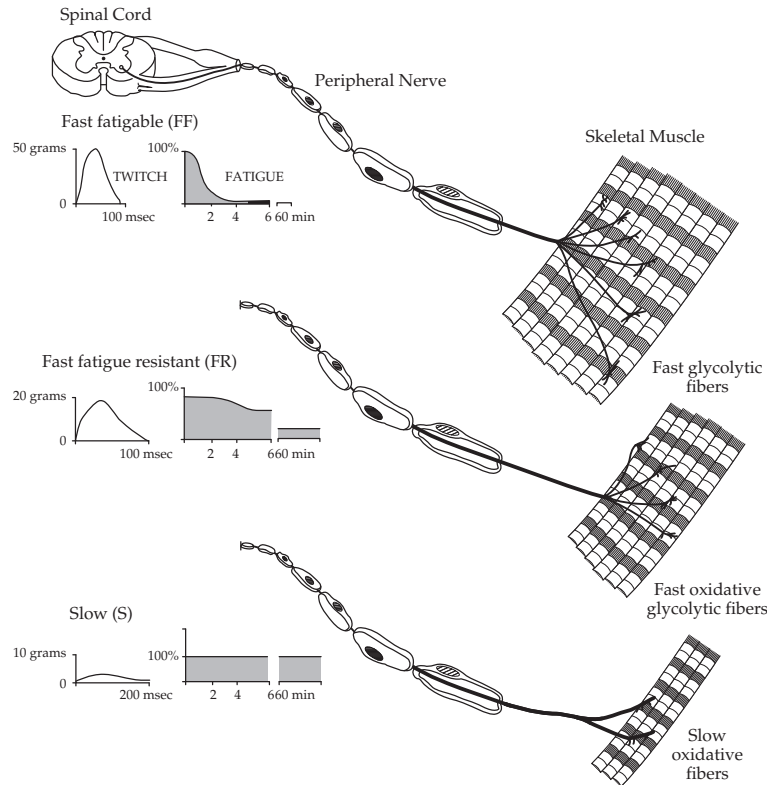


Figure 1.2 Schematic representation of the anatomic, physiologic, and histochemical properties of the three motor unit types. FF units (top) have large axons that innervate many large muscle fibers. The units generate large tensions but fatigue rapidly (tension record insets). FR units (middle) have moderately sized axons that innervate many muscle fibers. The units generate moderate tensions and do not fatigue a great deal. S units (bottom) are composed of small axons that innervate a few small fibers. The units generate low forces but maintain force for a long time. Reproduced with permission from Lieber R. L. (2003) *Skeletal Muscle Structure, Function, & Plasticity*, © Lippincott Williams and Wilkins (<http://lww.com>)

have primarily slow type fibers in the same muscle for the purpose of high resistance to fatigue, but at the same time the rate of force production is lower than in their sprinter counterparts. It has been reported from studies with monozygotic twins that genetic factors strongly influence the variation observed among individuals in muscle fiber composition (Komi et al., 1977) of a specific muscle. Differences in muscle fiber composition observed among athletes have thus raised the question as to whether the fiber composition of an individual athlete is an acquired phenomenon or is due to a genetically determined code.

As discussed in more detail in Chapter 13, the order of the motor unit recruitment follows the

so-called “size principle” (Henneman, 1957). The influence of size on recruitment order is attributable to its effect on input resistance. The small motor neurons have a high input resistance, and they are the first to be recruited in response to an increase in depolarizing synaptic currents. As a consequence, smaller motor units are activated before larger units. Due to the relation between the size of the motor neuron and the properties of the muscle fibers it innervates, this recruitment sequence results in slow-contracting and fatigue-resistant motor units being recruited before fast-contracting and fatigable motor units. Although there is some variability in the recruitment order of motor units with similar thresholds, the recruitment order of motor

units is essentially the same for isometric and dynamic contractions, including shortening and lengthening contractions and during rapid (ballistic) isometric and shortening contractions.

Muscle–Tendon Mechanics

Skeletal muscle has different properties, which influence its functional characteristics. Based on the earlier discussion about the different types of motor units (e.g., fast versus slow), the property of the alpha motor neurons should then be mostly responsible for the final product of the function: mechanical performance of the muscle. The human skeletal muscle is not, however, only the muscle; it contains an important element of the tendon, which in its turn has special mechanical characteristics. Consequently, the performance of skeletal muscle is under the influence of the innervation, the histochemical make ups of the contractile elements and the tendon, which connects the muscle to the insertion sites on the bones. In human (or animal at large) body, these elements and profiles must function together to produce a well-controlled movement. In addition, a muscle in the body acts often together with its synergist and antagonist muscle. The final mechanical output is therefore a complex phenomenon, and it is almost an impossible task to predict the true movement from measurements performed with individual components (activation profile, contractile part, and tendon) only in an isolated form. However, the contribution of the scientists, who have done pioneering work with isolated preparations, must not be forgotten. This work, which started already in the early 1900s, has been fundamental to understanding muscle mechanics in vivo locomotion. Several steps needed to be taken and the following paragraphs make attempts to present this basic information.

Twitch Characteristics of Isolated Muscles

As already referred to, action potentials traveling along the final common pathway (alpha motor neuron) reach finally the t-tubule and release calcium from its stores in the SR. Even a single action potential results in calcium release with subsequent binding with the troponin C and generation of

force in the actomyosin cross-bridges. Almost at the same time the free calcium is taken back into SR. During this short-lived “active” period when calcium is attached to troponin C, the generated force can be recorded, and it is called “twitch.” The rate of rise of the twitch tension as well as its relaxation are under influence of several factors, including the availability of free calcium, the speed of the binding of the myosin heads, rate of the calcium uptake back to SR, and the rate of the cross-bridge dissociation. Both the rates of the force development and relaxation are under influence of elastic properties of the cross-bridge. Most importantly, however, it is the activation profile that determines the twitch properties of the motor unit or the entire muscle.

When the electrical stimulation is used for twitch experiments, its strength must be strong enough to depolarize the muscle fiber (or muscle) membrane. Single stimuli of identical strength should then produce exactly similar force records (twitches). This indeed happens, if the two stimuli are separated by suitable interval. However, when the second stimulus is given before the first twitch is over, the resulting second force peak is usually higher than the first one (see Figure 1.3). The increase of the compound force becomes more pronounced the closer the consecutive twitches are brought together. This is illustrated in Figure 1.3. If the time interval between twitches is reduced so that the train of shocks comes with the frequency of 30Hz, the force records of the consecutive twitches do not return to zero. If the stimulation frequency is further increased, e.g., to 50Hz, the compound record may still increase and the curve looks like an unfused tetanus. Further increase in stimulating frequency (until 100Hz, corresponding to a 10ms stimulus interval) will finally result in complete fusion of the twitch responses. Thus as the frequency is increased, the ripple over the force record is reduced. Relationship between degree of this oscillation and the mean (or sometimes peak) force is used to imply the speed of muscle. Fast twitch muscle usually has a higher tetanic frequency as compared to the slower muscle. In a particular muscle, however, the 50Hz stimulation is usually enough to reach almost the full maximum isometric force level, and the additional

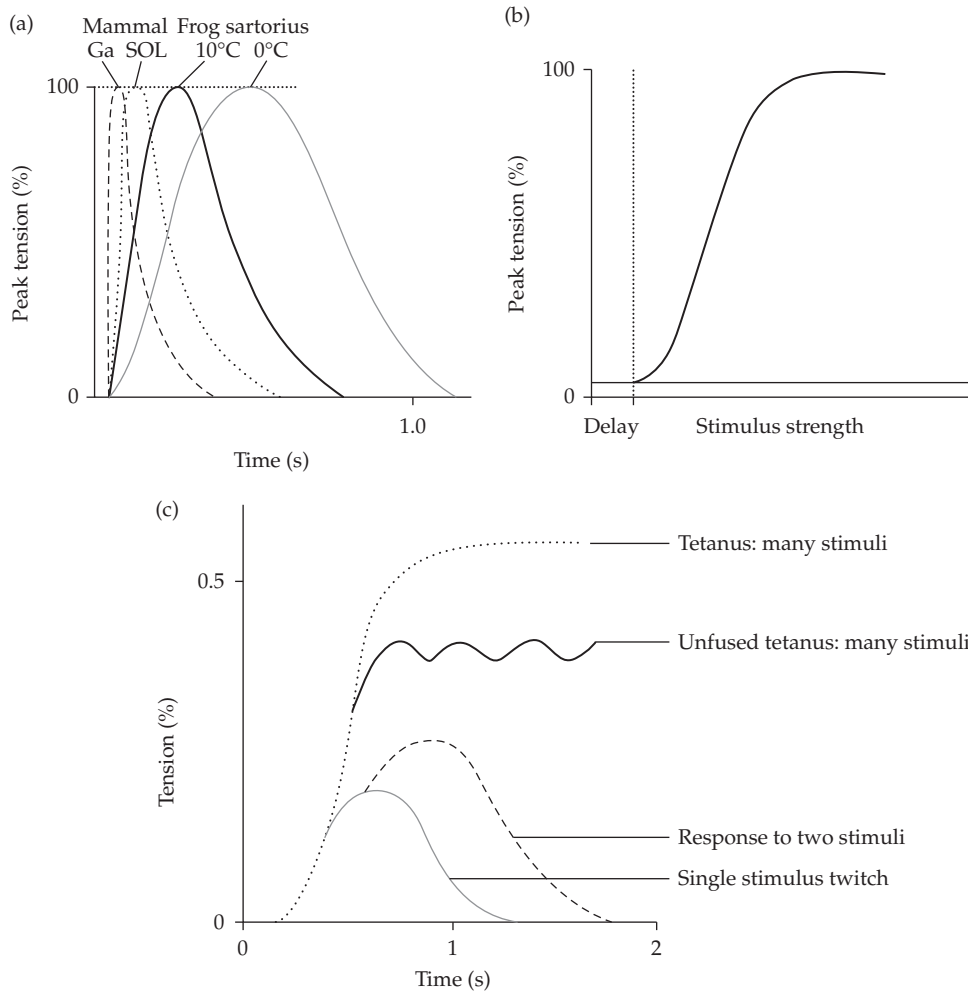


Figure 1.3 (a) The mechanical response (twitch) of mammalian muscle (GA, gastrocnemius; SOL, soleus) and frog (sartorius) muscle. In the frog, the twitch was measured at two incubation temperatures. Force is normalized to its peak (100%) for all conditions. (b) The relationship between stimulus strength and size of the twitch response. There is a delay before tension starts to rise. In normal locomotion, this delay is taken away by preprogrammed muscle action, such as occurs before contact with the ground during running. (c) When the stimulus is repeated, the force begins to summate, eventually reaching a state of tetanus. (Modified from Wilkie, 1968.)

increase of frequency up to 100Hz does not necessarily increase the peak force, but has considerable influence to increase the RFD (see Figure 1.4). This force–frequency relationship has given the basis for the force–time (F–T) relationships in the conditions of maximal voluntary activation. The fast type muscle can increase the force much faster than the slow type muscle, and this is primarily

due to the faster rate of activation. This has subsequently been applied to strength and power training, in which the increase in the RFD indicates improvement in the explosive force production. As explained in Chapter 13, specificity of training calls for modification of the F–T curve, the changes of which take place in the different parts of the curve depending on the training intensities and loads.

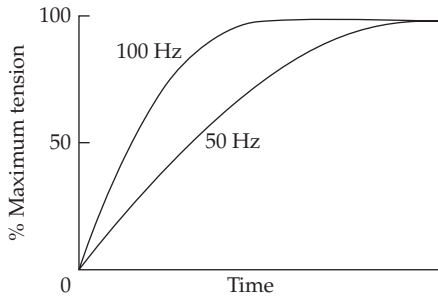


Figure 1.4 Effect of high-frequency stimulation on RFD. Stimulation at 100 Hz produces a greater RFD but no greater peak isometric force than at 50 Hz. Thus the very high motor unit firing rates observed at the onset of ballistic contractions (100–120 Hz) serve to increase the RFD of ballistic contractions. Such high rates would also increase the peak force of dynamic actions done at high velocity. (According to Sale, 1992 with permission.)

There are also indications that young girls have much slower rates of force development as compared to their male counterparts of the same age category (Komi et al., 1977). This was interpreted to indicate that females may have slower activation profile as the boys in the explosive F–T test, where the force production is started with zero activation. This difference may not be so apparent in more normal locomotion, such as stretch-shortening cycle (SSC) type muscle function, in which the preactivity plays an important role in efficient force and power production (see Chapter 2). Further discussion of the importance of the F–T curve and its practical relevance is given in the following paragraphs.

Basic Muscle Mechanics

Types of Muscle Action

In order to understand the way that skeletal muscle functions in normal locomotion, the relation between stimulus and response needs to be examined in more isolated forms of muscle actions: isometric, concentric, and eccentric. The term “contraction” maybe thought of as the state of muscle when it is activated via its alpha motor neurons, and generates tension across a number of actin and myosin filaments. Depending on the external load,

Table 1.1 Classification of muscle action of exercise types

Type of action	Function	External mechanical work*
Concentric	Acceleration	Positive ($W = F(+D)$)
Isometric	Fixation	Zero (no change in length)
Eccentric	Deceleration	Negative ($W = F(-D)$)

*W, work; F, force; D, distance.

the direction and magnitude of action is different as shown in Table 1.1. In a concentric action, the muscle shortens (i.e., the net muscle moment is in the same direction as the change in joint angle and mechanical work is positive). In an eccentric action, the muscle actively resists while it is being lengthened by some external force, such as gravity. In this case, the resulting muscle moment is in the opposite direction to the change in joint angle, and the mechanical work is negative. The use of the term “muscle contraction” is therefore sometimes confusing, and we would prefer to follow a suggestion made by Cavanagh (1988) that the term “contraction” should be replaced by “action.”

The muscle action most frequently used to characterize the performance of human skeletal muscle is the isometric action, which by definition refers to the activation of muscle (force production) while the length of the entire muscle–tendon unit (MTU) remains the same, and the mechanical work is zero. The use of isometric action in locomotion is not, however, meaningless; it plays a very important role in the process of preactivation of the muscle before the other actions take place.

Force production in all types of muscle actions can be seen in the internal rearrangements in length between the contractile and elastic elements. Figure 1.5 depicts these events for the isometric and concentric actions. For the isometric action, in this simplest model of the muscle, force is generated through the action of the contractile component (CC) on the series elastic component (SEC) which is stretched. The resulting S-shaped F–T curve is shown on the right side of Figure 1.5. Concentric action, where the load is attached to the end of the muscle, is always preceded by the isometric phase

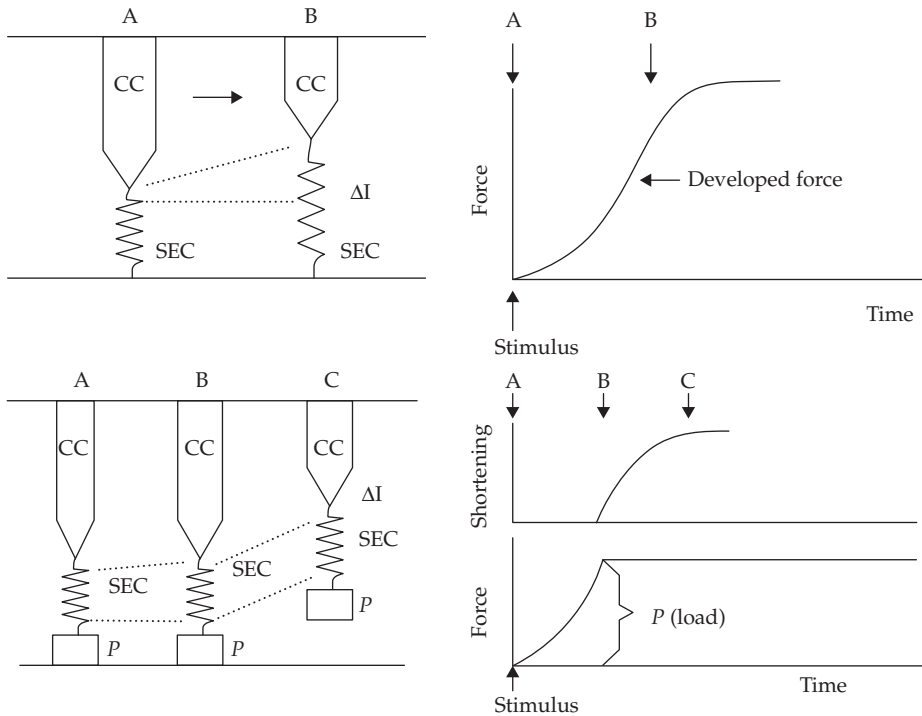


Figure 1.5 Models of isometric (A) and concentric (B) muscle action. In isometric contraction, the total length of the muscle does not change, but activation (A–B) causes the contractile component (CC) to shorten and stretch the series elastic component (SEC). Concentric action begins with an isometric phase where CC first shortens and stretches SEC (A–B). Actual movement occurs when the pulling force of CC on SEC equals or slightly exceeds that of the load P (B–C). (From Braunwald et al., 1967, with permission.)

with the rearrangement of the lengths of CC and SEC. The final movement begins when the pulling force of CC on the SEC equals, or slightly exceeds, that of the load. In eccentric action (not shown in Figure 1.5), some external force, e.g., gravity and antagonist muscles, forces the activated muscle to lengthen.

Of the two “dynamic” forms, eccentric action plays perhaps a more important role in locomotion. When the active MTU is lengthening—after the preactivation (isometric) phase—it forms the basis of an SSC, the natural form of muscle function in sports and normal daily life involving movement of the joints or the whole body. Before going into detail about SSC, the main mechanical attributes of muscle function need some consideration. This will help the reader to understand why SSC has such an important role in force and power production.

Force–Time Characteristics

As is evident from Figure 1.5, to perform movement at a joint requires time, as calculated from the first intentional “command” either from the CNS or via reflexes from, for example, proprioceptive feedback. This time delay has several components, including both neuronal conduction delays such as synaptic transmission, events for excitation–contraction coupling, as well as mechanical characteristics of the muscle fibers that receive the command signal. In this regard, isometric action is very convenient to describe the stimulus–response characteristics of human skeletal muscle. The first principle of muscle mechanics, the “F–T” relation, varies as a function of stimulus strength as well as between muscles and different species. As already discussed, the size of a single twitch response depends on the stimulus strength: a single shock, if strong enough, produces

only a small twitch; a second repetitive shock adds to the force of the first stimulus, when it is given before complete recovery of the first response. If one imagines a real movement situation, in which the load is fixed to the end of the muscle, that load does not begin to move before the stimulus strength to the CC to pull the elastic component of the muscle equals or exceeds the total load. When stimulus frequency is increased, the force gradually reaches a tetanic state that ultimately describes the maximum F-T characteristics of a muscle in isometric actions. As already referred to, the isometric F-T relationships are different between muscles and species. The most fundamental feature for human locomotion is the difference between the fast type and endurance type muscles: muscles consisting of a majority of fast twitch fibers (and consequently innervated more heavily by fast conducting alpha motor neurons) have a faster RFD compared with muscles possessing a majority of slow (endurance) type fibers (Komi, 1984).

In spite of this rather clear difference that has been observed in isolated muscle preparations, the existing experimental evidence in humans does not always support the interrelationships (structure versus function) found in isolated muscles. For example, some studies (Viitasalo & Komi, 1978) have demonstrated a significant relationship between structure and function in the case of isometric force production, while the same authors (Viitasalo & Komi, 1981) have failed to do so in another study. Similar contradictions have been observed for the vertical jump test. Consequently, F-T characteristics of either isometric or dynamic origin seem to be under strong environmental influence. Effects of training, for example, on the F-T curve are probably of greater importance than the muscle structure itself. Voluntary explosive force production requires a well-controlled, synchronized activation process. Thus the experimental situation is very different from that of isolated preparations, which utilize constant electrical stimulation either on the muscle or its nerve. In normal human locomotion, the movement is seldom, if at all, initiated from zero activation. Preactivation is a natural way to prepare the muscle for fast force (and movement) production, and to set a zero electromyographic (EMG)

activity as a required condition, may not be so successful in all individuals. The important role of preactivation will be discussed later.

Force-time curve has considerable practical interest. In sporting activities, the time to develop force is crucial, because the total action times for a specific muscle may vary between less than 100 ms to a few hundred ms. Thus, if the F-T curve is measured, for example, for the leg extensor muscles, the peak force is sometimes reached after 1000 ms, implying that a specific movement in a real life situation would already be over before these force values were reached. Consequently, training studies have recently concentrated on examining the F-T curve in its early rising phase (see Chapter 13). Several methods have thus been used in the literature to assess the RFD. As recently examined by Mirkov et al. (2004), most of these methods may be considered as fairly reliable, but their "external validity" to evaluate the ability to perform rapid movements remain questionable. The F-T curve also reveals that if the movement begins at the point of zero EMG activity (the force is also zero), then the practical consequences would be catastrophic. This is naturally corrected by preactivating the muscles appropriately before the intended movement begins. Preactivation is preprogrammed (Melvill-Jones & Watt, 1977) and is introduced to take up all the slack within the muscle before the initiation of fast movements. The preactivation refers usually, but not always, to the isometric phase before the other forms of action take place. Its EMG magnitude is a function of an expected load to move or an impact load to receive, such as in running (Komi et al., 1987). This preactivity corresponds to the initial stimulation that is a necessary component in the measurement of concentric and isometric actions. This requirement is in agreement with the measurement techniques applied in isolated preparations (Hill, 1938; Edman, 1978).

Force-Length Relationship

The skeletal muscle is not only "muscle;" it contains several connective tissue structures, which are not under efferent nervous control. Contribution of these passive structures can be measured by

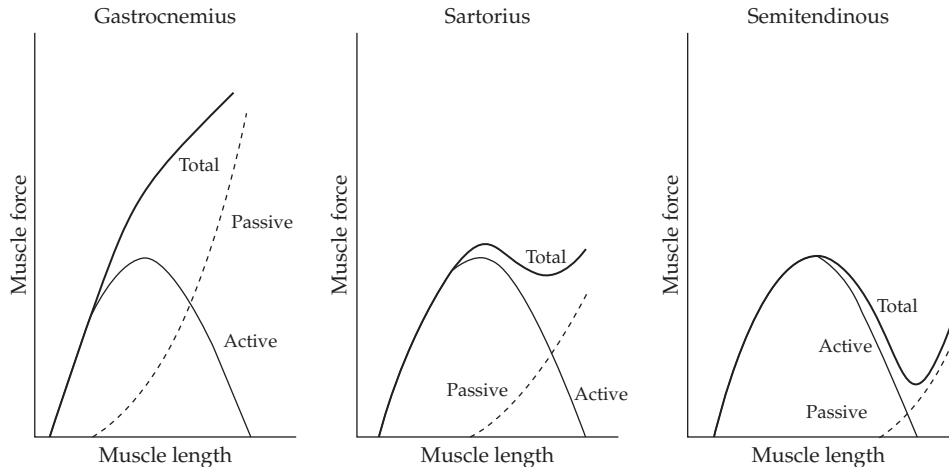


Figure 1.6 Active and passive F–L relationships of three frog muscles. The total muscle force curve is the sum of these curves. Note that the passive curve adds to the total force at different regions of the active curves but always at some length greater than maximal force development. (Modified from Wilkie, 1968.)

simply stretching the muscle without stimulation to a number of constant lengths. This procedure demonstrates that resting muscle is elastic and able to resist the force that stretches it. During this stretching, the muscle becomes more and more inextensible, i.e., the force curve becomes steeper with larger stretches. The resulting curve represents a passive force–length (F–L) relationship that is determined largely by the connective tissue structures such as endomysium, perimysium, epimysium, and tendon. Muscles differ especially in the form (and position) of the passive force curve. Figure 1.6 gives examples of three muscles, gastrocnemius (GAST), sartorius, and semitendinosus. From these the GAST muscle contains greater proportion of connective tissue, and consequently the passive force curve becomes steeper earlier than in the sartorius muscle. The active curve in Figure 1.6 constitutes the CC, whose form represents the contribution of the contractile material (fascicle or muscle fibers) to the total force curve, which is the sum of the active and passive forces at given muscle lengths. It must be emphasized that the active curve is not a continuous curve. It represents discrete data points observed when the muscle is held at different lengths and then stimulated maximally (or supramaximally) in each length position. The

total F–L relationship differs between the muscles, and for this reason no definite F–L relationship can be described that would be applicable to all skeletal muscles. From these curves, as shown in Figure 1.6, the active component has received much more attention as it resembles the F–L curve of individual sarcomeres. As will be discussed later, the working range of the sarcomere F–L curve seems to be different depending on the activity. The form of the active F–L curve depends upon the number of cross-bridges that are formed at different sarcomere lengths. The sarcomere number is not fixed, even in adult muscles, being capable of either increase or decrease (for details see Goldspink & Harridge, 2003). For the entire MTU, however, exhaustive fatigue has been shown to shift the total F–L and torque–angle curve to the right (Komi & Rusko, 1974; Whitehead et al., 2001), and in severe eccentric exercise this shift has been considered to reliably indicate the degree of muscle damage (Jones et al., 1997). In addition to differences between muscles, the type of muscular exercise seems to determine the portion of the F–L curve (descending limb, plateau phase, or ascending limb) in which a particular muscle operates during locomotion.

It should be mentioned that until recently, it was very difficult to obtain anything other than

a measure of the torque–angle relationship in humans, leading to an estimate of the F–L changes. At present, accurate tensile force calculations can be performed *in vivo* by applying devices such as buckle transducers (Komi, 1990) or the optic fiber method (Komi et al., 1996) directly to human tendons. With the development of real-time ultrasonography (US), it is now possible to examine noninvasively and *in vivo*, the respective length changes of the fascicles and tendinous tissues (TT: aponeuroses and the free length of the in-series tendon) during exercise. In general, the obtained results highlight the complexity of interaction between fascicle and TT components (see Chapter 10).

Force–Velocity Relationship

It is a common experience that a muscle can shorten faster against a lighter load than it does against a heavier one. In sport an example can be given from shot put, where the lighter shot can be put much further than the heavier shot. The inertia of the weight (shot) is partly responsible for this difference, but the main cause can be found

on the muscle level. The muscles can produce much less force when actively shortening against a lighter load. When the load (shot, in our example) is extremely heavy, it cannot be put or lifted. In fact the load that cannot be moved at all, despite the maximum voluntary effort, corresponds to the maximum isometric force of the muscle involved. Work performed with the isolated muscle preparations has explained this phenomenon. Hill's classical paper (1938) describes this force–velocity (F–V) relationship of an isolated muscle preparation (Figure 1.7a).

This curve can be obtained with constant electrical stimulation against different mechanical loads. The muscle is maximally (or supramaximally) stimulated and when the isometric force reaches its maximum, the muscle is suddenly released, and depending on the magnitude of the extra load the resulting shortening speed can be determined. In this relationship, the maximum force decreases in the concentric mode in a curvilinear fashion, and as a function of the shortening speed. It must be emphasized that the obtained curve is not a continuous one, but a discrete relationship of distinct data points. This classical curve demonstrates the

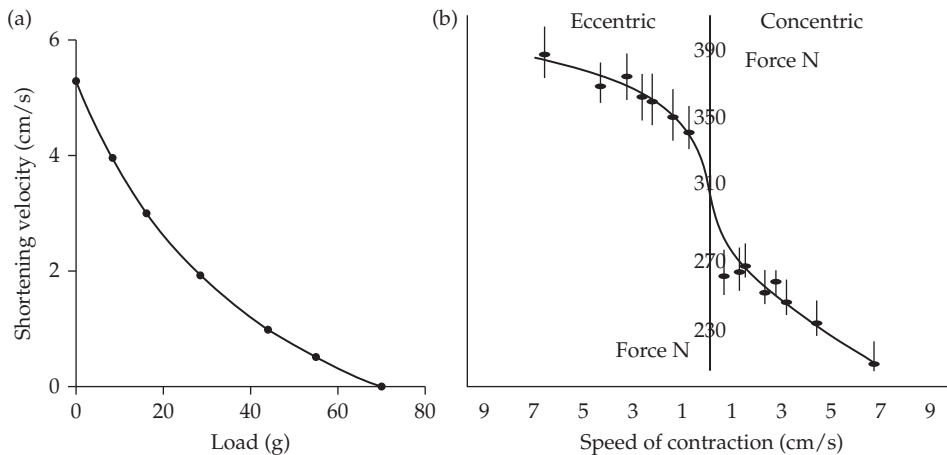


Figure 1.7 (a) The classic Hill F–V curve for the frog sartorius muscle. The line is not a continuous one but represents discrete relationship of distinct data points. (b) F–V relationship in eccentric and concentric muscle actions for elbow flexor muscle in humans. The measurements were performed with an electromechanical dynamometer, which was designed to arrange a constant velocity of shortening or lengthening for the biceps brachii muscle. Note that both in (a) and (b) the muscle was activated maximally, either in electrical stimulation (a) or voluntarily (b). In case of (b), there was no difference in maximal EMG activity between the two action types. (From Komi, 1973 with permission.)

fundamental properties of the skeletal muscle, and its form has also been confirmed in human experiments with maximal efforts against different loads (Wilkie, 1949) or with maximal efforts at different constant angular velocities (Komi, 1973). When the F-V measurements are extended to the eccentric side by allowing the muscle to actively resist the imposed stretch that begins after the maximum (isometric) force level has been reached, maximum force increases as a function of stretching velocity, as shown in Figure 1.7. An important prerequisite in the measurements of F-V curves is the strict control of the maximum preactivation before the movement begins. Although this principle is equally relevant to the measurements of both concentric and eccentric sides, the eccentric force measurements have not followed these principles carefully enough. Figure 1.8 gives this requirement of full preactivation in the eccentric force measurement performed with human knee extensors. The presentation is exactly similar to that used for isolated muscle fiber/sarcomere preparations (Edman et al., 1978).

In both cases, the muscle tissue is fully activated (electrically in the case of isolated muscle fiber and voluntarily in human knee extensors). The stretching of the fiber or muscle must not begin before the preactivation brings the force to full isometric maximum of the particular length of the fiber or the

muscle. One can imagine that if the stretch phase begins when the muscle has zero activation (no preparatory activity), the performance of the muscle will be reduced and especially in fast stretch situations the peak force may not be reached at all during the entire stretching phase. As the full isometric activation in human skeletal muscle takes considerable time (sometimes up to 1 s), it is important to obtain the full preactivity in all velocity conditions of shortening (concentric) and lengthening (eccentric) actions. When human experiments have followed the methods of isolated models (Hill, 1938; Edman et al., 1978), the voluntary concentric and eccentric F-V relationships were rather similar to those of isolated preparations (Wilkie, 1949; Komi, 1973; Linnaam et al., 2006). This includes the finding of similar maximal EMG activities across all contraction modes (eccentric, isometric, and concentric) and velocities (Komi, 1973). The observation that voluntary eccentric force can sometimes be less than isometric force (Westing et al., 1991) may well be explained by the fundamental differences between experiments, especially when the preactivation was not maximal before recording the concentric and eccentric forces at different velocities of shortening and stretch, respectively. This possible reduction in eccentric force as compared to isometric force has also been suggested to be due to the inhibition of EMG activity. Again the differences

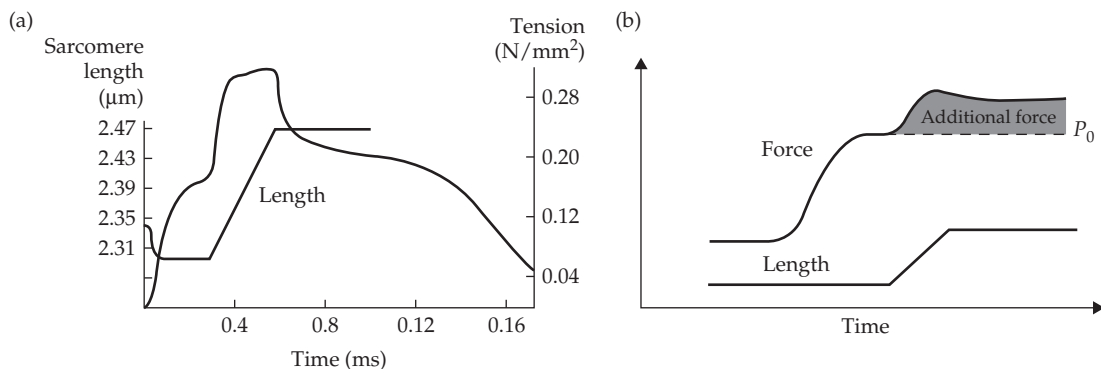


Figure 1.8 (a) Sarcomere F-T relationships when it was stimulated first maximally in isometric situation and subsequently stretched. (Based on Edman et al., 1978 with permission.) (b) The same relationship for human knee extensor muscles, which were activated maximally in isometric situation followed by stretching of the muscle. Note considerable force enhancement during the stretching phase. (Komi, 1973 unpublished observations).

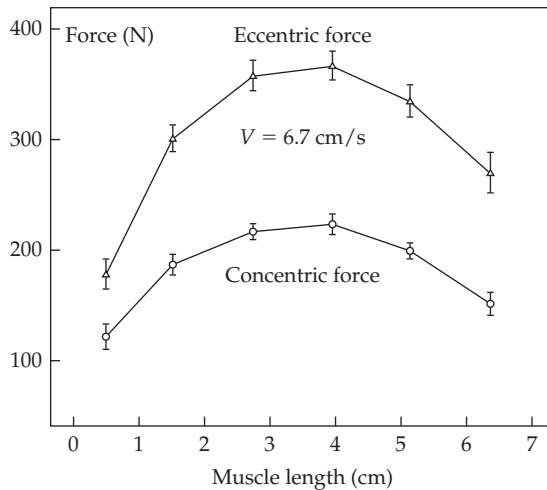


Figure 1.9 Comparison of the F-L curves between eccentric and concentric actions of the human forearm flexors measured at constant velocities of 6.7 cm/s. (From Komi, 1973 with permission.)

in protocol between these experiments and those from the classical model could be looked at as a possible source of reduced EMG and the respective force level in eccentric action. Consequently, it is quite clear that it is the eccentric mode in which the force and power characteristics of skeletal muscle are greatest. In normal nonfatigue situations, the difference between maximal eccentric and concentric forces can be seen in all muscle lengths (or elbow angles) (Figure 1.9).

Although the Hill curve was not introduced to describe the instantaneous F-V relationship as shown later in Chapter 2, it has been used successfully to follow specific training adaptations of human skeletal muscle. These adaptations deal with the concept of power training, especially for sporting activities requiring high levels of force and speed (see Chapter 13). From the Hill curve, it can be calculated that muscle mechanical power (the product of force and velocity) usually reaches its peak when the speed and forces that are involved represent about $\frac{1}{3}$ to $\frac{1}{2}$ of the discrete points in the F-V relationship. Figure 1.10 shows the mechanical power values for both concentric and eccentric sides. In the eccentric actions, the force increases

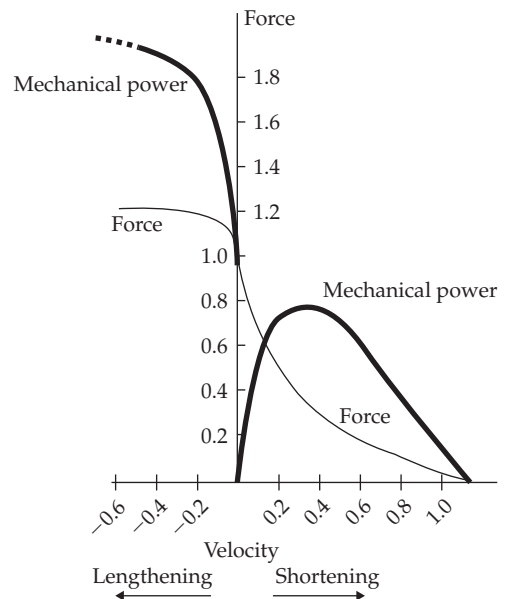


Figure 1.10 P-V curves in concentric (right) and eccentric (left) actions drawn to follow the F-V relationships. In concentric mode, the peak power is at velocity of $\frac{1}{3}$ to $\frac{1}{2}$ from maximum. The mechanical power is lowest at both ends of the velocity axis. In eccentric action, however, peak force increases initially above the isometric maximum, and to certain degree as a function of increase in stretching velocity. The resulting mechanical power shows dramatic increase in early part of the eccentric phase. The mechanical power was calculated from the product of tension (force) and shortening (concentric) or lengthening (eccentric) velocities.

(up to a certain point) as a function of increase in stretch velocity, and the resulting mechanical power values also increase in parallel, and reach values which are many times higher than in the concentric mode. The peak power in the concentric action is very sensitive to differences in muscle fiber composition. Faulkner (1986), among others, demonstrated in human skeletal muscle that the peak power output of fast twitch fibers was fourfold that of slow twitch fibers due to a greater velocity of shortening for a given afterload. In mixed muscle, the fast twitch fibers may contribute 2.5 times more than the slow twitch fibers to the total power production. In human experiments, it is difficult to utilize shortening (and also eccentric) velocities that

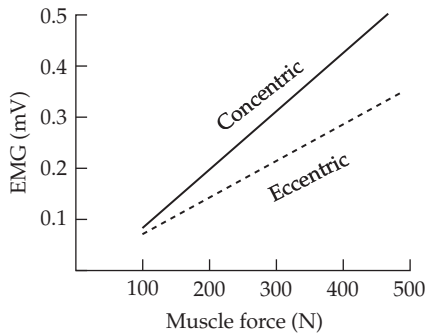


Figure 1.11 Electromyographic (EMG) force relationship in concentric and eccentric muscle action as measured with forearm flexors. (From Komi, 1973 with permission.)

can load the muscles with a suitable protocol (as described earlier) across the entire range of physiological speeds. The maximum speed of most of the commercially available instruments can cover only 20–30% of the different physiological maxima. As Goldspink (1978) has demonstrated, the peak efficiencies of isolated fast and slow twitch fibers occur at completely different contraction speeds. Therefore, it is possible that in measurements of the F–V curve in humans, when the maximum angular velocity reaches a value of 3–4 rad/s, only the efficient contraction speeds of slow twitch fibers will be reached. The peak power of fast twitch fibers may occur at angular velocities more than 3 times greater than our present measurement systems allow. Notwithstanding, Tihanyi et al. (1982) were able to show clear differences in F–V and power–velocity (P–V) curves in leg extension movement between subject groups who differed in the fiber composition of their VL muscle. These measurements have been restricted to the concentric part of the F–V curve only. In human muscle, similar efforts have not been made to explore the effect of muscle fiber composition on the eccentric F–V and P–V curves.

If the F–V (and P–V) curve demonstrates the primary differences between concentric and eccentric actions, there are some additional features that stress the importance of the performance potential between these isolated forms of exercise. As already mentioned, the maximum EMG activity

between concentric and eccentric actions should be approximately the same. However, it is well documented that the slopes representing EMG and force relationships are different in these two forms of exercise (Bigland & Lippold, 1954; Komi, 1973) (Figure 1.11). To attain a certain force level requires much less motor unit activation in eccentric than concentric action. Logically then, oxygen consumption is much lower during eccentric exercise than in comparable concentric exercise (Asmussen, 1953; Knuttgen, 1986). Furthermore, in relation to movement in general, these earlier findings, including the important reference to Margaria (1938), emphasize that mechanical efficiency (ME) can be very high during eccentric exercise as compared to concentric exercise. This information is then used to explain why the ME in normal locomotion can subsequently be high also in normal locomotion involving SSC exercise (for details see Chapter 7).

One additional and particularly relevant question is “what happens to the fascicle length (magnitude and change of length) during different muscle actions?” In our recent studies, we were able to demonstrate that during pure concentric actions the fascicles show normal shortening (Finni et al., 1998), the magnitude of which maybe intensity dependent (Reeves et al., 2003). In pure eccentric actions, fascicle lengthening (resistance to stretch while muscle fibers are active) should be expected and has indeed been well demonstrated by Finni et al. (2003) for the VL muscle. The fascicle lengths during eccentric action remained constant at all measured isokinetic speeds, but they were also shorter than those measured at higher concentric velocities. Although the latter finding does not directly imply the magnitude or even direction of shortening/lengthening they may stress an important point: the fascicle length change maybe dependent on the muscle and also on the specific movement. This notion becomes even more important, when the fascicle–tendon interaction is studied under conditions of different intensity SSC exercise. Both the chapter to follow (Chapter 2 on SSC) and Chapter 10 (on Ultrasound methodology) will discuss these issues in more detail and in situations of “true muscle function.”

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Chapter 2

Stretch-Shortening Cycle of Muscle Function

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Introduction

The discussion on muscle mechanics in Chapter 1 has given the basic information how the skeletal muscle functions in different isolated forms of contraction: eccentric, concentric, and/or isometric. It was then referred to the possibility that from these actions, the eccentric type plays a very important role in locomotion. This was concluded from the finding that the maximal eccentric force can be higher than the maximal concentric force, and even higher than the one of the isometric condition. This was shown in Figure 1.8 at both isolated muscle fiber level and schematically also at the whole muscle level.

The force and power potential of the skeletal muscle is indeed greatest under eccentric actions. The fundamental prerequisite to demonstrate this potential is that the stretch phase in the eccentric mode is performed immediately after the full tetanic force level (in isolated muscle) or full isometric force level (in voluntary conditions in humans) has been reached. Figure 2.1 clarifies this condition even further by emphasizing that “the force potentiation” during the stretch phase is dependent on two factors: (1) amplitude of the stretch and (2) velocity of imposed stretch. In the moderate stretch situation (Figure 2.1a), the force rises first quite rapidly but shows then a slower component of force increase when the stretch is continued. In another situation

(Figure 2.1b), the stretch speed is much higher and so is also the rate of force potentiation. However, the second phase is different from situation in Figure 2.1a: when the stretch continues with the same speed, the force declines rapidly already before the end of the stretch. Consequently, slowing of the force increase or rapid force reduction during the perturbation must refer to the capability of the sarcomere cross-bridges to resist the forceful

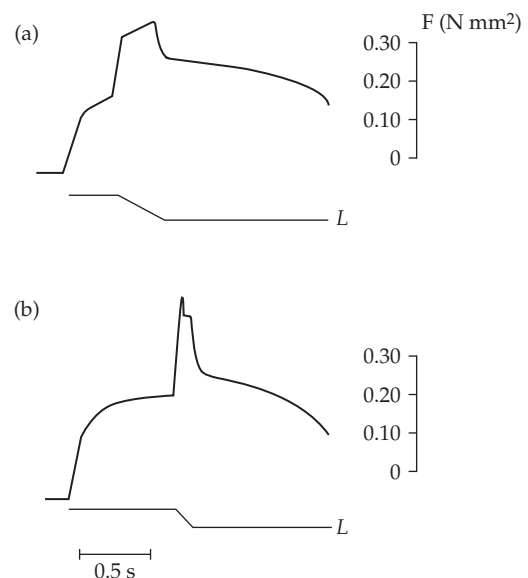


Figure 2.1 Recordings of the changes in tension during stretches of different amplitudes and velocities during the plateau phase on isolated frog semitendinosus muscle fiber. Note that the stretch velocity increases from (a) to (b). (Adapted from Edman et al., 1978, with permission.)

stretches. As introduced by Flitney and Hirst (1978), the phenomenon refers to the concept of “short-range stiffness,” the end of which is shown by the rightward reflection of the force curve. When the perturbation takes place very forcefully, the sarcomere cross-bridges slip and/or become detached momentarily. The detached cross-bridges make efforts to find the new binding site, but the time is too short for effective reattachment. As it is believed that the cross-bridges have also considerable elastic potential, the detached bridges will be lost from the source of the elastic storage.

Question must then be raised as to what this force potentiation phenomenon in eccentric action will have to do with actual locomotion. Although the detailed mechanisms are still under debate, the answer seems quite simple: the natural form of muscle function does not reflect any of the types of muscle actions in their isolated forms. Normal movements of the skeletal muscles are performed in combination of all forms of actions: isometric, eccentric, and concentric actions and indeed in this order. This sequence of muscle actions is called “SSC of muscle function.” By definition SSC means

that the preactivated muscle (isometric phase) is first stretched (eccentric phase) upon contact with the ground, in case of leg extensor muscles during running or hopping, for example. This eccentric phase (during the braking phase of the contact) is then followed without delay by the shortening (concentric) action during the final push-off phase. Schematically, this sequence of events in SSC can be drawn as shown in Figure 2.2a. Figure 2.2b demonstrates the distribution of EMG activities of different leg muscles in these phases. The major points to emphasize from this figure are the following: (1) the muscles are very active during the precontact and braking phases, and (2) less active in the push-off phase of the cycle. In some muscles, the EMG records are almost zero for the push-off phase. This suggests that this submaximal running can be performed with very little concentric (or positive) work effort. The muscles are naturally producing force during this phase, but it most probably results from the recoil of elastic energy, the details of which will be discussed later.

It must be noticed that the example in Figure 2.2b is from running at submaximal velocity. In

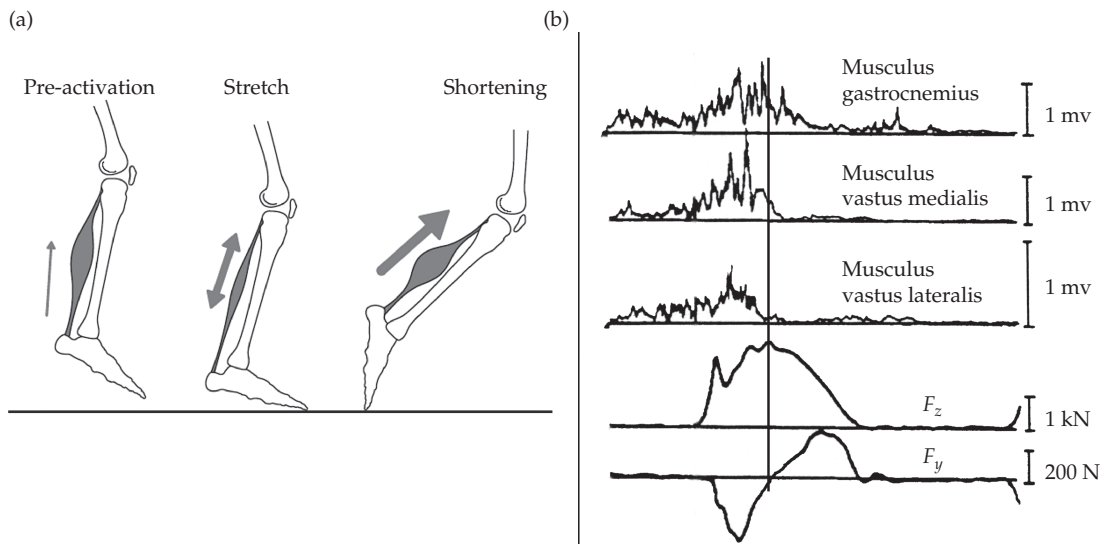


Figure 2.2 (a) The SSC of the triceps surae muscle during running. The cycle begins with the preprogrammed preactivity before toe contact with the ground. During contact, the activated muscle is first stretched (eccentric action), followed by shortening (concentric) muscle action. (b) EMG activities and GRFs during running at moderate speed. (Komi et al., 1986, with permission.)

maximal sprinting, however, one would expect to see maximal or close to maximal muscle activation patterns in both braking and push-off phases. The example of the submaximal running suggests that performance in the concentric phase is potentiated and/or made more economical by the behavior of the muscles during the preceding eccentric phase of the cycle. This indeed is what happens in SSC.

Stretch-shortening cycle has important functions in locomotion: (1) to minimize unnecessary delays in the F-T relationship by matching the preactivated force level to the required level to meet the expected eccentric loading, and (2) to make the final concentric action (e.g., push-off phase in running) either more powerful (in maximal effort) or generating force more economically (in submaximal conditions) as compared to the corresponding isolated concentric actions. In running, the amount of GAST preactivation is directly dependent on the expected impact load (Komi et al., 1987). The preactivated MTU is then lengthened during the ground impact. This active eccentric (braking) phase is followed without delay by the shortening (concentric) action. Depending upon the intensity of effort, this shortening or (push-off phase in running) can take place in many cases as a recoil phenomenon, with relatively low EMG activity.

It is often difficult to identify precisely the eccentric and concentric phases for the muscles. At single-joint levels, especially when EMG activities have not been measured, the reference cannot be made exactly for specific muscles, but in more general terms. In these cases, for example, the notion of eccentric and concentric phases are not correct, but should be replaced by "braking" and "push-off" phases, respectively. This approach has been used in running and cross-country skiing studies, in which cases the negative angular velocity implies the braking phase and the positive velocity the push-off phase in the particular joint movement (Ito et al., 1981; Komi & Norman, 1987). Consequently, other forms of identification have often been used, such as the lowest point of body center of mass (CoM) or transition of the horizontal ground reaction force (GRF) record from the negative (braking) to the acceleration (push-off) phase. Taking these limitations into consideration, the following few

paragraphs make efforts to describe both methodologically and mechanistically the various features of SSC. This approach aims to clarify to the reader also the complex nature of SSC, both in terms of definitions and specific features, when SSC is looked at the fascicle and tendon levels of the MTU.

Background of Performance Potentiation in SSC

Performance increase over the isometric force level can be demonstrated both with practical type natural movements or using isolated muscle preparations. To start from the latter examples, the pioneering work of Cavagna and his colleagues need to be acknowledged. They demonstrated in two studies (Cavagna et al., 1965, 1968) that when an isolated muscle is first stimulated isometrically and then stretched eccentrically while maintaining the same stimulation as in the isometric phase, the force then increases as expected. As shown in Figure 2.3, the muscle is then allowed to shorten after two different intervals: immediately after reaching the end of stretch (Figure 2.3b) or after a delay of 5s (Figure 2.3a). In both cases, the force curves come back to the original isometric condition. However, these two concentric phases are fundamentally different from each other. In Figure 2.3a, the delay of 5s causes the force to decline from 200 g (point *c*) to 100 g (point *d*). The hatched area drawn under the shortening phase shows then the work produced during the concentric phase. In Figure 2.3b, no delay was allowed between stretch and shortening and the work done in this case is much higher than in the situation, where the delay was zero. This delay is often called as coupling time between stretch and shortening. Cavagna et al. (1965) sought explanations for this phenomenon from the elastic properties of the muscle. The French researcher Goubel needs also recognition for his efforts to explain the mechanisms for elastic potentiation. His fundamental works are introduced in a textbook form (Goubel and Lensel-Corbeil, 2003).

Before their work, Hill (1950) had indicated that mechanical energy stored in the SEC can be used to produce a final velocity greater than that at which the CC itself can shorten. Cavagna's

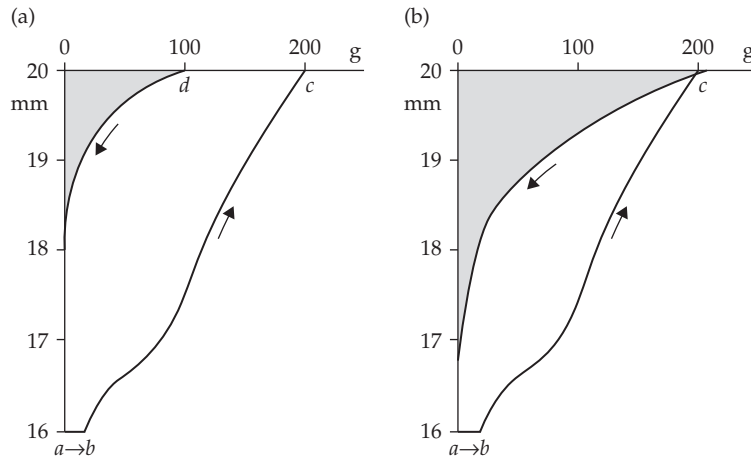


Figure 2.3 Utilization of elastic energy depends on the coupling time between the prestretch and subsequent shortening phases. The frog's sartorius muscle is first electrically stimulated ($a \rightarrow b$), thereafter it is actively stretched to the length of 20 mm. This resulted in the increase of tension ($b \rightarrow c$). (a) After this active prestretch, the constant length of 20 mm has been kept for 5 s and it resulted in decline of tension from c to d . At the point d , the muscle is allowed to shorten to its original length ($d \rightarrow b$). The shaded area expresses the work performed during the shortening phase. (b) Otherwise, the same as in (a), but there is no delay between stretch and shortening. The resulting work during shortening is much greater as compared to the situation in (a). (According to Cavagna et al., 1965, with permission.)

demonstration in Figure 2.3 simply means that stretching of an activated muscle leads to greater work and power output during the subsequent shortening phase of contraction, and that the amount of the concentric work is dependent on the coupling time. Consequently, elastic energy is stored during the negative (eccentric) phase and recovered in part during the following positive (concentric) work phase. However, this potential energy can be wasted as heat (Fenn & Marsh, 1935), if the concentric action is not followed immediately after the eccentric one. This emphasizes the transient nature of muscle elasticity changes, and makes a quick movement more profitable than the slow one (see also Cavagna & Citterio, 1974). Keeping the coupling time as short as possible has many practical advantages, among others improvement of economy (movement efficacy), which has been shown to be increased in SSC movements performed with a shorter coupling time between the braking and push-off phase in more natural type hopping movements (Aura & Komi, 1986).

Cavagna et al. (1968) made further demonstrations of performance potentiation, both in isolated

muscle preparations, as shown earlier, as well as with human forearm flexors. In isolated muscle, the stimulation was electrically induced and in human experiments voluntarily. The results were similar in both of these experimental models. Figure 2.4 gives an example of the results, which showed essentially the same phenomenon as in their earlier paper (Cavagna et al., 1965) that an active muscle is able to perform a greater amount of work when shortening immediately after being stretched.

Additional findings were of importance: the potentiation effect was the greater the faster the stretching speed and its amplitude were. The last two points have again considerable practical relevance. Although the curve in Figure 2.4 shows the work-velocity relationship, it can be used to describe the rightward shift in the actual F-V curve, the phenomenon that is so important in power training, for example (see Chapter 13). But even in these experimental situations without training, the effect of active prestretch on enhancement of the positive work can be of the order of 1.5–2.3 times the work performed when starting from maximal isometric situation. The results were explained as

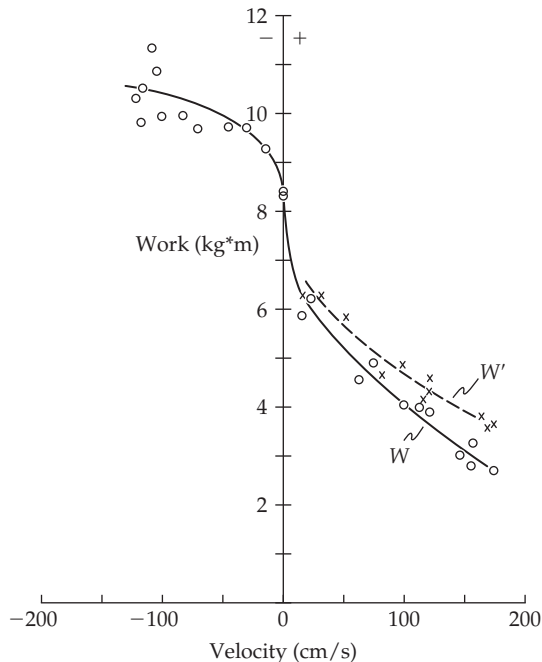


Figure 2.4 Ordinate: Negative and positive work performed by man's forearm flexors in maximal voluntary contraction during stretching and shortening, respectively. Abscissa: Speed of stretching and shortening. Note the shift in the work-velocity relationships when the elbow flexion (positive work) is performed following the prestretch. This is a representative record of one subject. (According Cavagna et al., 1968, with permission.)

follows: in these experimental situations of SSC: (1) "part of the work done on the muscle during eccentric phase is recovered as elastic energy which adds to the pull in the shortening phase of muscular contraction, and (2) the energy output by the CC after negative work is also increased" (Cavagna et al., 1968).

In the experiments described earlier, the coupling between stretch and shortening was of relatively short duration: 20ms for the isolated frog and toad muscles, and 26ms for the human forearm flexors. The importance of the coupling time in SSC actions cannot be neglected. Figure 2.5 presents this clearly: in this example, the subject performed maximal knee extensions using the principle of full activation during the entire SSC. The finding confirms the

observations discussed earlier that the time delay ("coupling time") between the stretch (eccentric) and shortening (concentric) phases plays an important role in SSC. As shown in this figure, if the shortening phase does not occur immediately, the maintained plateau phase is characterized by a systematic drop in force despite of maintained muscle activation. As has already been implied, part of the stored elastic energy can be potentially dissipated as heat and/or stocked in sarcomeric structures with damping properties (Cavagna et al., 1994). This can be avoided by keeping the coupling time short. This short coupling together with the velocity of stretch (Bosco et al., 1981, 1982; Komi, 1983; Aura & Komi, 1986) is most likely the two leading candidates to affect the performance potentiation in SSC. Note that the condition of zero coupling time (Figure 2.5b) shows also an important phenomenon: in SSC the concentric force after the prestretch can be increased considerably even above the preceding eccentric force. Practical examples are naturally many. Unfortunately, there are not many attempts to examine them with objective research methods.

While the coupling time was quite easy to control in animal models and also to some extent in human experiments in laboratory (Figure 2.5), the prestretch velocity can also be controlled to cover the range of different values. However, to explore this problem in more natural type movements, the stretch velocities must be adjusted in more global way. Drop jump (DJ) on the force plate is one of the means to regulate these conditions. The different dropping heights are used to indicate the stretch velocities during the braking phase of the contact. Consequently, the maximal rebound (vertical jump in centimeter) is measured as a function of the dropping height (cm) (for details see Komi & Bosco, 1978). In these measurements, all subjects demonstrate an initial increase in the height of rise of center of gravity (CoG). This increase in performance levels off and finally decreases after a certain drop height (breaking point). The breaking point in the jump height-drop height curve is very likely sensitive to gender, so that the males can tolerate and utilize higher stretch loads than their female counterparts. In adult women, this may occur at drop heights between 25 and 45cm and in adult

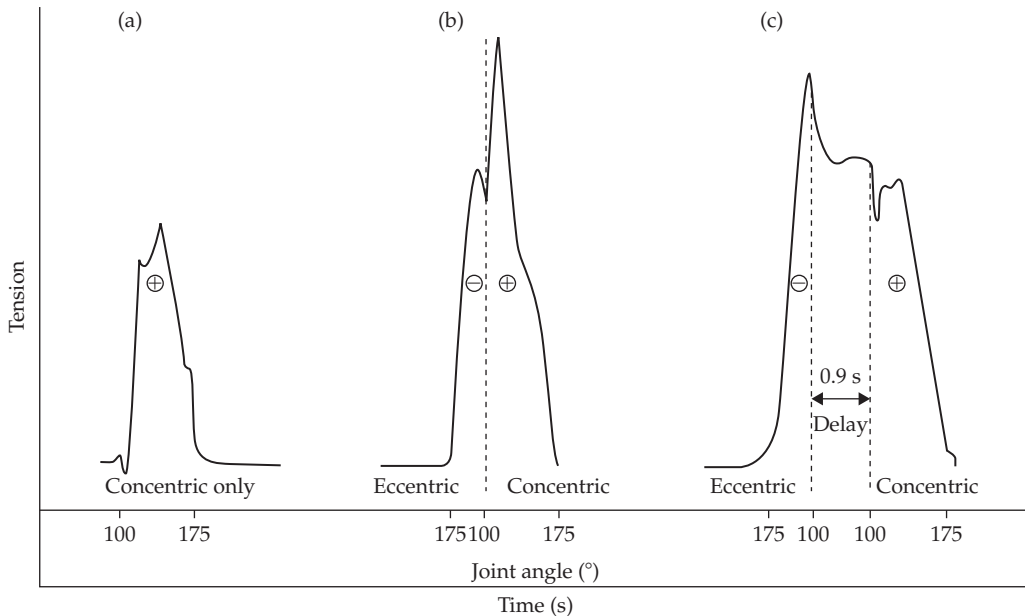


Figure 2.5 Three F–T traces for knee extension (100–175°), all performed with maximal voluntary activation. (a) A pure maximal concentric action. (b) Concentric muscle action is preceded by an eccentric (stretching) action, but no delay is allowed between these actions; note the force enhancement. (c) An identical action, but with a time delay between stretching and shortening; concentric force potentiation is reduced. (Adapted from Komi, 1983.)

men between 35 and 60 cm. There is great intersubject variability, and therefore the group averaged results do not always show the curvy relationships presented in Figure 2.6. Hopping training can, however, influence this relationship by shifting the curve to the right in both sexes (Komi & Bosco, 1978). This critical limit (breaking point) is also sensitive to growth and aging as shown by Bosco and Komi (1980), so that during growth and development (from 4 to 25 years of age) the best DJ performance is reached at increasing drop heights, but with further increase in age (25 and up) performance declines indicating reduction in tolerance to stretch loads (Figure 2.7). Recent experiment of Hoffrén et al. (2008) have shown that also in older individuals, the regular hopping training can increase the DJ performance and subsequently the tolerance to increasing stretch loads.

The jump performance at the breaking point stretch load represents the maximal jumping performance. The original notion of Asmussen and Bonde-Petersen (1974) was that in these DJs, the leg

extensor muscles are storing elastic energy during the braking phase, and part of this stored energy can be used to increase the performance, but only up to a certain limit. This upper limit of performance raises then questions of the possible mechanisms of performance reduction. There are certainly possibilities for the inhibitory and facilitatory stretch reflexes to be operative so that the facilitation increases initially with increasing stretch loads (drop heights). Inhibitory inputs (most probably of Ib type/Golgi tendon organs, GTOs) will then become more dominant when the breaking point load is approached in DJ. Possibilities for reflex intervention in SSC will be discussed in more detail later in this chapter. A third possibility that may appear independently or together with the reflex interventions could involve the slipping and/or detachment of sarcomere cross-bridges when the stretch load becomes extremely high.

This problem was approached by following the behavior of the fascicles of the GAST muscles in normal and extremely high DJ conditions. In the

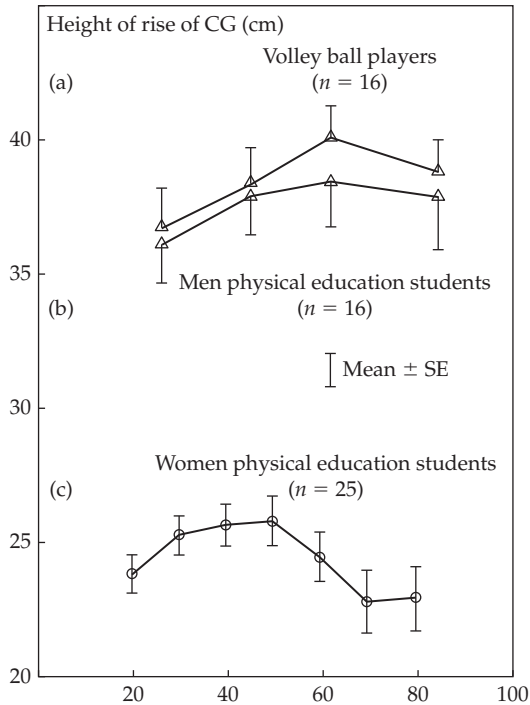


Figure 2.6 Group comparison of the height of rise of the total body center gravity in vertical jumps performed immediately after dropping on the platform from different heights. (a) Volleyball players, (b) men students, (c) women students. (From Komi & Bosco, 1978, with permission.)

high drop conditions, the fascicles showed sudden increase in length immediately after impact on the force plate (Ishikawa et al., 2005b). This did not occur in jumps with lower stretch loads, such as in medium or low DJ. This phenomenon is explained in more detail in Chapter 10, especially in Figure 10.8. At this point, it suffices to state that the detailed mechanisms and consequences of this fascicle length increase in high impact load conditions are yet to be explored. For instance, its timing maybe indicative of the critical stretch load, beyond which the medial gastrocnemius (MG) fascicles loose their capability to assist in effective utilization of tendon-muscular elasticity.

The observed phenomena reinforce the possibilities that can regulate the occurrence of the breaking point in the DJ performance: (1) the stretch load is mechanically so high and rapid that some of the actin-myosin cross-bridge interactions are simply detached or they slip apart, (2) inhibitory Golgi-tendon responses aimed at injury protection may become more dominant, (3) increased central input may cause presynaptic Ia afferent inhibition as a protective strategy to prevent tendomuscular injury due to the high stretch load. All of these mechanisms maybe operative either together or independently during extremely high impact SSC exercises.

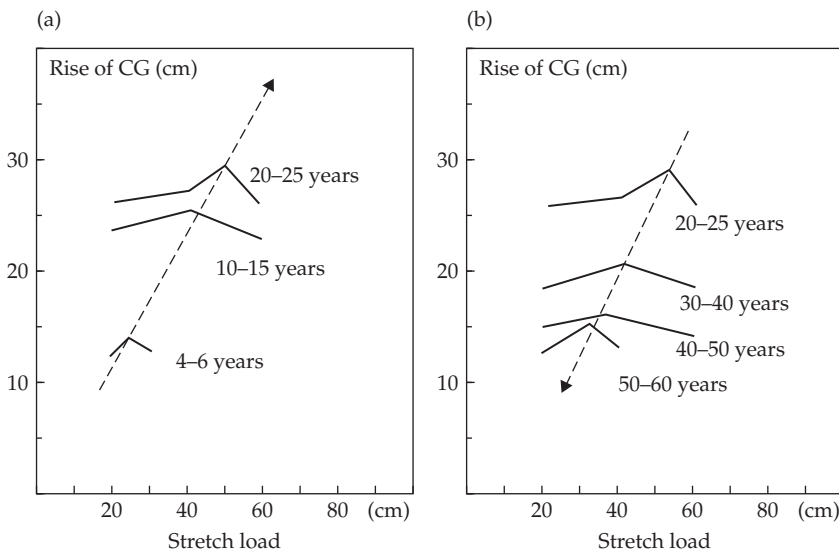


Figure 2.7 Influence of age on the rebound performance of the DJ. The jump performance increases at younger age (a) but decreases then after 20–25 years of age (b). The dashed line sketches the approximate breaking point of the jump height–drop height relationship. (From Bosco & Komi, 1980, with permission.)

One additional note is, however, necessary: in the later paragraphs, the sudden stretch of the MG fascicles will be discussed in relation to the possibilities of stretch reflex contribution during SSC. This is not to be confused with the fascicle stretches occurring in the extremely high impact load condition as demonstrated earlier. This present fascicle stretching deals with longer lasting phenomenon and “true” cross-bridge slipping. When we relate the sudden fascicle stretch to reflex action, the duration of the sudden stretch is very short-lived, and does not mean forceful breaking of cross-bridges.

What is the Best Movement to Demonstrate SSC?

Hopping, jumping, and running are considered as most typical forms of SSC. Walking as well as countermovement jumps (CMJs) are also SSC actions, because they include the sequence of stretch and shortening. The stretching (braking phase) is, however, very often so slow in these two activities that both slow walking and CMJ are difficult to use to explore the mechanisms of performance potentiation in SSC. The definition of SSC needs also to be looked at more critically. DJs cannot also be regarded as perfect demonstrations of performance potentiation of skeletal muscle during SSC. In isolated preparations and also in isolated joint motions (Cavagna et al., 1968), one knows exactly what muscles are involved

in the measurements, and that there are no other joints involved. DJ is a multijoint movement, and consequently the loading of the muscles should not be the same in all joints. Fukashiro and Komi (1987) made attempts to clarify how the hip, knee, and ankle joints are contributing to the joint moment–angular velocity relationships in maximal DJs. Largest peak values of the moment was clearly in the ankle joint (182Nm), the next one was the knee (163Nm), and the smallest was the hip joint (only 30Nm). Figure 2.8 demonstrates this together with the comparison of the joint moment–angular velocity curves measured under maximal efforts of the squatting jump (SJ) and the CMJ. This latter jump has often been considered as good demonstration of storage and utilization of elastic energy and consequently as a good movement to characterize SSC (Komi & Bosco, 1978). In CMJ, as shown in Figure 2.8, the mechanical work of the positive phase in the knee and ankle were very similar to the value of SJ. However, the work done by the hip (not shown in the figure) was much greater than that of SJ. Therefore, the difference in performance between SJ and CMJ, which was about 14cm in height of rise of center of gravity, may depend mainly on the difference of mechanical work of the hip joint. This suggestion is in contradiction to the earlier assumption that the performance difference between CMJ and DJ can be used to indicate the elastic characteristics of the leg extensor muscles (Bosco & Komi, 1982). Thus, it can be suggested that it is hopping that

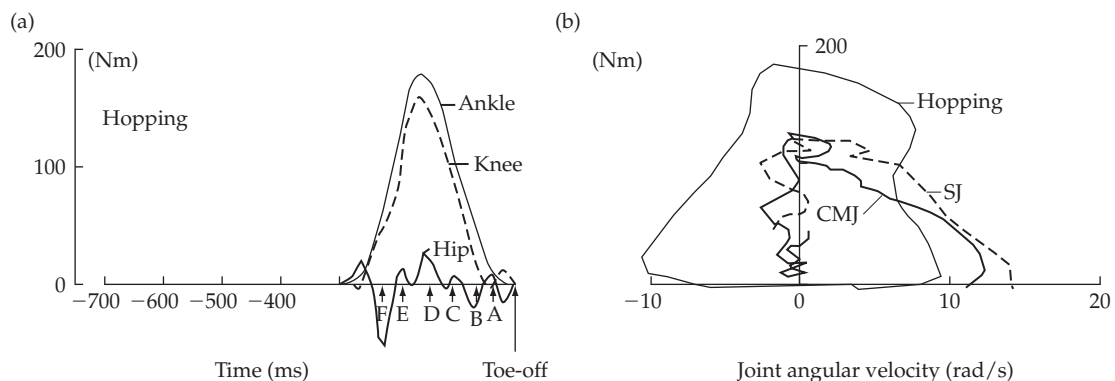


Figure 2.8 Moments of the joint forces (a) and the resulting moment–angular velocity relationship (b) in three different jumps: squatting jump (SJ), countermovement jump (CMJ) and hopping. For explanation, see the text. (According to Fukashiro & Komi, 1987.)

can be regarded as a ballistic type movement where the SSC behavior is very important. Storage and utilization of elastic energy may therefore be typical for this kind of jumping. The elastic nature of the muscle function, associated with the possible reflex-induced performance increase makes this jump more ideal to explore the secrets of SSC both in normal and fatigue situations.

The previous discussion suggested that the following three conditions are fundamental for effective SSC action: a well-timed preactivation of the muscle(s) before the eccentric phase, a short and fast eccentric phase, and immediate transition (short delay) between stretch (eccentric) and shortening (concentric phase). While CMJ can easily be demonstrated to produce higher jumping height as compared to the SJ (Asmussen & Bonde-Petersen, 1974; Cavagna et al., 1971; Komi & Bosco, 1978), it does not meet well all the criteria for an efficient SSC. For many neurophysiological and mechanical aspects, it is evident that CMJ is not the most suitable model to

elaborate on the specificity of SSCs. Instead, one has to look for more “normal” activities such as running and hopping, where the conditions of preactivation, faster stretch, and short transition time are well met. This may apply also to the possibilities of stretch reflex contribution of SSC performance. In addition, the forces measured in the muscle-tendon complex (MTC) during these activities can present a typical “bouncing ball” type form.

Instantaneous Force-Velocity Relationship during SSC

The classical force-velocity relationship, often referred to as Hill-curve, was introduced in Chapter 1. This curve represents conditions, in which the fully activated muscle is allowed to shorten against variable loads. Figures 1.7 and 1.10 can be referred to for further details. It must be emphasized that the Hill curve does not represent a continuous condition during movement, and it should not be confused with the instantaneous f-v relationship and the possible performance potentiation.

As discussed above, the true nature of force potentiation can be studied during SSC, and more specifically by measuring parameters that can be obtained with the in-vivo tendon force recordings. Figure 2.10 presents the results of such an analysis from fast running, and it covers the functional ground contact phase only. It is important to note from this figure that the force-length curve demonstrates a very sharp increase in force during the stretching phase, which is characterized by a very small change in muscle length. The right hand side of the figure shows the computed instantaneous force-velocity comparison suggesting high potentiation during the shortening phase (concentric action). The simultaneously recorded EMG activities (not shown in the figure) show that muscle activity levels are variable and primarily concentrated for the eccentric part of the SSC cycle. This is important to consider when comparing the naturally occurring SSC actions with those obtained with isolated muscle preparations and constant activation levels throughout the cycle. To make further comparison between the Hill curve and the instantaneous f-v curve, figure 9.13 in Chapter 9 can be consulted”.

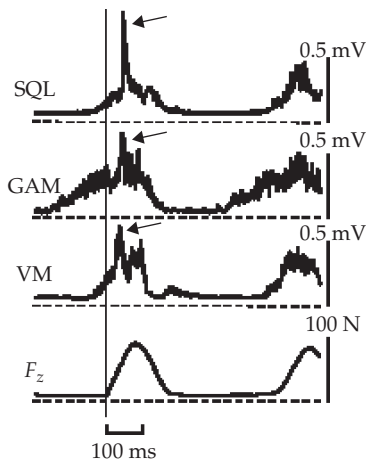


Figure 2.9 Short contact SSC type hopping introduces clear bursts in rectified EMG records. This representative example is from a DJ performed from 60 cm height. Timing of the sharp EMG reflex peak occurs within 40–45 ms after the initial ground contact corresponding to a SLC in Lee and Tatton classification (1982). The reflex EMG peak (indicated by an arrow) in these jumps is usually very clear in the soleus (SOL) muscles, but can be identified for the gastrocnemius (GAM) and vastus (VM) medialis muscles as well. F_z signifies the vertical GRF. (From Komi & Gollhofer, 1997.)

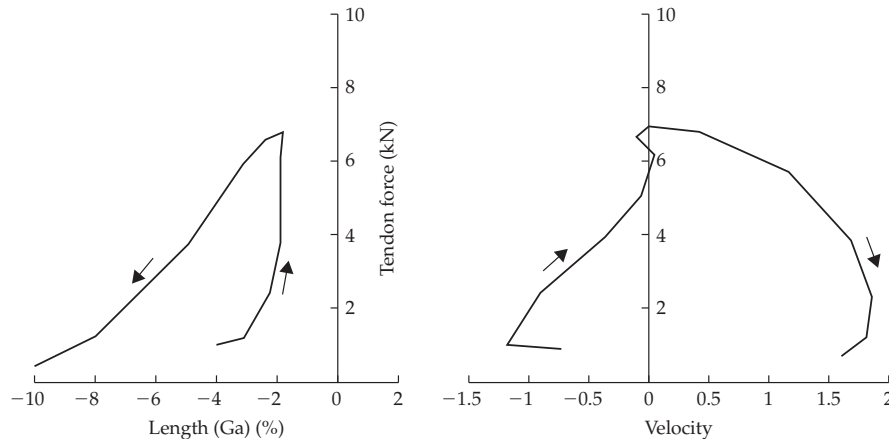


Figure 2.10 Instantaneous force–length and force–velocity curves of the gastrocnemius muscle for stretch–shortening cycle when the subject ran at a fast speed ($9 \text{ m} \cdot \text{s}^{-1}$). The upward deflection signifies stretching (eccentric action) and the downward deflection shortening (concentric action) of the muscle during ground contact. The horizontal axes have been derived from segmental length changes according to Grieve *et al.* (1978). (From Komi 1992.)

Demonstration of Stretch Reflex Intervention in SSC

It was the technique of averaging the rectified EMG bursts, which helped realizing true existence of stretch reflex in locomotion. The first evidence for the short-latency stretch reflex (SLR) component in the *GAST* muscle came from the study of Dietz *et al.* (1979). It was later confirmed by Fellows *et al.* (1993), who used the ischemic blocking method to isolate the Ia afferent information acting on spinal pathways during moderate speed running (Figure 2.11). During ischemic blocking, the *GAST* EMG activity was dramatically reduced during the contact phase, but there was no change in preactivation. The control (nonischemic) runs demonstrated that the *GAST* had a clear stretch reflex component during the contact phase, with the average EMG activity being 2–3 times higher than the activity during maximal voluntary isometric plantar flexion. Furthermore, it was shown that under conditions of submaximal running, the stretch reflex contribution to the global EMG increased with increasing running speed. This should not be interpreted to mean that further increase in running velocity will always result in increased SLR component.

Since these pioneering studies of Dietz and collaborators, other studies have noticed that a commonly observed EMG burst in running and hopping

signifies intervention of Ia afferents in SSC activities. The short-latency component (SLC), appearing about 40ms after the ground contact, is visible in all examined muscles and is especially strong in the soleus (SOL) muscle. These records were obtained by averaging the rectified EMGs over several trials involving two leg hops with short contact times. Appearance of these reflex components is a very common and repeatable observation (Komi & Gollhofer, 1997; Figure 2.9). It is true, however, that in normal movements with high EMG activity, the magnitude and net contribution of reflex regulation of muscle force is methodologically difficult to assess. This has led to much controversy about their functional contribution to the produced force and power output. This controversy may sound unjustified especially if references are made to the classical work of Hoffer and Andreassen (1981). The authors demonstrated that when reflexes are intact, muscle stiffness is greater per same operating force than in an areflexive muscle (Figure 2.12). This could be a logical consequence of how muscle spindles and GTO operate in the control of muscle length and tension (Houk & Rymer, 1981). This leads us to the hypothesis that stretch reflexes may make a net contribution to muscle stiffness already during the eccentric phase of SSC, provided that the time constraints do not limit this to take place.

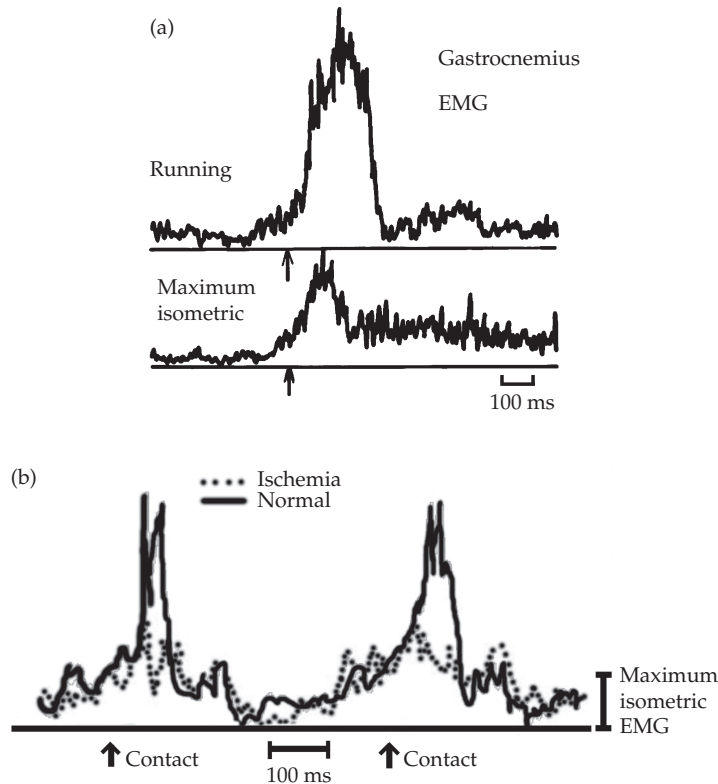


Figure 2.11 Functional significance of stretch reflex during treadmill running. (a) Maximal electromyography (EMG) of the gastrocnemius muscle was first recorded during forceful isometric plantar flexion movement. Thereafter, the same subject ran on the treadmill at 70% of maximum speed. Note that during submaximal running, the recorded EMG from the gastrocnemius muscle exceeds that of the maximum isometric condition. (b) The same subject was then running on the treadmill after the blood circulation was occluded by compression of 20 min. The recorded EMG during running was then at the same level as in maximal isometric condition without blood occlusion. The dramatic increase in (a) was explained by the contribution of the stretch reflexes to the global EMG activity. (According to Dietz et al., 1979, used with permission.)

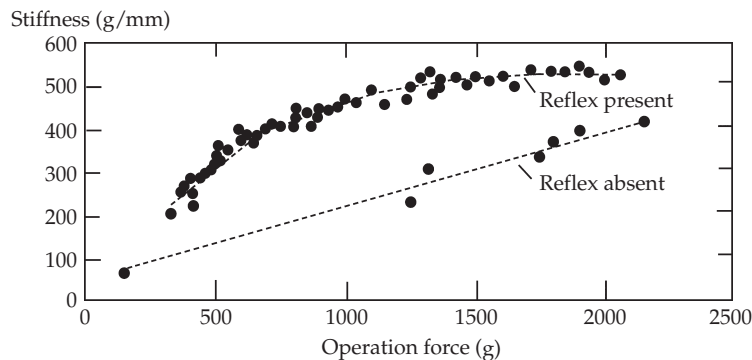


Figure 2.12 Muscle stiffness versus force in the cat SOL muscle. When the stretch reflex is intact, the stiffness, measured as the increase in force for a small stretch, is a sharply rising function of force at the low end of the force range, but remains nearly constant at moderate and high forces. By contrast, when the reflex is eliminated by cutting the efferent SOL nerve and electrically stimulating the cut end at 10–50 Hz to maintain tension, the isolated muscle shows a lower stiffness which is a steadily rising function of force (lower curve). (According to Hoffer & Andreassen, 1981, used with permission.)

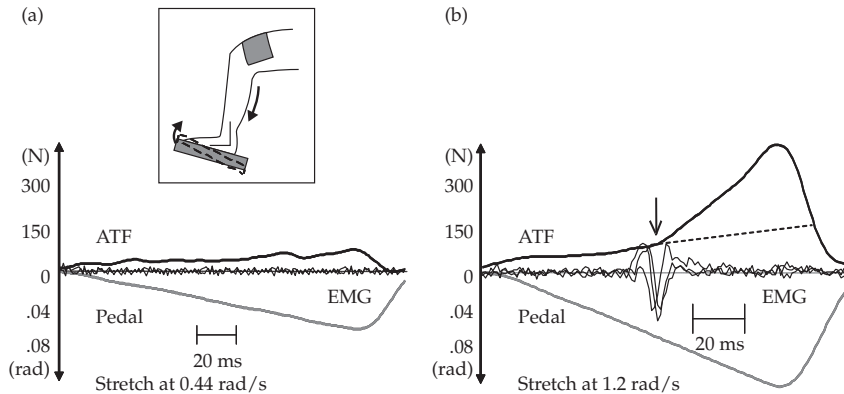


Figure 2.13 Demonstration of passively induced stretch reflexes on the Achilles tendon force (ATF). (a) Passive dorsiflexion at slow stretch caused no reflex electromyographic (EMG) response and led to a small and rather linear increase of the ATF (pure passive response). (b) In case of faster and larger stretches, the reflex contribution to ATF corresponds to the additional ATF response above the pure passive influence represented by the dashed line. The vertical arrow indicates the beginning of the reflex-induced mechanical response (From Nicol & Komi, 1998, used with permission.)

Do Stretch Reflexes Have Time to be Operative in SSC?

The possible effective role of stretch reflexes has been questioned by the argument that the time constraints limit their possibility to have any functional meaning during the stance phase of running for example. A first counterargument came from our studies (Nicol & Komi, 1998) in which the Achilles tendon force (ATF) was quantified in vivo using the buckle transducer technique during pure passive dorsiflexions (0.06–0.12 rad of amplitude and 0.44–1.9 rad/s of mean velocity) induced by a powerful ankle ergometer (Figure 2.13). This passive stretch situation was convenient to identify the stretch reflex EMG and the subsequent mechanical response. During the fastest stretch, the ATF started to increase clearly 10–13 ms after the onset of reflex EMG response (Figure 2.13), showing stretch reflex-induced force enhancement between 200% and 500% over the pure passive stretch response (without a reflex EMG response). The subsequent use of the optic fiber (OF) technique (Nicol et al., 2003) gave similar results.

In order to prove the true existence of stretch reflexes in SSC activities, the best methodological choice would be to study directly the length changes of muscle spindles. This is most probably not yet possible at least in human locomotion.

However, the new developments in US have made it possible to study in vivo the length changes of the fascicles in vivo and also in very fast movements. As the fascicles represent the contractile extrafusal fibers, their length changes should reflect parallel changes in the intrafusal muscle fibers. In fact, the earlier attempts with US gave the impression that especially the MG fascicles were not rapidly stretched during the contact phase in walking and running (Fukunaga et al., 2001; Ishikawa et al., 2003, 2005; Kawakami et al., 2002), for example. These findings were obtained with relative low US “scanning rate” between 25 and 50 Hz. Since then, the US scanning rate has been considerably increased (96–196 Hz). This technical improvement revealed a clear short-lived stretch of the MG fascicles during the very early stance phase of running (Figure 2.14) (Ishikawa et al., 2007; Ishikawa & Komi, 2007), resulting in the occurrence of SLRs.

In the synergist SOL muscle, the muscle fascicles are continuously stretched during the braking (MTU stretching) phase and the timing of the resulting stretch reflex seems to be the same in different conditions. However, timing of the short-lived MG fascicle stretch can differ between different conditions (Ishikawa & Komi, 2007). When compared at two different running velocities (6.5 m/s vs.

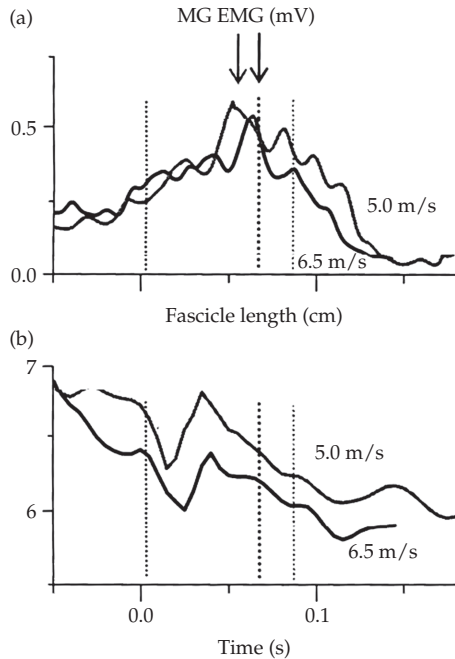


Figure 2.14 Electromyographic (EMG) activity (a) and fascicle length changes (b) of the medial gastrocnemius (MG) muscle before and during the ground contact of two different running speeds. The first dotted vertical line represents the moment of ground contact. The two other dotted vertical lines show the ends of the MTU stretch, which correspond to the end of braking phases of two running speeds, 6.5 and 5.0 m/s. The arrows show the stretch reflex activity. The curves are from one subject only but demonstrate a general pattern across subjects. (Adapted from Ishikawa & Komi, 2008, with permission.)

5.0 m/s), MG fascicles showed a sudden stretch in both conditions, but a slightly delayed timing at the faster one. The MG fascicle stretch occurred approximately 26 ms after ground contact (18 ms in the 5.0 m/s condition), and the corresponding peak SLR occurred approximately 69 ms (56 ms in the 5.0 m/s condition) after ground contact. The end of the braking phase was approximately 68 ms and 87 ms after ground contact in the 6.5 and 5.0 m/s condition, respectively (Figure 2.14). When we consider the electromechanical delay (10–15 ms) between the onset of SLR activities and the mechanical response (Nicol & Komi, 1998), the SLR activities can still contribute to force enhancement during the push-off phase in the 6.5 m/s condition. Consequently,

the results imply that the MG SLR during the stance phase of running either influences fascicle stiffness in the braking phase of slower speed running (5.0 m/s) or stretch-induced force potentiation during the push-off phase of faster running (6.5 m/s) and that the contribution of the stretch reflex can be specific depending on running speed. The occurrence of a sudden MG fascicle stretch during the braking phase of running is a unique but expected finding, and is in accordance with the logical nature of the stretch reflex contribution.

What is the Functional Significance of Stretch Reflexes in SSC?

Despite the possibility to record *in vivo* the muscle-tendon force in a reliable way, magnitude of the reflex contribution to stiffness and force enhancement of tendon-muscle complex is largely unknown in normal locomotion. This is mostly attributed to the difficulty to differentiate the reflex-mediated force response from the resistive force of the intrinsic CCs and from the passive tissues and relaxed muscle fibers.

A partial answer to this question came from the comparison in a given subject of direct ATF measurements (with the buckle) in pure passive stretch conditions and in hopping (Nicol & Komi, 1998). This study revealed that the highest reflex-induced ATF recorded on the ergometer corresponded to 6.7% of peak ATF (2750 ± 51 N) recorded in hopping. In normal running and hopping, even when performed submaximally, the reflex contribution should be much greater due to substantially larger number of motor units receiving Ia afferent stimuli from the condition of relatively high stretch velocities (10–12 rad/s) at the ankle joint. The stretch velocity is naturally expected to play a role in inducing the stretch reflex EMG, although the human experiments have utilized stretches (2.0–4.0 rad/s) that were 5–10 times lower than those occurring in natural forms of locomotion. (Gollhofer & Rapp, 1993; Gollhofer et al., 1995) As shown in Figure 2.15, the subsequent data obtained with the OF technique while introducing a long plateau duration in the pedal movement between the two successive stretches revealed that the ATF continues to rise until about 100 ms (Nicol & Komi, 1998; Nicol et al.,

2003). This is in line with the twitch contraction time reported earlier for the SOL and GAST muscles (McComas & Thomas, 1968; Sale et al., 1982). In the absence of reflex EMG response at slow stretching velocity (upper graph, Figure 2.15), the force–stretch curves reflected the pedal movement, but demonstrated a steeper ATF rise during the second stretch as compared to the first one. Although the two mechanically induced stretches were very similar, the second one started from a 3% more dorsiflexed position, emphasizing the potential influence of the tendon compliance on the recorded ATF.

During various forms of locomotion, the stretch reflex mechanical response takes place while the muscle is actively stretched. This situation can be mimicked in the laboratory by introducing the second stretch at the different rising and decreasing phases of the first twitch mechanical response (twitch) curve (Figure 2.16). The rising phase of the twitch response should correspond to the attach-

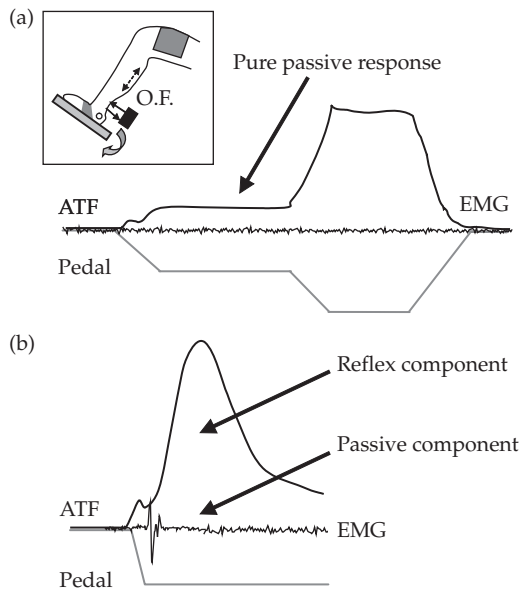


Figure 2.15 Passive dorsiflexions were induced by the ankle ergometer while the Achilles tendon force (ATF) was recorded in vivo using the optic fiber (OF) technique. At low stretching velocity (a), the absence of reflex response resulted in quantification of the “pure passive ATF response to stretch” during the plateau phase. This was used at higher stretching velocities to isolate the “pure reflex component” (b). EMG, electromyogram. (Nicol et al., unpublished observations.)

ment phase of most of the involved cross-bridges, whereas the decreasing phase should correspond to the detachment of most of them. The results show that when the second stretch occurred during the rising phase of the first stretch-induced twitch contraction, it led to clear enhancement of the rate and peak of ATF (Figure 2.16a). However, when the stretch occurred at the onset of the decreasing phase of the mechanical reflex response, the combined effect was reduced as compared to the expected summation of the reflex and stretch effects (Figure 2.16b). The combined stretch and reflex potentiation very likely depend on the sarcomere kinetics, so that the resistance to stretch is particularly efficient during the initial part of the reflex response as discussed earlier for the sarcomere level situation (Edman, 1980). This concept needs naturally to be studied further, but the explanation is attractive as it is in line with the short-range elastic stiffness hypothesis according to which the cross-bridge resistance to stretch is especially efficient during the early part of the cross-bridge attachment (Edman, 1980). Therefore, the rapid reflex-induced cross-link formation could play a substantial role in the force generation during stretch.

Thus, evidence exists that stretches in the early contact phase of SSC actions, such as running and jumping, are powerful enough to induce sufficient muscle spindle afferent activation. This would also mean that stretch reflexes are contributing to the efficacy of motor output by making it more powerful. According to Voigt et al. (1998), the combination of the “pre-reflex” background activation and the following reflex activation might represent a scenario that supports yield compensation and fast RFD. The concept of elastic storage favors also the existence of reflex activation since high muscular activation during the braking phase of SSC is a prerequisite for efficient storage of elastic energy. All these aspects may contribute to the observation that ME in natural SSC is higher than in pure concentric exercise (Aura & Komi, 1986; Kyröläinen et al., 1990; see also Chapter 7).

Task-Dependent Modulation of the Reflex Gain

As discussed earlier, the common finding of many DJ studies is that, as the height of the drop preceding

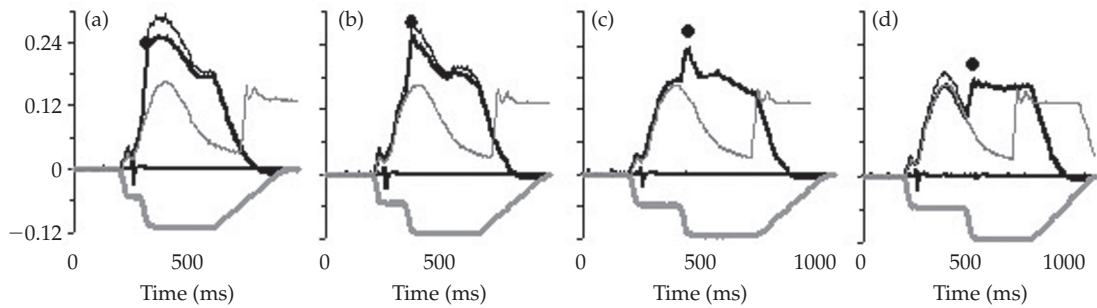


Figure 2.16 ATF potentiation when the second stretch occurs during the rising phase (a and b) or during the decreasing phase (c and d) of the reflex-induced mechanical response. Individual ATF records are presented as mean + standard deviation (SD) of seven trials. The exact summation of the reflex and stretch effects are represented by dots that can be compared to the actual combined effects. (Nicol et al., unpublished observations.)

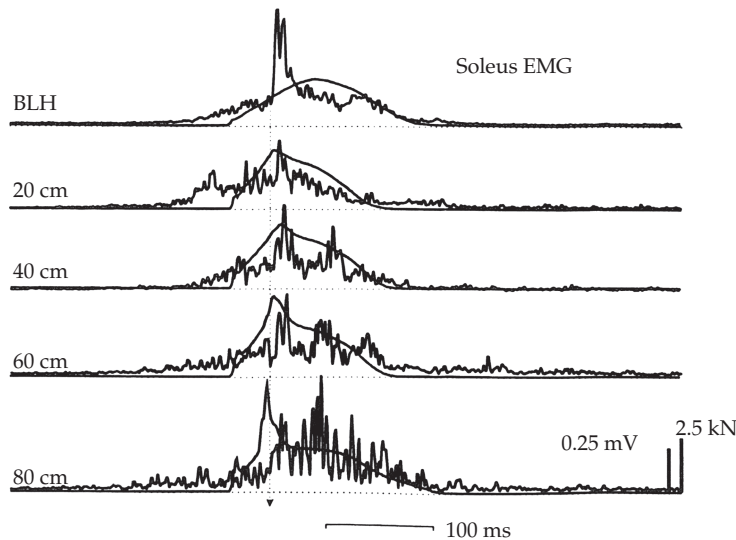


Figure 2.17 Rectified and averaged electromyographic (EMG) pattern of the SOL muscle and vertical GRF in both leg hopping (BLH) and in various DJs from different dropping heights (20–80 cm). The figure illustrates the modulation in the EMG pattern and in the force record with increasing stretch load. The broken vertical line indicates the initiation of the phasic activation with a latency of 40 ms after ground impact. (Adapted from Komi & Gollhofer, 1997.)

the rebound is increased, performance can initially improve (Asmussen & Bonde-Petersen, 1974; Bosco et al., 1981), but eventually will decrease (Komi & Bosco, 1978). In DJs (Figure 2.17), the short-latency reflex (SLR) response showed higher amplitude with increased drop height from 20 to 40 cm and 60 cm (Komi & Gollhofer, 1997). However, in jumps from excessive heights (80 cm), the SLR was diminished. Despite larger impact loads and higher stretch velocities, the SLR was decreased suggesting decreased

facilitation from muscle spindles and/or increased inhibitory drive from various sources such as GTOs or voluntary protection mechanisms (Komi & Gollhofer, 1997). Changes in excitability of the Ia afferent pathway could occur at the spinal level or could be induced by an altered fusimotor drive.

To our knowledge, direct information regarding the fusimotor drive during jumping is lacking, although the H-reflex at the spinal level has been recently examined (Leukel et al., 2008). In this study,

H-reflex excitability during the SLR component was compared in the landing phase of DJs from excessive and normal heights. H-reflex excitability and H/M ratio were found to be reduced at SLR at excessive heights as compared to normal heights. The results support the earlier hypothesis of a “prevention strategy” to reduce eccentric stress on the tendomuscular system (Schmidtbleicher & Gollhofer, 1982; Komi & Gollhofer, 1997). Presynaptic inhibition (PSI) of Ia afferents was thought as most likely responsible for the change in H-reflex excitability between the two jump conditions (Leukel et al., 2008). This may explain also a common finding that in DJs with high stretch loads, neuromuscular inhibition is often observed prior to reflex activation (Figure 2.18) (Gollhofer et al., 1992). Similar reflex behavior has been observed in cats (Prochazka et al., 1977).

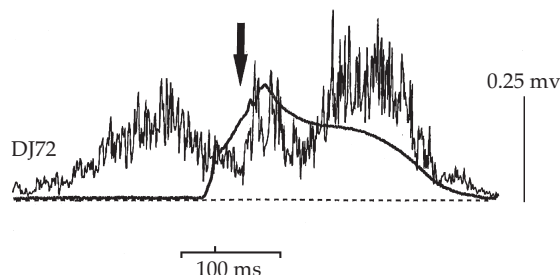


Figure 2.18 Rectified and averaged EMG pattern of the gastrocnemius muscle and vertical GRF of a drop jump (DJ) from 72 cm height. Note that the EMG profile shows a clear reduction in amplitude already prior to contact, which is consistent until the mid-phase of contact. The phase of reduction is dispersed by a phasic EMG contribution (see vertical arrow) occurring 43 ms after the ground impact. (From Komi & Gollhofer, 1997, with permission.)

Final Comments

The chapter discussed the phenomenon of SSC, which is the natural way of muscle function in normal locomotion. The SSC is actually the nature’s way to combine the available resources, CC, elastic structures, and central and reflex activation profiles, in such a way that both the peak performance (rate and amplitude of the force production) and movement economy are considered in most appropriate way in each particular movement situation. The

fascicles and tendons (through their interaction) are the major “players” in this task. SSC and its potential to improve performance have been clearly demonstrated both with isolated neuromuscular models and with studies in humans. The consequences are obvious and have resulted in numerous attempts to apply the SSC concept for performance enhancement and improvement of economy in various sport activities. Chapters 3–6 will discuss these issues as they apply directly to the specific sport disciplines.

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Chapter 3

Utilization of Stretch-Shortening Cycles in Cross-Country Skiing

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Introduction

High-performance cross-country ski racers give the impression of very smooth movements of the legs and arms throughout the stride pattern regardless of the technique in use—classical diagonal stride, some version of ski skating, or double poling. To the naked eye, some skiers seem to bob slightly at the torso or bounce a little during each step during diagonal stride but because the pattern is repeated on every cycle, the movement still looks smooth. In fact, “smoothness” in movement patterns is often seen as a characteristic of highly skilled performance in many sports and coaches can frequently be heard to instruct their athletes to “smooth out the movement.”

However, viewing of video, particularly in slow motion, will sometimes show a small flexion of the shoulder (angle of upper arm relative to the torso) and elbow upon pole plant just before the pole thrust begins, and at the hip, knee, and ankle just before the onset of the strong leg push. Some skiers can also be seen to use a slight trunk flexion that precedes extension of the arms and legs and becomes an obvious trunk extension or even a hyperextension (angle of the trunk relative to the thigh of the pushing leg). When observed in this

detail, the movement pattern for some skiers is not as smooth as it seems in real time rather than in slow motion.

The authors believe that there are neuromuscular and biomechanical benefits to skiers who show these brief and rapid flexion/extension patterns at some joints. It maybe possible for these skiers to produce higher muscle and GRFs, or the same forces as other competitors—but at lower metabolic cost.

Are these simply idiosyncratic movement patterns that started at an early age for some skiers that gradually became “grooved” as they evolved from young successful racers into world-class competitors over the years? Are these uses of body joints purposely adopted by some skiers by trial and error learning as they are developing because they find that they ski faster or more efficiently when they do this? Are some competitors actually taught by some coaches who are very familiar with neuromuscular physiology and biomechanics to ski this way? These patterns are not used by all skiers, not even by all world-class skiers. Is there now sufficient evidence for the feasibility and benefits of using muscles and joints in this way in cross-country skiing to recommend that all young competitors be taught to use this technique?

These repetitive extension/flexion patterns are typical of muscle SSC and are commonly observed in sports such as running, jumping, and “windups” in throwing. For example, when children throw balls, even at very young ages, they seem to naturally resort to a kind of “windup” that precedes the

throw—an extension/flexion pattern at the shoulder joint. This pattern usually does not have to be taught for throwing although it can be refined by coaching.

SSCs Reviewed

Although extensively discussed in Chapters 1 and 2, we review here briefly for cross-country skiing—when electrically active muscles across a body joint are stretched while decelerating a joint flexion or extension, the muscle action is called “eccentric” (e.g., shoulder joint, elbow, hip, knee, ankle, lumbar spine joints). If a joint is flexing or extending under the activity of shortening muscles, the muscle action is called “concentric.” In a cycle of fast movement that goes quickly from flexing a joint to extending it after being decelerated by extensor muscles, stopped momentarily, then extended in concentric action by the same extensor muscles, the extensors are said to be undergoing an SSC. In muscle action terms, the movement is said to be an eccentric/concentric cycle (ECC). Of course a joint may also undergo a movement pattern from extension immediately followed by a flexion. The joint flexors, when active, decelerate the extension and begin the flexion. While the joint is extending, the active flexor muscles are stretching but when these joint flexors begin to close the angle at the joint they start to shorten. Again we have an SSC or ECC.

Some interesting things are known about the differences between concentric and eccentric muscle actions that are summarized in Table 3.1.

Table 3.1 Functional differences between concentric and eccentric muscle actions

Higher force potential in ECC, therefore higher performance.
Lower metabolic cost in ECC for the same absolute work load or power, therefore higher metabolic efficiency in ECC.
Higher mechanical work output in ECC at lower metabolic cost; therefore higher mechanical efficiency.
Repeated use of ECC actions in running results often in muscle pain and damage. However, this is less frequently observed in cross-country skiing, because the impact loads are much lower than in running, for example.

ECC, eccentric/concentric cycle.

Evidence of Use of SSC in Cross-Country Skiing

To confirm the possible presence of ECC (SSC) in cross-country skiing, a strong pushing (thrusting) activity from the arms and legs for propulsion, it is necessary to show, at least, an angular displacement or angular velocity curve at a joint (e.g., hip joint) that goes quickly and smoothly from a flexion to an extension and EMG activity in the extensor muscles during both the flexion and extension phase, i.e., active not passive extensor muscles at that joint. Angular velocity curves derived from the displacement data that change from positive to negative or vice versa, together with EMG activity data are easier to follow regarding the possibility of SSCs. Slightly less direct evidence is demonstration from GRF data of a quick unloading of the leg or pole (reduction in GRF) immediately followed by an increase in loading and EMG activity in the extensor muscles during both the unloading and loading phase. Direct measures of SSC-type length changes in a single MTU and/or muscle fascicle, the active part of this unit, would be most convincing.

Komi and Norman (1987) reported some kinematic angular velocity data at the shoulder, hip, and knee, GRF data on the skis and poles, and EMG data on world-class skiers. They showed the possibility of utilization of SSCs or ECC by some skiers, but not by all that they analyzed. However, the evidence was on only a few skiers and incomplete at the time. Unfortunately, they were able to collect only kinematic data from frame by frame film analysis and use of a linked segment biomechanical model of competitors at world-class competitions but not EMG data during these events. They did record EMG and GRF data in a controlled environment subsequently but not kinematic data at that time. They suggested that there are physiological and biomechanical advantages to skiers who use their muscles this way, deliberately or by accident of how they happened to develop their skiing techniques as they rose to international class.

Kinematic Evidence of Use of SSC

Figure 3.1 is a computerized linked segment model of a skier from which positions of joints, their

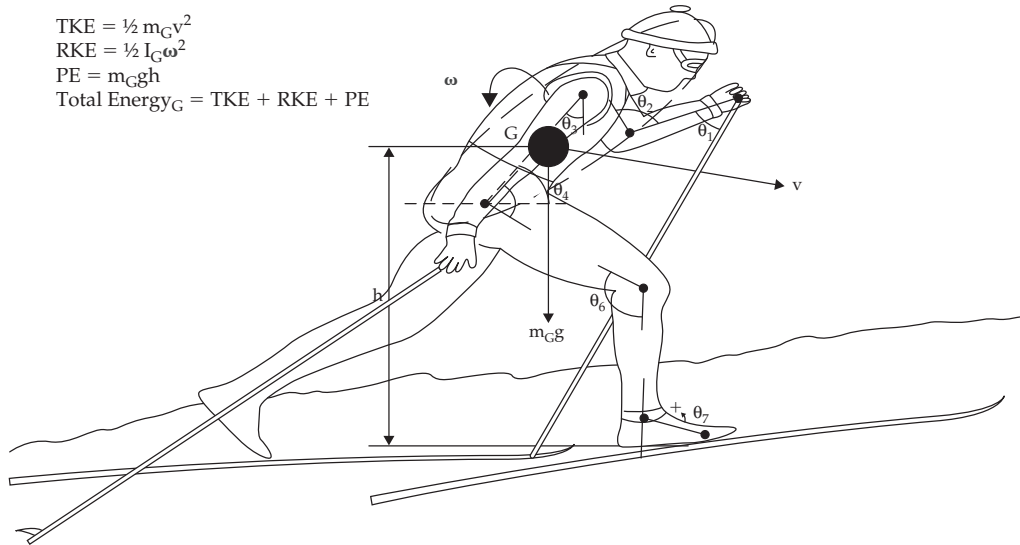


Figure 3.1 Linked segment biomechanical model of a skier. TKE, RKE are translational and rotational kinetic energy of a body segment, respectively; PE is potential energy of body segment above a baseline (ski track); h is height of G above a baseline; m is mass of a body segment; G is CoG of a segment; I_G is moment of inertia of segment about G ; g is gravitational acceleration constant; v is velocity of segment in translation; ω is velocity of segment in rotation.

displacements, velocities, accelerations, mechanical energy levels, and other useful analytical variables can be measured, calculated, or estimated.

Figure 3.2 shows how one world-class skier, J.L., uses an apparent SSC at both his hip and knee joints on a 9.0° uphill in a 15km world championship competition in Lahti, Finland in 1978. These flexion/extensions and possibly those at the ankle joint and frictional forces produce oscillations in the velocity of the CoG of his body. The data were obtained using the model in Figure 3.1.

Figure 3.3 is a comparison of J.L. and M.P., two highly successful world-class skiers, of their use of their hip joints in propulsion. J.L. finished first in this 15km World Cup race. M.P. finished third.

The skiers used their hip joints substantially differently on the same uphill. Just prior to extension of his hip joint, J.L. quickly flexes that joint (negative velocity on the curve shown by the dark hatched area), presumably during activation of the hip extensors to slow, stop, and reverse the movement (positive velocity on the curve). This appears

to be a good example of a skier who uses an SSC at the hip joint. In comparison, M.P. has a much smoother hip angular displacement curve that results in a considerably smoother angular velocity curve than that of J.L. However, there is no prestretch phase of hip extensor muscles in M.P.'s pattern, therefore, no SSC.

Table 3.2 compares these two skiers in somewhat more detail just prior to and during the pushing phase of a leg and a pole during a step cycle.

Note that J.L. shows a flexion velocity prior to the extension (pushing) motion at all three joints. M.P. has no preloading flexion at all at the hip. The subsequent extension velocity at the hip for J.L. is much faster than that of M.P. The elbow flexion velocity of J.L. is of much shorter duration and much faster than that of M.P. At the knee, flexion and extension velocities are similar but the duration of flexion of J.L. is much shorter. The graphs and the table of data seem to show better use of the SSC by J.L. particularly at the hip joint, a major source of leg thrust. However, these are data on only two skiers.

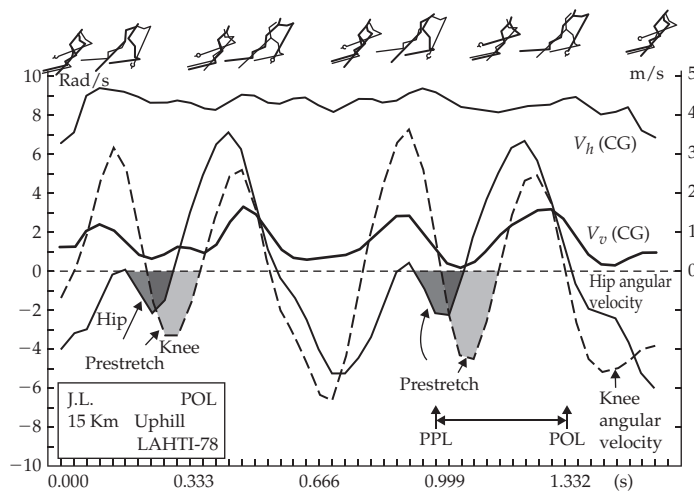


Figure 3.2 Horizontal velocity (V_h) and vertical velocity (V_v) of the body CoG together with the angular velocity curves of the hip and knee. The hatched areas represent the parts of the cycle where the extensor muscles of these joints are assumed to be prestretching (negative velocity) just prior to propulsion extension (positive velocity). PPL denotes the left pole plant and POL denotes the pole off the snow points. (From Komi & Norman, 1987.)

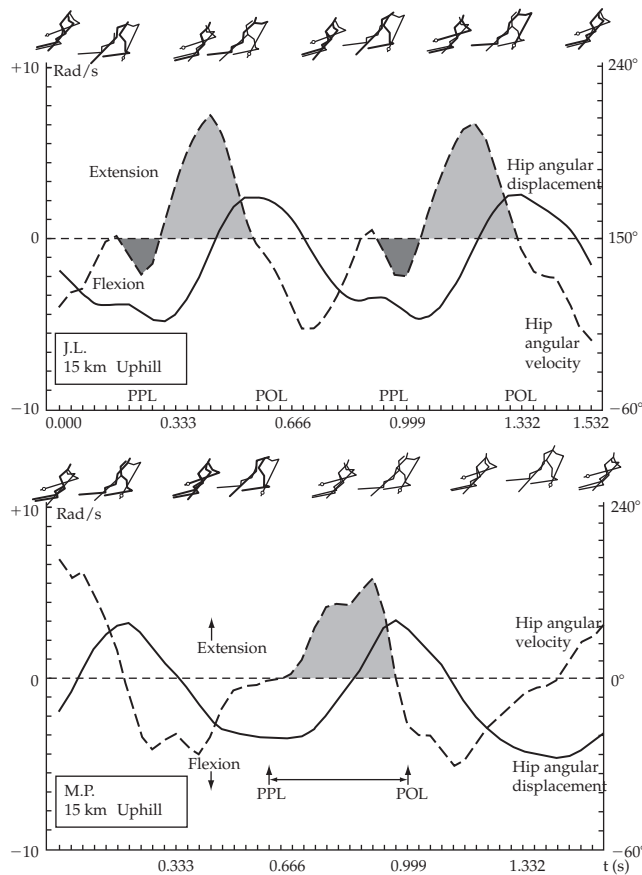


Figure 3.3 Comparison of hip angular displacement and velocity curves of two elite skiers. PPL denotes the left pole plant and POL denotes the pole off the snow points. (From Komi & Norman, 1987.)

Table 3.2 Selected kinematic data of two skilled skiers J.L. and M.P. The elbow action of flexion and extension is for the period of pole push. Knee action occurs during the leg (ski) push and hip action slightly precedes but occurs mostly during pole push

	Hip joint		Knee joint		Elbow joint	
	J.L.	M.P.	J.L.	M.P.	J.L.	M.P.
Range of motion						
Flexion	112–105°	0	0	0	120–80°	93–80°
Extension	105–172°	118–180°	140–120°	153–130°	80–154°	90–154°
Flexion velocity, maximum (rad/s)	–2.1	0	–3.7	–3.3	–4.8	–1.5
Extension velocity, maximum (rad/s)	7.2	5.9	5.4	5.5	12.0	11.4
Duration of flexion (ms)	118	0	137	181	118	198
Duration of extension (ms)	268	320	179	151	179	203

(From Komi & Norman, 1987.)

Figure 3.4 is an overlay of hip joint velocity curves of five world-class skiers including J.L. and M.P. but on a flat part of the course, not on an uphill. In the stride presented in this figure, all five skiers show a brief hip joint flexion prior to extending the joint in thrust, possibly using an SSC at this joint.

EMG and GRF Evidence of Use of SSC

Kinematic description of joint movements and velocities can be complemented with other simultaneous recordings. For example, use of the GRF and EMG activity measurements together with the kinematics can substantially improve the accuracy of SSC prediction, especially for the functional phases such as the propulsive thrust and the preceding gliding phase on skis. The ground contact phase is easy to examine in running, but requires special and quite costly equipment for skiing. Komi was able, with the help of the able engineers in his laboratory, to be the first one to report construction of a force platform system designed to record GRFs during normal snow conditions outdoors (Komi, 1985). This system consisted of 4 rows of 6m long force plates that when embedded into the snow could reliably record GRFs of both skis and poles simultaneously. The system gave considerable new information with regard to ski and pole forces

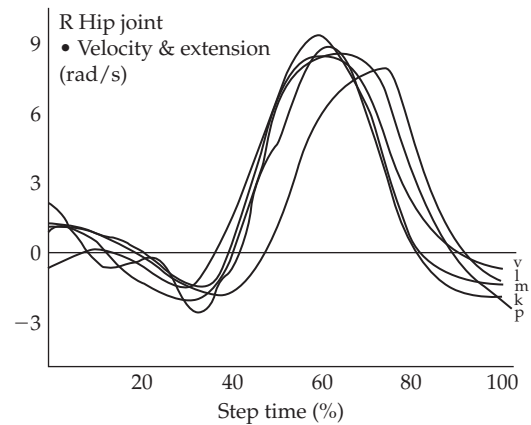


Figure 3.4 Hip joint velocity curve overlays of five skiers on a flat part of the course during a 15km World Cup race. The possible prestretch phase occurs when the curve is negative, just prior to becoming positive. The curves are similar but not the same, particularly in the prestretch phase. (From Komi et al., 1982.)

during diagonal skiing (Komi & Norman, 1987). The system was later (Vähäsöyrinki et al., 2008) expanded to a 20m long system permanently installed into the ski tunnel of the Vuokatti Sport Institute in Finland (Figure 3.5).

The lowest curve in Figure 3.6 shows a representative set of ski force recordings made with this

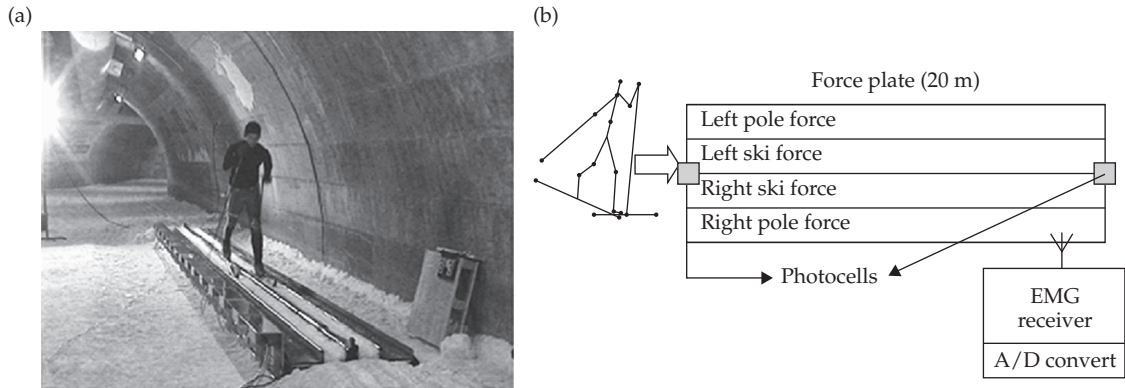


Figure 3.5 (a) The force plates were placed in the special indoor ski tunnel at 2.5° slope. (b) A schematic view of the force plate configuration. EMG, electromyogram. (From Vähäsöyrinki et al., 2008.)

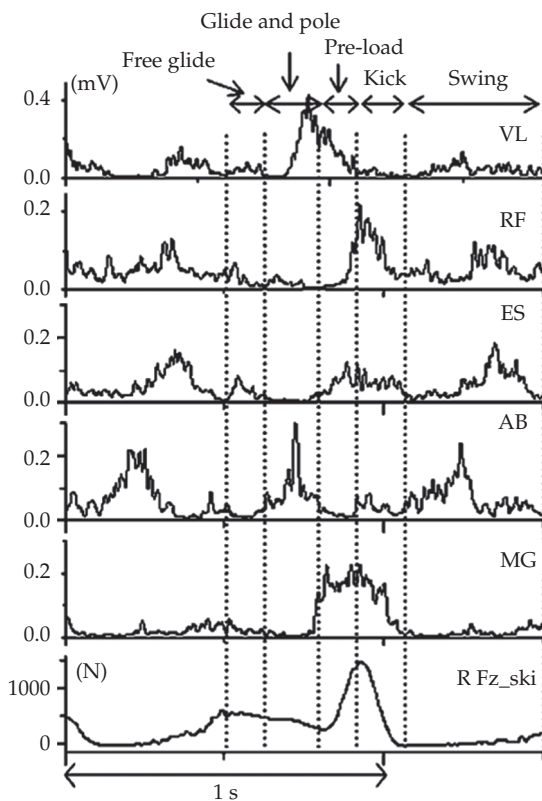


Figure 3.6 Representative examples of EMG activities from many muscles as they appear together with the GRF patterns of skis and poles. VL (vastus lateralis), RF (rectus femoris), ES (erector spinae), AB (abdominalis), MG (medial gastrocnemius), R Fz_ski (right ski vertical force). (From Vähäsöyrinki et al., 2008.)

newer longer force plate system. The other five curves are EMG patterns from relevant trunk and leg muscles. To assist the understanding of SSC, the ski force curve can be used to identify different functional phases indicated by fluctuations or changes in slope in this vertical ski force measured from the right side (the same side as the EMG electrodes were attached). It must be noted that these forces represent the “global” pattern, and can therefore express the overall possibility for the occurrence of SSC during the gliding and subsequent push-off phase. Thus, this information alone is meaningful to understand that the diagonal skiing is indeed an activity where the “preloading” and “braking” phases precede the final kick (thrust) phase.

If SSC is being used, EMG data should show muscle activity in extensors of the hip, knee, ankle, and trunk, not only during the extension phases of the movements when one would expect to see it, but also during the brief flexion phases that precede the extensions. That is to say that the flexions must be controlled by extensor muscles to decelerate the joint flexion, stop it, and turn the action at the joint into a strong extension. This EMG pattern is clearly shown in Figure 3.6, particularly for VL, erector spinae (ES), and MG which are all very active in preload and to some extent for rectus femoris (RF).

From these muscles, the VL muscle begins its activation earlier than the other two muscles.

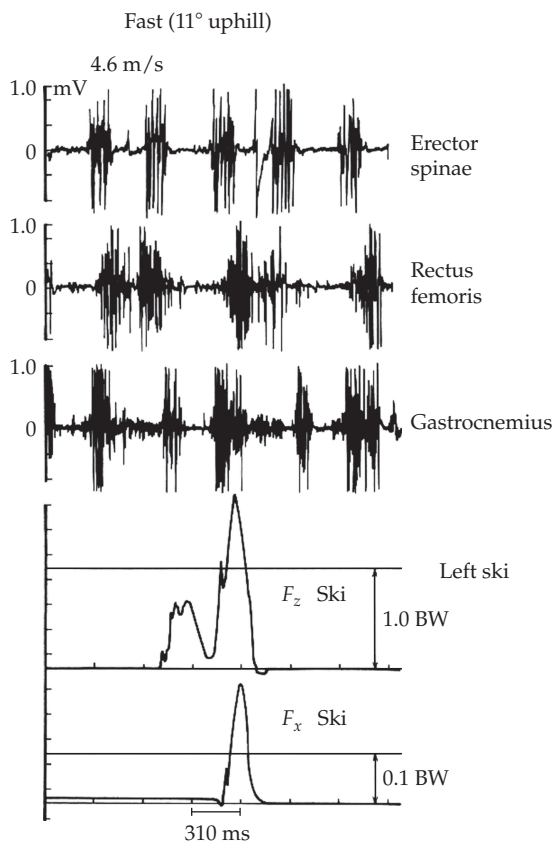


Figure 3.7 EMGs of three extensor muscles together with vertical (F_z) and horizontal (F_x) GRFs on a steep uphill (11°). (From Komi & Norman, 1987.)

Interestingly, the ES and abdominal muscles show a nice coordinated activity pattern that explains well the hip flexion–extension motion and angular velocity curves of the skier J.L. shown earlier in Figure 3.3.

The EMG activities in Figure 3.6 were recorded from a high-level cross-country skier, and can thus be used as a model of representative EMG patterns. Komi and Norman had earlier observed for three separate extensor muscles (see Figure 3.7) that the ski forces and EMGs fluctuate in a very coordinated and efficient way. Note from Figure 3.7 how the EMG activities of the trunk extensors (ES), knee extensors (RF), and ankle extensors (GAST) are timed with the occurrence of the unweighting of the

ski. These muscles are the major extensors contributing to the thrust in the skiing stride. These flexion/extension patterns shown earlier in Figures 3.2 and 3.3, particularly of the heavier body segments such as the trunk and thigh, can consequently be seen as confirmation of the SSC-like behavior by the GRF changes. When forces of the snow reacting on poles and skis are recorded from embedded transducers or from very long force plates, the effects of these body joint flexion/extension cycles are evident first as reductions in force quickly followed by increases in force.

Note that the ES and GAST activities, in particular, clearly begin during the unweighting of the ski, prior to commencement of the thrust that accelerates the skier. The unweighting is seen as the dip in the F_z curve. The forward thrust is seen in the F_x curve. Extensor muscles are active prior to the extension of the joints that they serve, again adding to the evidence that SSC is used in cross-country skiing. Consequently, the evidence collected with kinematic, GRF, and EMG recordings strongly suggests that SSC-like behavior can be identified in diagonal stride of cross-country skiing to occur in the hip, knee, and ankle joints. Specific examination of the timing of these cycles suggest that they are used as a sequential flow of the cycle, occurring during the leg braking push-off phase first in the hip, then in the knee, and ankle joints (Komi & Norman, 1987).

In addition to these recording techniques and data on trunk and leg muscles from our group, the recent work of Lindinger et al. (2009) shows evidence of use of SSC in flexion–extension movement at the elbow joint. They combined the kinematic, EMG, and pole force (load cell instrumented poles) data in the double-poling technique of cross-country skiing. They recorded the triceps brachii (TB) EMG activity simultaneously with the axial pole force and elbow angular displacement in the double-poling action, which is also used in diagonal cross-country skiing. The pole force and elbow flexion–extension movement clearly showed speed-dependent patterns (Figure 3.8).

The poling time decreased dramatically with increase in skiing speed and resulted in increase of the flexion phase speed of the elbow angle. This

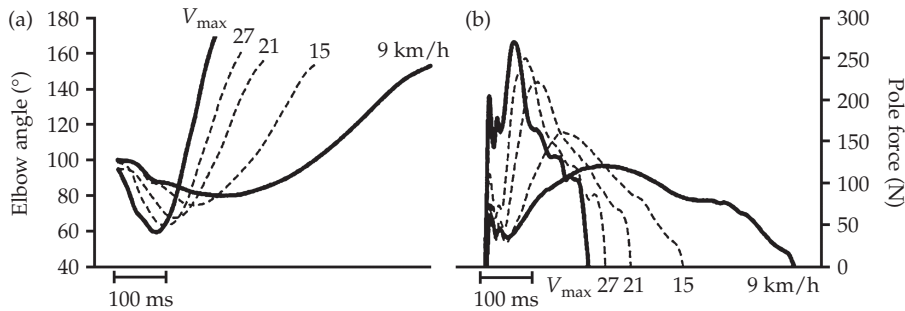


Figure 3.8 Representative elbow angle (a) and pole force (b) curves of one elite cross-country skier across several double-poling velocities from very slow (9 km/h) to maximum velocity (V_{max}). Time courses are mean. (From Lindinger et al., 2009.)

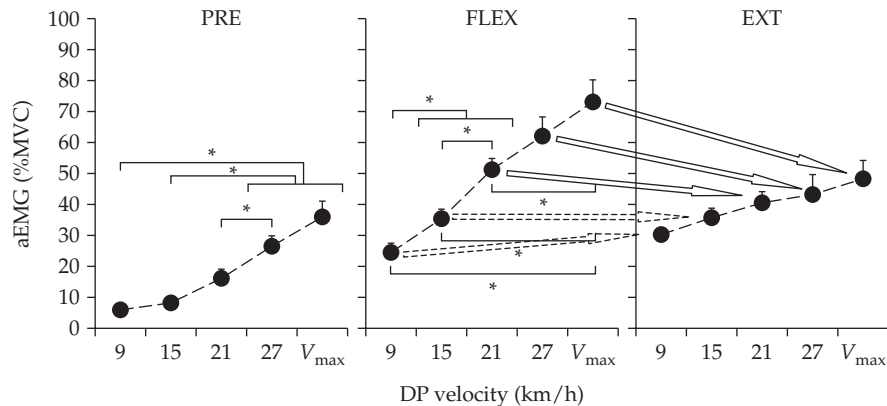


Figure 3.9 Changes in TRI (TB) preactivation (PRE), and activation during flexion (FLEX) and extension phase (EXT) across double-poling velocities (9 km/h, V_{max}). The data are mean + SE. * $P < 0.05$. The solid arrows indicate significant changes and the dashed arrows nonsignificant changes ($P < 0.05$) from the FLEX to EXT phases separately for each velocity. (From Lindinger et al., 2009.)

flexion corresponds to the high-activity phase of the TB muscle. Its activation before the pole plant (preactivation phase) and during the flexion phase (eccentric action) increased dramatically and even much more than in the final elbow extension during push-off (concentric action) of the SSC cycle (Figure 3.9).

These findings are well in line with the basic benefits of the SSC actions that were introduced in Chapter 2. In particular, they refer to the timing of preactivation, short coupling between stretch and shortening, as well as economic use of activation during the concentric phase. Lindinger et al. (2009) also recorded EMG activities from the muscles affecting more the shoulder joint action and found

even more dramatic changes in their EMG activity patterns when the skiing speed increased. As the shoulder joint kinematics could not be measured during these trials, some caution needs to be used in the interpretation of these findings. The observation on the TB behavior is, however, without any doubt an excellent demonstration of its SSC function. The flexion–extension behavior of the elbow joint during pole plant in the manner of SSC has been observed also by other authors, especially in double poling (e.g., Smith et al., 1996).

It is indeed remarkable how expert skiers have learned (or are born with the ability) to activate their muscles during different phases (gliding, braking, and push-off) to obtain low friction in

gliding but high kicking force in the push-off. In fact, instead of naming this phenomenon as SSC, one should more correctly call it braking and push-off phases, in which several muscles (and joints) are involved. This information is, however, important when identifying the possible SSC behavior of individual muscles. Here we give additional examples from abdominal muscles to emphasize how their activities during the hip flexion phase are timed to influence the pressure on skis. The systematic pattern of force fluctuations and the parallel unweighting and loading actions are especially apparent at higher speeds of skiing. Vähäsöyrinki et al. (2008) interpreted that when the average vertical force during “free gliding phase” and “glide and pole phases” decreases with higher skiing speed, this would naturally mean lower ski friction and subsequently better gliding qualities of the skis. The abdominal muscles play an important role here, as their activation is timed to occur with the greater reduction of the ski F_z force from glide to glide and pole phases. Especially at higher speeds this results in rapid downward movement of the CoM during the glide and pole phases. As already mentioned, this rapid movement can be important to produce the braking force effectively during the preload-ing phase.

Ultrasound Measurements on Muscle Fascicles as Evidence to Support the SSC Concept in Diagonal Skiing

The definition of SSC as discussed extensively in Chapter 2 is based on the behavior of the entire “muscle,” that is to say the whole MTU. Consequently, the eccentric phase of the SSC cycle refers to stretching of the whole unit including its tendon. Thus the definition is limited in its possibilities to see how the various parts of the skeletal muscle are functioning during locomotion, e.g., during the contact phase of running and skiing. Human (or animal) muscle is however, more than just a “muscle.” It contains two basic elements: contractile and tensile. In the contractile element, bundles of fibers are called muscle fascicles, and they play an important role in the function of the entire MTU. Limiting the SSC concept to the MTU only

does not necessarily give correct impression of the length changes in the two compartments. Inside a muscle, the fascicles and tendons may not experience similar length changes. Furthermore, synergists can also experience different length changes. For example, SOL and MG show considerable difference in the activation strategy during the simple task of hopping (Moritani et al., 1990). As the fascicles are controlled both by external stretch and internal activation, it may not be realistic to expect a uniform pattern of the fascicle–tendon behavior across muscles and movement conditions.

Ultrasonography is a technique which can be used to study the fascicle and tendon length changes during movements. The reader is referred to Chapter 10 on ultrasound (US) measurements during locomotion for methodology as well as major observations. The chapter does not contain any reference to cross-country skiing, however. This is because until recently, the use of US scanning devices in special conditions of cross-country skiing including the cold environment and requirement for recording of several strides and muscles was not possible. We were fortunate to apply a newly developed, portable US machine (Aloka, SSD Prosound C3cv, Japan) in natural skiing conditions of the Vuokatti ski tunnel.

The US machine with the weight of 5kg was put on the rucksack of the skier and the probe was then attached onto the skin of the following muscles: VL, RF, TB, MG, SOL. Each muscle fascicle behavior during ski was studied separately using the same skiing speeds of approximately 4.5 m/s^1 to represent moderate speed. The slope of the skiing track was set at 2.5° similar to the set up shown in Figure 3.5a. An experienced skier was used as a subject. He was able to control the skiing speed quite well to be the same in all trials, and consequently the skiing speeds were comparable between muscles. The measurements also included EMGs from the same 12 muscles as the US scanning was recorded. The 3D kinematics and the GRF recordings were also performed (for details, see Ishikawa et al., in preparation). The results are summarized in Figure 3.10.

The graphs show that for the VL, RF, TB, MG, and SOL muscles, the fascicle–tendon interaction

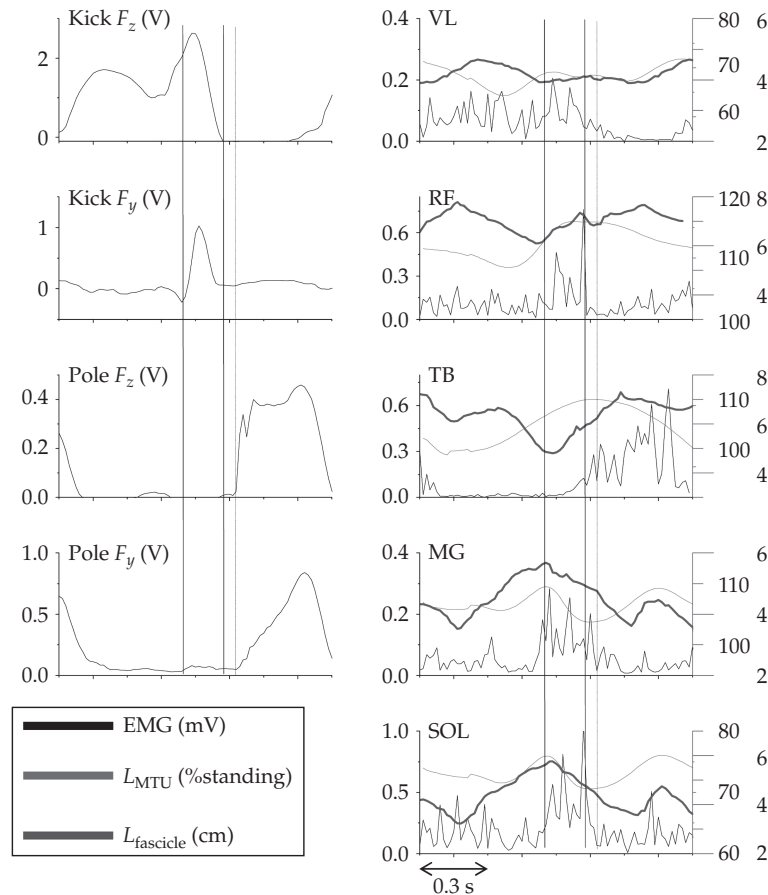


Figure 3.10 Unique records to combine the electromyographic activity (EMG), length of the fascicles ($L_{fascicle}$), and length of the total MTU (L_{MTU}) of the vastus lateralis (VL), rectus femoris (RF), triceps brachii (TB), medial gastrocnemius (MG), and soleus (SOL) muscles together with the vertical (F_z) and horizontal (F_y) forces of the skis and poles as measured in the normal snow conditions in a special ski tunnel equipped with sophisticated instrumentation shown in Figure 3.5.

was similar, i.e., the fascicles of these muscles behaved the same way as observed in the global SSC function. They were stretched in the eccentric phase followed by shortening in the concentric phase. The results were surprisingly uniform across muscles, but were even more pronounced in the case of the triceps surae muscle when the subject used higher skiing speed of 6 m/s, suggesting that it is at high (competitive) speeds that utilization of SSC becomes more meaningful. The RF muscle showed an interesting behavior. This muscle has basically two functions: knee extension

and hip flexion. Figure 3.7 showed the two-phase activation pattern: (1) end of the braking phase and early push-off, (2) immediately following the push-off phase. From these, the first one is characterized as SSC for the knee joint extension and the second one for the hip flexion. This double SSC behavior was confirmed by the US recordings (Figure 3.10). This explains why the RF showed greater fatigue-induced EMG reduction in a 90 km Wasa cross-country skiing race as compared to other knee extensors, the medial and lateral vastii muscles (Viitasalo et al., 1982).

Mechanical and Metabolic Power Implications of Exploiting SSC

As summarized in Table 3.1, there should be a metabolic advantage to a skier who uses SSC because the oxygen cost of the eccentric part of the cycle, the “negative work” part, is lower than that of the concentric muscle action part of the cycle, the positive work part. Negative work in human motion is more efficient than an equal amount of positive work although the efficiency of negative work is not a constant (Margaria, 1968; Aura & Komi, 1986).

One way of testing this hypothesis would be to directly measure rate of oxygen consumption (metabolic rate, metabolic power output) and oxygen debt on skiers who differ in utilization of SSC as determined from simultaneous kinematic and EMG measures. Metabolic measures can now be telemetered breath-by-breath with minimal interference to the athlete and have been used on skiers skiing on different slopes (Doyon et al., 2001; Welde et al., 2003). Of course, athletes may still be highly resistant to any interference with their performance such as carrying even light-weight analyzers during competition but race simulations are commonly used now.

These light-weight portable analyzers were not available during the 1980s. So the metabolic rates were estimated from biomechanical calculations of mechanical power output on skiers filmed, unobtrusively, during World Cup and Olympic competitions and during controlled training trials on flats and uphill (Norman & Komi, 1987; Norman et al., 1989). Film analysis also allowed us to calculate kinematic data, some of which has been presented earlier in this chapter.

We also calculated the percentage of the mechanical power output that was produced during the skiing stride cycle from “negative work” and from positive work. The rate of oxygen consumption that would be needed to sustain that mechanical power output was calculated, for illustrative purposes, by using an oxygen equivalent of 0.049ml O₂ per Joule (J) of mechanical work, an efficiency of positive work of 0.25 and negative work of 1.2 (Margaria, 1968).

To reiterate, on an overall movement pattern basis, skiers who ski fast but use more negative

work (more use of SSC), should use less oxygen than skiers who ski as fast but use less negative work because negative work is more metabolically efficient than positive work. The SSC skier might have more left at the end of a race for a sprint. Alternatively, for skiers who have the same MVO₂, the skier who uses more negative work (uses more SSC) should be able to ski faster. Figure 3.11 shows a typical set of ME curves over approximately four steps (two strides).

We present calculated mechanical energy costs, estimated metabolic energy costs, and differences between world-class skiers during competition that tend to support differences in use of SSC using a work/energy/power output analysis and possible implications. This complements evidence of use of SSC in cross-country skiing from the kinematic, EMG, force plate, and US analyses presented earlier.

Work is done by or on the body segments to produce the oscillations in the curves. Periods of negative work occur when the component energy or total body energy curves go down. Periods of positive work are rising phases of the curves. The total work over the cycle time is calculated by adding the positive and negative work. The power output is the rate of doing this work. Dividing the total work output by the cycle time (roughly 1.5s in Figure 3.11) gives the power output over the four skiing steps, two strides.

Not all body segments or all muscles that produce the work on the segment are using SSC or doing all positive or all negative work at the same time. Not all of the energy in a segment at any instant in time is produced by muscles acting directly on the segment. Friction on a ski and inertia of a moving segment can also cause energy-level changes on that or other segments. It is possible mechanically, anatomically, and physiologically to transfer some energy from one body segment to another and from potential to kinetic energy and vice versa within body segments such as occurs as a result of gravity acting on a pendulum. Summing energy curves that are out of phase with each other on a graph implicitly assumes transfers of energy from kinetic to potential or vice versa within a

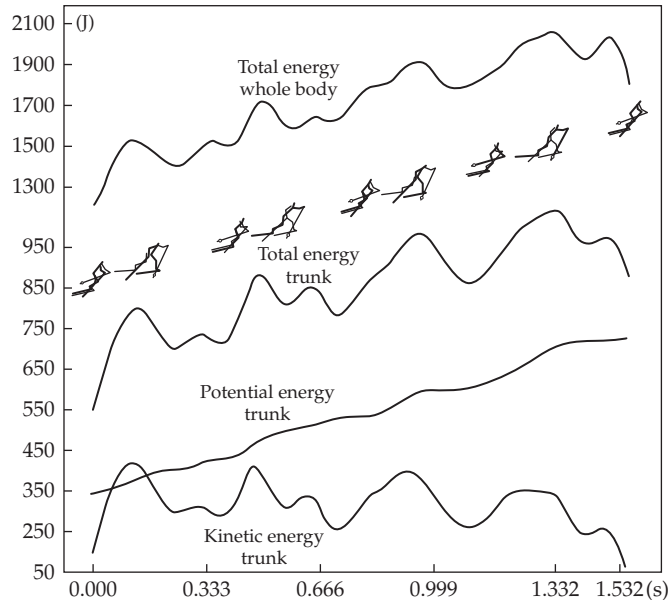


Figure 3.11 Mechanical energy curves of a skier on a 9° uphill during a World Cup competition. The translatory kinetic energy, the potential energy and its sum, the total energy is shown for only one body segment, the trunk, albeit a very important segment because of its high mass. These curves were produced and summed for all of the body segments in the linkage shown in Figure 3.1 to produce the total body energy curve at the top. The gradual rise in the potential energy and total energy curves is caused by the fact that they are skiing uphill. The kinetic energy curve of the trunk oscillates because of velocity oscillations. But even the potential energy curve shows slight oscillations because of vertical rises and falls in body segments although the overall trend of curve rises as the skier progresses up the hill. The basic energy equations are shown for one body segment in Figure 3.1. (From Norman & Komi, 1987.)

body segment, e.g., the trunk, and transfers of energy between segments (e.g., thigh to lower leg) and even nonadjacent segments, e.g., the lower leg to trunk. In fact the main controversies amongst biomechanists regarding how to properly calculate mechanical work and power output revolve largely around how to mathematically and biomechanically treat energy transfers (Cavagna et al., 1964; Winter, 1979; Williams & Cavanagh, 1983).

Table 3.3 summarizes some of these data from selected skiers including J.L. and M.P. whose kinematic data were shown earlier for comparison.

In the 1978 Lahti World Championships, the average velocities over the entire race of J.L., M.P. and K.S., respectively, were 305, 303, and 275 m/min. In Calgary the average race velocities of the three fastest skiers, three skiers placing 49th to 56th, and the 35th place skier were 354, 313, and 325 m/min, respectively.

The overall race speed was much slower in Lahti than in Calgary but the speed on the 9° slope was much faster than on the 11.8° slope in Calgary, a slope difference of less than 3°. Snow conditions, wax, and other factors, in addition to the slope difference, might explain this.

The substantially higher velocities on the slope studied in Lahti compared with 1988 Calgary Olympic Games were produced by much higher power outputs by the skiers selected for illustration. The high mechanical work rates (power) had to be supported by high metabolic energy rates, predicted VO_2 from 150 to 169 ml/kg/min compared with only 61–90 in Calgary. These world-class skiers probably had maximum oxygen uptakes ranging from about 70 to 80 ml/kg/min (Bergh et al., 1978; Holmberg et al., 2006). One, Y.B., was known to be in this range on a ski treadmill.

Table 3.3 Mechanical power output, negative work as a percentage of mechanical power, and predicted oxygen consumption to race uphill on two demanding but different slopes in world-class competitions. The velocities shown are on the uphill analyzed, not the entire race average. Efficiency values for positive work (0.25) and negative work (1.2) were selected from the literature to enable a prediction of VO_2

Skier/place	Velocity (m/min)	Mechanical power (Watts)	Negative work % mechanical power	Predicted VO_2 (ml/kg/min)
9° uphill; Lahti 1978 World Cup 15 km race; classic technique				
J.L./1st	256	1253	30	169
M.P./4th	233	1228	23	168
K-S/58th	214	1030	27	150
11.8° uphill; Calgary 1988 Olympic Games 30 km race; classic technique				
Mean 1st, 2nd, 3rd	165	725	22	90
Mean 49th, 51st, 56th	142	579	21	77
YB/35th	139	381	11	61

If we take the predicted oxygen utilization rates at face value, understanding that many assumptions have been made in the predictions, all of the skiers shown from the Lahti data were going into substantial oxygen debt on an uphill that took about 25 s to complete. The high predicted oxygen utilization rates in the Lahti data are not out of the question. Anaerobic glycolysis has at least twice the power of the aerobic component and can produce an O_2 equivalent at a rate of more than 160 ml/kg/min (di Prampero, 1981). The high-energy phosphagen system is unlikely to be used in skiing except possibly in a short sprint at the end of the race. Moreover, Holmberg et al. (2006) have shown that for world-class skiers during a roller skiing maximum oxygen uptake test using diagonal stride and on a steep slope near the point of exhaustion (10–12°), the degree of arterial oxygen desaturation was only mild, not extreme. Nor did they find an extremely high blood lactate concentration. They speculated that cross-country skiers may better maintain their arterial oxygen saturation when they use a technique that does not require the attainment of $\text{VO}_{2\text{ max}}$. One such technique could be the use of SSC. We have also argued that the mechanics of moving oneself using diagonal stride may limit the oxygen transport that can be achieved because if the leg thrust is too strong, particularly on an uphill and with less of that ideal

wax, the skier will slip and have to save himself using his poles.

Interestingly, in Calgary on a steeper slope than in Lahti, the top three skiers were only marginally above their probable MVO_2 , incurring little debt. Skiers 49–56 were near the MVO_2 expected of a world-class skier and Y.B. was skiing substantially below his known MVO_2 . Whether these differences were strategic decisions by the skiers in Calgary is not known. But skiing at a power output almost 20% below his MVO_2 (Y.B.) suggests a problem or not very good race strategy. It could have been something as direct as bad wax preventing this skier from skiing faster on this rather steep uphill. Skiers passed this site 3 times during the 30 km race. Of the fastest three skiers and nine possible “no slip” analysis samples we obtained eight. Of the three skiers finishing 49th to 56th, we saw four slips on nine passes. Y.B. did not slip on any lap in the film site.

The oxygen costs predicted from the film data appear to be reasonable. Doyon et al. (2001) used a portable gas analysis system to directly measure peak VO_2 on competitive university-level skiers during a maximum high-resistance roller ski up a moderate slope. They peaked at about 60 ml/kg/min. Welde et al. (2003) also used a portable gas analyzer to measure the metabolic energy cost of high-level female junior skiers on snow during a

simulated race of 6 km using diagonal stride and in a different race, freestyle. Regardless of technique, these skiers were unable to ski at intensities close to their MVO_2 (about 63 ml/kg/min) or maintain an average intensity above that corresponding to their onset of blood lactate accumulation (OBLA) even during races that lasted less than 25 min. Over the entire race, the average VO_2 was 55 ml/kg/min (80% of their skiing maximum) and peaked on an uphill at 62 ml/kg/min (94% of their maximum). The mean VO_2 was not higher than that corresponding to their OBLA.

We have argued, from the kinematic data, that some skiers use SSC more effectively than others. This should be seen as higher percentages of negative work. Only 11% of his total power output was produced by negative work by skier Y.B. on the uphill studied. He had to use relatively high-cost positive work, perhaps causing him to slow down to sustain the cost. The other skiers listed in the table were over 20% negative work in the same race. On a lower slope, skiers J.L. and K.S. show relatively high negative work in their technique (27–30%). Skier M.P. produced only 23% of his power from negative work, approximately the same as high finishers skiing much slower and lower finishers on the steeper slope in Calgary. Recall that skier J.L. also showed kinematic evidence of good use of SSC, particularly at the hip but also at the knee and at the elbow during pole thrust while M.P. was much less effective, particularly at the hip, according to this type of evidence.

We can also argue from the kinetic data (force plates, instrumented poles) and US recordings that SSC in cross-country skiing has different loading characteristics on the whole body, joints, and muscles than in running (see Table 3.1). Exhaustive-type running performed on the track (e.g., 10,000 m) or on the asphalt road (marathon run) is usually characterized by delayed onset muscle soreness (DOMS), which lasts several days and has a bimodal recovery pattern, which is naturally individual, but which may peak on day 4 postexercise. The complete recovery can take 7–8 days. The associated muscle damage processes have been explained in detail in Chapter 12. One of the

features responsible for the damage appearance is the high impact load repeated for a long time and until exhaustion. This phenomenon is rarely observed in cross-country skiing despite similar number of repeated SSCs as in running. The peak impact loads are considerably lower in skiing; the peak vertical GRFs seldom exceed 1.5 times body weight. SSCs in diagonal-type cross-country skiing are still efficient mechanically, and they can be sustained longer than those in running, for example. The easiest comparison is perhaps between marathon run and 50 km cross-country skiing. Both are metabolically (energy expenditure) equally demanding, but the difference is clear in the recovery phase from these exercises. It is often possible for the cross-country skiers to repeat the 50 km race in 2 days, but the marathon runner needs 2–3 weeks for full recovery. This comparison may also apply to the free (skating) technique of cross-country skiing. SSCs have been reported to occur in this technique as well, during roller-ski skating (Perrey et al., 2000). The imposed impact (stretch) loads maybe even smoother than in the diagonal technique.

Use of a Ski Tester to Simulate SSCs During Cross-Country Skiing with Relevance to Explore Various Ski Structures and Wax Conditions on Performance

Vähäsöyrinki et al. (2008) emphasized that one of the important features of diagonal skiing is stopping of the ski effectively during the end of the gliding phase, and for preparation of the important kick phase. It is generally known that the EMG activities of the relevant muscles, such as those shown in Figures 3.6 and 3.7, increase with higher skiing speed. These higher activations continue during the kick phase, and the observed patterns suggest that, especially for the ES and VL muscles, high activation during the preloading phase prepares the skier not only to produce greater force during the kick phase, but also to stop the ski. It is very likely that the MG muscle functions importantly to stop the ski. The stopping of the ski is a typical and important feature of diagonal

skiing and its occurrence is timed well with the SSC activities of the relevant muscles. This observation led then to the development of the ski tester, where various preload conditions and stopping of the ski can be simulated to match the real loading conditions of diagonal skiing.

Force production in SSC during cross-country skiing is naturally influenced by several factors, some of which are equipment related, such as grip waxing of the skis. While the ski friction should be

minimized during the gliding phase, it should be maximized during the kicking phase. Skis waxed with poor grip have been shown to produce much less horizontal force compared with good grip skis (Komi, 1987; Vähäsöyrinki, 1995; Piirainen, 2008). Horizontal force of slippery skis can be only 30–50% of good grip skis while the difference in vertical forces is much less—almost nothing. Figure 3.12 shows the clear difference in maximal horizontal leg force while vertical leg forces and pole forces

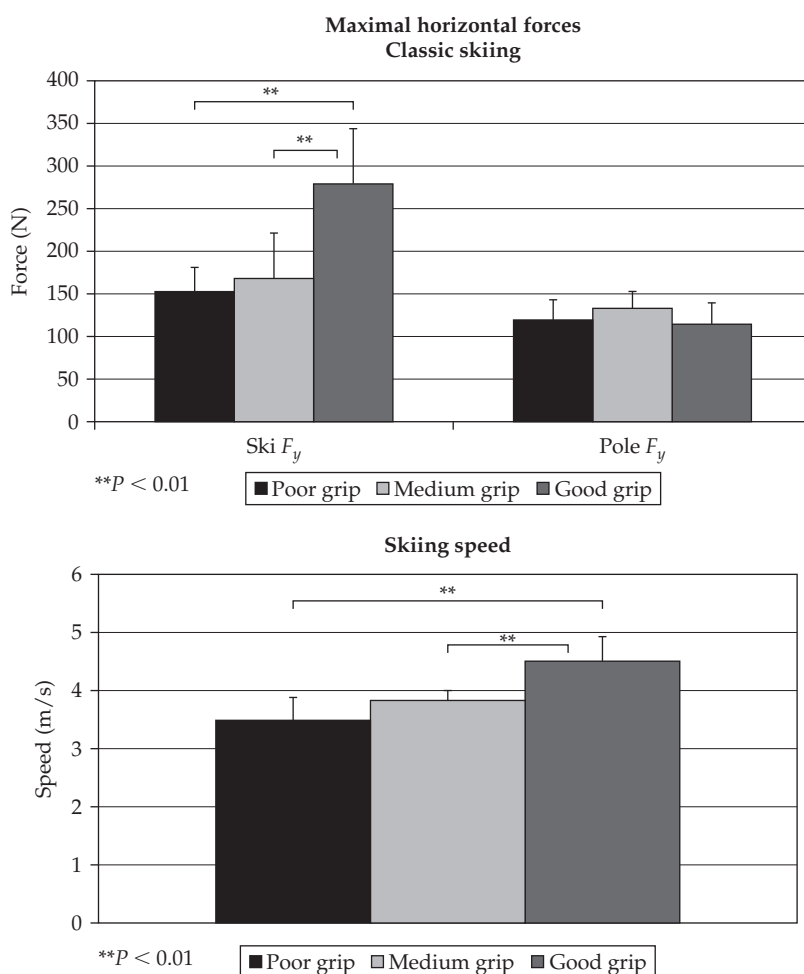


Figure 3.12 Maximal horizontal leg and pole forces in and maximal skiing speed in 11° uphill classical skiing. (From Piirainen, 2008.)

were almost similar with different waxing conditions in steep uphill (11°) skiing at maximal velocity.

In diagonal skiing, the ski should be stopped effectively from full gliding speed so that the kick can be made from a completely stopped ski. As discussed earlier, this process maybe more effective with the use of SSC seen as reduction in force in the preloading phase (Komi & Norman, 1987). In order to be able to accurately estimate the combined effect of SSC and waxing properties on the force production, the measurements should be made in standard repeatable conditions. This would mean that the skier should be able to perform several consecutive attempts with similar preloading and kicking phase force production patterns. To avoid these problems, a novel ski tester was constructed to simulate the force production of natural skiing

and allow accurate measurements in laboratory conditions (Linnaamo et al., 2008). The tester is 13.7 m long powered by a 7.5 kW motor attached to a linear unit which is equipped with a pneumatic cylinder and 6 pieces of 1 m long force plates. A ski is attached to the cylinder and pressed against the force plates. During measurements, the following parameters can be controlled: weight of the ski, stiffness characteristics of the ski, velocity of skiing, and the style of skiing (the form of the force curve).

Under normal snow conditions, even an experienced skier may show considerable trial to trial variations in force production even at constant skiing velocity (Figure 3.13). The tester, on the other hand, has proven to be very repeatable as can be seen in Figure 3.14 with the consecutive force curves

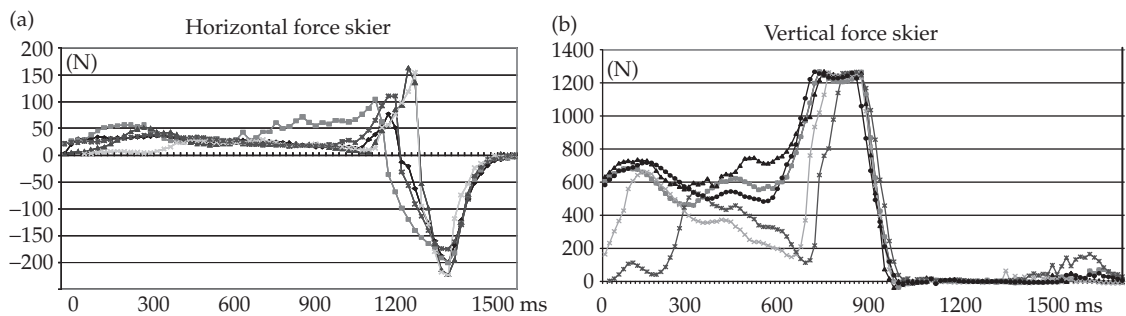


Figure 3.13 Horizontal (a) and vertical (b) force in natural skiing (4 m/s, 2.5° uphill) of the skier in five consecutive trials. (From Linnaamo et al., 2008.)

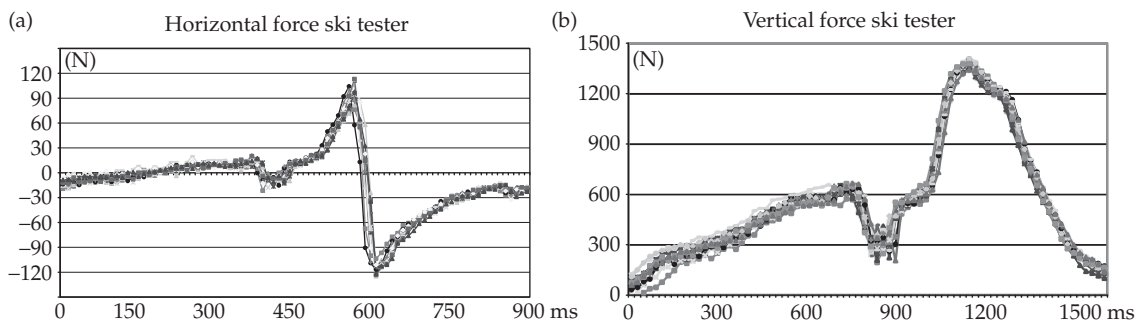


Figure 3.14 Horizontal (a) and vertical (b) forces of the tester in 10 consecutive trials. (From Linnaamo et al., 2008.)

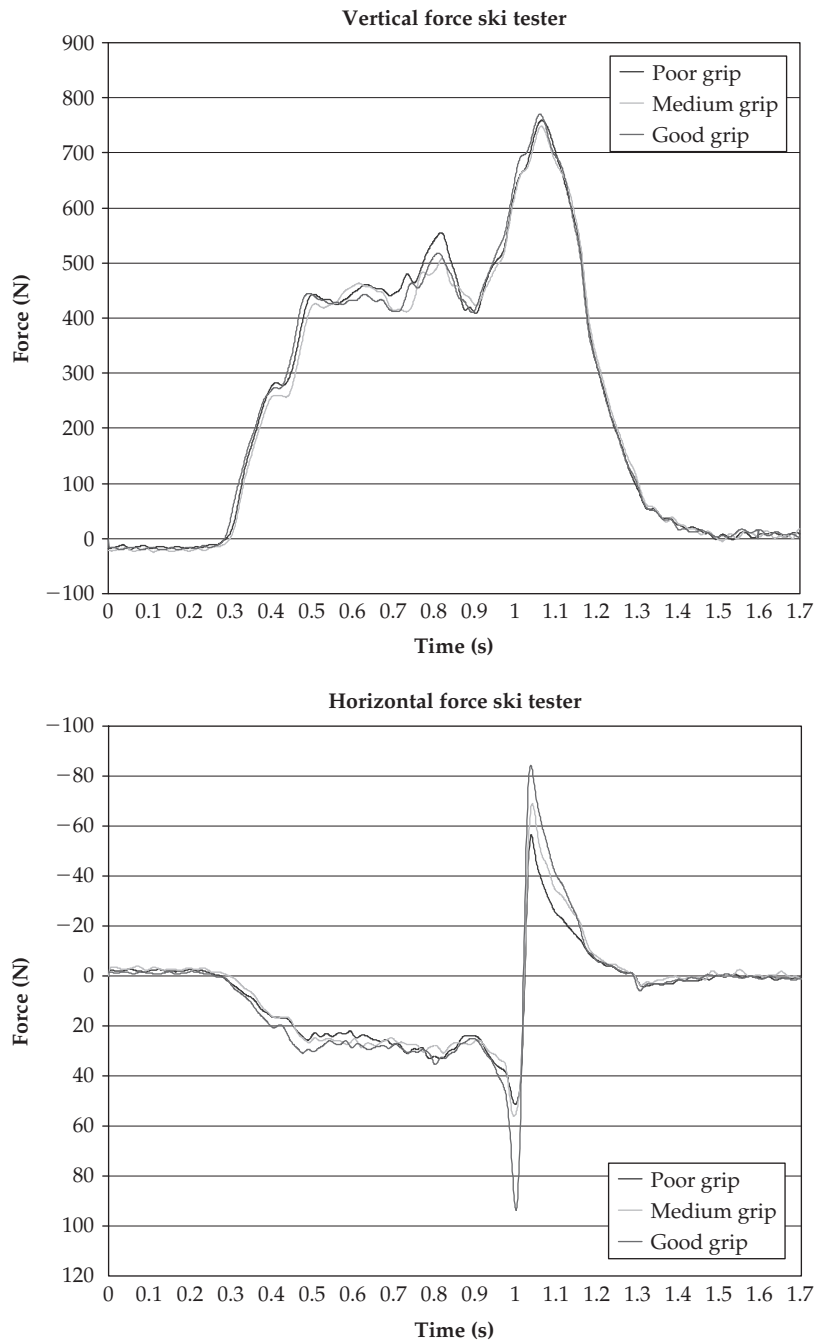


Figure 3.15 Simulated vertical and horizontal forces of one skier (60% body weight) in three waxing conditions in the ski tester. (From Piirainen, 2008.)

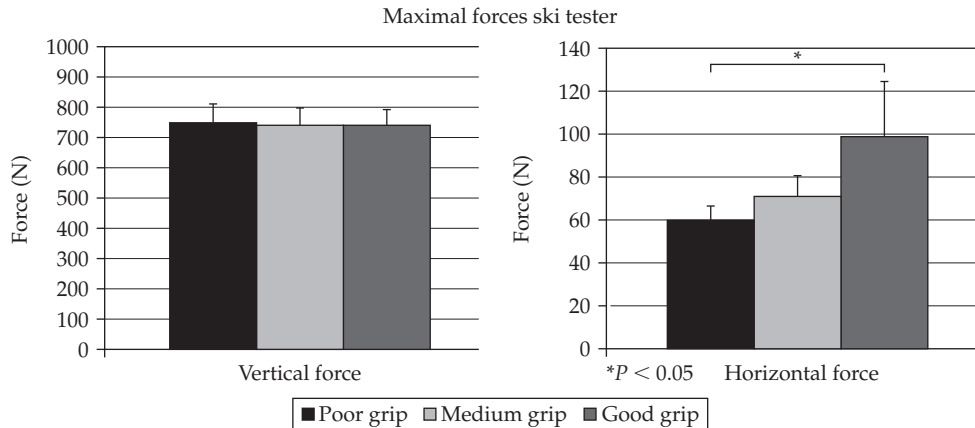


Figure 3.16 Maximal vertical and horizontal forces in the ski tester (60% body weight) in three waxing conditions in the ski tester. (From Piirainen, 2008.)

produced by the ski tester with the same settings being almost identical (Linnamo et al., 2008).

In a recent experiment, 7 male skiers first skied 11° uphill with maximal velocity with 3 different waxing conditions. Thereafter, the skis were run through the tester using 60% of the skier's body weight. The data from the ski tester confirmed that grip waxing mostly affects the horizontal force production. Figure 3.15 shows an example from one skier and Figure 3.16 the average curves of all seven athletes. The data shows convincingly that the vertical force remained similar, while with better grip the horizontal forces increased.

The data are very promising. Once the characteristics of SSC performance are clearly identified from the force data in natural skiing, a wide variety of force curves can be simulated in the machine. This will allow accurate testing of the properties of the skis and different waxing conditions in laboratory conditions.

Summary and Discussion

Our own research in the 1980s and data from others since then have shown use of SSC by some, but not by all skiers, skiing on snow and roller skis, using classical diagonal and skating techniques. Several different types of analyses support

the feasibility and presence of use of SSC in cross-country skiing: body joint angular displacements and velocities, EMG, negative work from mechanical energy analyses, predictions of oxygen costs on flats and steep uphill, force plates, instrumented skis and poles, and US. The evidence also convinces us that use of SSC in skiing is advantageous physiologically, biomechanically, neurophysiologically, and anatomically. Since the first paper that we wrote on this topic, Komi and Norman's (1987) more sophisticated analysis methods have been developed and used in the difficult data recording environment of cross-country skiing. They have been used to better understand the current limits of human capability for the basic research value of simply "understanding." They have also been used to help athletes and coaches who work at the extremes of human performance to push the boundaries higher. Understanding the phenomenon of "stretch-shortening cycles," SSCs, is part of this.

Quantitative video recording and analysis systems eliminate the need for tedious film analysis. Portable, small, and very light breath-by-breath oxygen analysis systems have been invented and used to minimize interference with performance of skiers. A "ski tunnel" has been built in Finland. It houses force plates and other sophisticated

data recording devices and permits training and advanced biomechanical, physiological, and neurophysiological study of skiers of all ages and skill levels year around. US methods have recently been used at the microscopic level of the muscle fascicle to better understand muscle utilization of skiers while they ski. A ski tester has been produced that simulates loading of the ski by actual skiers, including loading changes that can reflect utilization of SSC. This system allows better understanding of the effects of wax, ski design, and other equipment features under simulated but realistic loading conditions based on the complicated F-T curves of skilled skiers.

Skiing techniques have become more sophisticated as well. For example, ski skating was in its infancy in competition in the 1980s but is commonly used in freestyle events now. Skis, boots and poles have been designed and methods have been invented to test and evaluate this equipment specifically to maximize ski skating performance.

In the 1980s, Komi and Norman suggested that the most important benefits of utilizing SSCs as much as possible during high-performance cross-country skiing are higher muscle force output and lower metabolic cost. The efficiency of the negative work during stretch should reduce the oxygen cost for a given skier velocity and thereby produce a metabolic or force generation advantage over a skier who did not use this technique. This contention still needs better proof using modern data recording methods such as those alluded to above. However, evidence over the past 25 years on lower level and highly skilled skiers using diagonal, ski skating, and various poling techniques suggests strong support for the feasibility and value of exploiting SSC.

Knowing a skier's maximum oxygen consumption and lactate tolerance from laboratory tests and their measured metabolic rate and oxygen debt from breath-by-breath analysis during various difficult uphill has enormous potential for race strategy and training. This is particularly true if a coach and athlete want to work on techniques that would help them better exploit SSC by using even qualitative video analysis to teach and monitor body joint positions and changes in body joint angles that produce SSC.

However, some major practical questions raised in the introduction remain relatively unstudied. The following are some of these questions:

1. Is use of an SSC pattern consciously learned or just an accidental technique adopted by some but not all skiers as they develop from young to world-class competitors? It seems that using SSC should be a natural consequence of high-speed self-propulsion just as it is for small children who seem to use it without coaching when learning to throw a ball. Is it possible that use of this somewhat natural SSC pattern is actually eliminated in the early years of skier development by coaches who teach their skiers to ski "smoothly"? SSC is not used by all world-class skiers!
2. Is it now time for coaches to teach skiers how to ski using SSC patterns? We believe that the existing research evidence shows that it should be.
3. If SSC is to be coached, is it helpful to the skier and coach to understand the basic concepts of ECC and the kinematics of how it can be produced by SSCs at the hip, knee, ankle during leg thrust and shoulder, elbow, and maybe wrist during pole thrust? Or is this insight unhelpful information overload to both the coach and skier? This is the classical debate on whether it is important that a coach and/or athlete understand "why?" to do something or just "what?" to do.
4. Can ski and pole F-T curves from force plates and/or instrumented skis and poles be used as biofeedback to teach ECC and exploitation of SSC?
5. Can ski ergometers allow an SSC pattern to be used? If so, can ergometers be used as SSC technique training devices?

We suggest that making some skiers aware of these mechanisms and teaching them to consciously learn to use the body joint movements that create SSC should be tried by some knowledgeable coaches, at least on an experimental basis to assess the efficacy for wider scale coaching implementation. The issues and questions above certainly need research-based answers.

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Chapter 4

Neuromechanics of the Cycling Task

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Introduction

The cycling task is used in a variety of applications including high level competition, exercise conditioning, rehabilitation, and research. While the focus is on performance, each application presents a set of conditions unique to rider position, bike setup, and cadence and load demands. For example, the position of the rider on the bicycle, load, cadence and the set up of the bicycle during competition will be specific to each rider and may even change during any given competition, e.g., long road races. Likewise, during a period of rehabilitation, the position of the rider, the load, and the cadence selected will be specific to the disability, e.g., knee injury versus stroke rehabilitation and the bike will be adjusted to accommodate the rehabilitation plan, i.e., it can change as the training or recovery plan progresses. The common element to all situations is the requirement that the interface between rider and bicycle, including the selection of the appropriate load and cadence, maximizes their experience and enables them to achieve the greatest result. Giving appropriate consideration to where muscles might function at their best given their individual mechanical properties, e.g., length-tension and F-V properties, fiber type, and so on, the quality and training state of the muscle, and how the nervous system might best use these

properties in control of the cycling task, modifying bicycle components to capitalize on the individual's unique motor capabilities will enhance the rider/bicycle interface and hence maximize performance.

There are a variety of bicycle designs used to achieve specific goals during competition, exercise, and rehabilitation. Stationary bicycles are most commonly used in rehabilitation for reasons related, in part, to safety but the bicycle can be upright or recumbent and have a variety of seat, handlebar, and/or pedal designs. Seat tube angle (STA) and mechanisms designed to regulate load can vary substantially as well. Competitive bicycle design may also vary substantially, e.g., road bicycles, mountain bikes, and so on, depending on the demands of the particular competition. Wind resistance is another factor that influences rider position in competitive road and track cycling but is not considered in the clinical or experimental environments. In all of these situations, the bicycle/rider interface will affect muscle function and influence how each muscle and/or group of muscles performs.

The purpose of this chapter is to describe how the cycling task has been used to study muscle mechanics and neural control. Being able to "personalize" the interface between the rider and the bicycle allows the trainer, coach, scientist, or clinician the ability to provide, for example, the range of motion necessary to enhance proprioceptive input in patients with certain neuromuscular disorders, to ensure muscle is able to use its mechanical properties to their advantage, to utilize the rhythmic

nature of the task to address problems faced by individuals with neurodegenerative disease, or to modify load to challenge muscle strength and performance. Emphasis in this chapter will be placed on the muscles in the lower extremity, as they assume the primary role in the production and transmission of power to the bicycle (note: the muscular system is considered a mechanical system as is the bicycle) and, as a consequence, endure the larger loads. A review of cycling mechanics will be followed by a discussion of general muscle mechanics and physiology related to cycling; muscles are major force producers as well as sensors that provide both length and force feedback, for example, to the nervous system during movement. Finally, we will discuss the use of the cycling task in the study of the neural control of movement with an exemplar application to a physically challenged population.

Cycling Mechanics

Kinematics

Efforts have been made to comprehensively describe lower extremity kinematics during cycling using 3D analyses. For example, internal and external rotation of the tibia about its long axis, knee translation in the frontal plane, and movement of the lower extremity outside the sagittal plane have all been reported (Hannaford et al., 1986; McCoy & Gregor, 1989; Ruby et al., 1992). Boutin et al. (1989), for example, showed data indicating that the knee joint center can move as much as 6 cm in the mediolateral direction during one pedaling cycle. McCoy and Gregor (1989) showed changes in center of pressure on the pedal surface during the pedaling cycle and data showing the knee to be medial of the pedal center of pressure throughout the power phase at three seat height conditions. Data illustrated in Figure 4.1 represent average patterns for 150 pedal revolutions from 10 subjects riding at 200 W at the three seat height conditions. The significance of these data lies in the fact that the lower extremity displays considerable movement in the frontal plane, that variation in bike setup has a marked effect on the amount of actual movement, and that these movement patterns will affect muscle function.

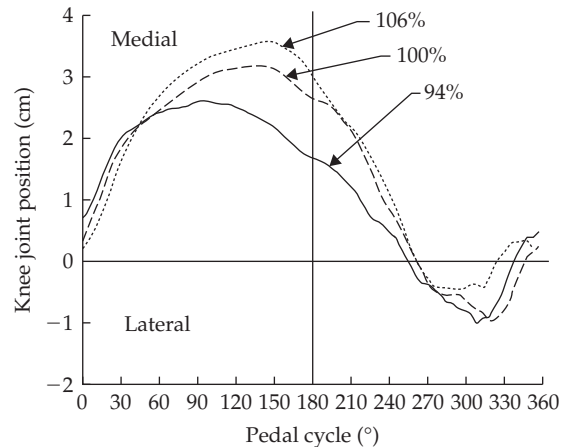


Figure 4.1 Position of the knee, at the mid-knee joint, with respect to the pedal center of pressure during the pedaling cycle in the frontal plane. Each curve is an average of 150 pedal revolutions (10 subjects, 5 trials) at 200 W and 80 rpm. Separate patterns are an average at three seat conditions: 94, 100, and 106% of leg length.

Linear and angular displacements, velocities, and accelerations of the thigh, shank, and foot are most affected by cadence and bicycle geometry, i.e., bike fit (e.g., seat height, STA, crank length, and so on). *Seat height* is defined as the distance from the top of the pedal to the top of the saddle when the crank is down and in line with the seat tube. The complex interactions between bicycle setup, rider position, and rider kinematics and attempts at “optimizing” the bicycle–rider interface have been the subject of many studies that systematically varied rider kinematics by changing rider position, pedaling cadence, or load (Gregor et al., 1991a, Sanderson and Amoroso, 2009). Peak flexion and extension of the joints in the lower extremity as well as movement in the frontal plane will also vary depending on the seat height and fore–aft seat adjustments (McCoy & Gregor, 1989).

Rugg and Gregor (1987) reported the effect of seat height changes on hip and knee range of motion as seat height varied from 100% to 115% of pubic symphysis height (i.e., height measured from the pubic symphysis, or crotch, to the floor). Data summarizing these results are presented in Figure 4.2. While this range of seat heights is rather extreme, an interesting feature is the similar shape of each joint’s kinematic pattern across the four seat height conditions. This

is true despite the increased peak knee extension and much smaller increases in peak hip extension as seat height increased. In a more recent report, Price and Donne (1997) showed similar trends suggesting kinematic patterns remain rather predictable in response to changes in seat height despite the absolute changes in range of motion.

Lower limb kinematics seem to be influenced to a lesser degree by STA (Too, 1991; Brown et al., 1996; Heil et al., 1997; Reiser et al., 2001) than by changes in seat height. Heil et al. (1997), for example, examined changes in lower extremity kinematics as a function of changes in STA and reported findings that show knee kinematics to be generally unaffected by STA while hip and ankle angles increased with increased STA. Reiser et al. (2001) reported similar results suggesting that the relatively small changes in knee joint kinematics across a broad range of body orientations could be due to regulation by the CNS to maintain single-joint knee extensors within an operational range of their F-L curve. This suggestion has merit given the fact the

knee joint contributes the most to power production during cycling (Broker & Gregor, 1994).

Other issues affecting muscle function as related to the mechanical properties of muscle include cadence, fatigue, and the specific strategies used by the individual rider to compensate for changes in muscle properties and the effect they have on the muscle's response to mechanical load. As described by Sanderson et al. (2006), as cadence increases, the ankle joint becomes more extended, i.e., plantarflexed, while the knee joint becomes more flexed (Figure 4.3). These kinematic changes may allow the GAST and SOL muscles, for example, to minimize increases in contraction velocity and possibly reflect a strategy designed to maintain an operating range better able to capitalize on the F-V and F-L curve profiles of the muscles in the triceps surae. Certainly, changes in joint kinematics relate to muscle length changes and changes in muscle velocities.

As mentioned earlier, fatigue also affects lower limb kinematics (Sanderson & Black, 2003; Dingwell et al., 2008) and has been studied in a variety of conditions using the cycling task. Exact changes vary across subjects but generally the trunk appears to be more horizontal (Dingwell et al., 2008), the thigh more vertical (Sanderson & Black, 2003; Dingwell et al., 2008), and the ankle more dorsiflexed (Sanderson & Black, 2003) as fatigue progresses. Little change in knee kinematics has been reported which is an interesting point given the observations mentioned earlier regarding the small changes in knee joint kinematics. Sanderson and Black (2003) showed degradation in the recovery phase "effective force" (see section on Reaction Force) with an increase in power phase "effective force" and suggested these changes were related to joint flexors fatiguing sooner than joint extensors. Dingwell et al. (2008) presented evidence of localized fatigue in joint flexors more often than joint extensors and that the changes in cycling kinematics could be related to the fatigue within those muscle groups.

Finally, it should also be considered that kinematic changes at one joint might be a result of fatigue in muscles crossing another joint. The lower extremity is a highly integrated system and compensations can take place in joint kinematics at one joint as a result of fatigue, for example, in biarticular muscles crossing an adjacent joint or in single-joint

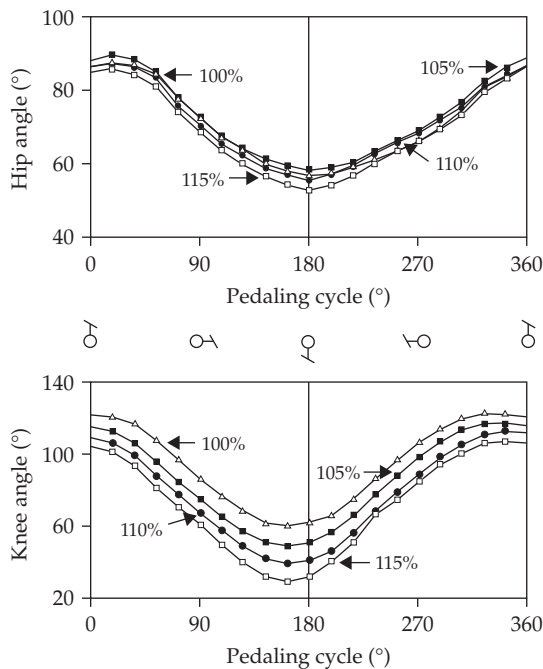


Figure 4.2 Angular displacement of the hip and knee for one pedaling cycle at four different seat heights: 100, 105, 110, and 115% of leg length.

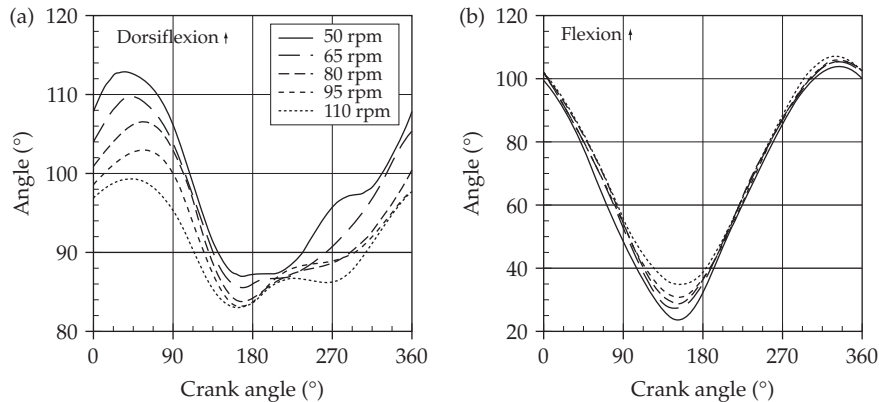


Figure 4.3 Mean joint angles for ankle (a) and knee joints (b) normalized for the pedaling cycle. Neutral position for the ankle joint was 90° where values greater than 90° are dorsiflexion. For the knee joint, full extension was 0° . (From Sanderson, DJ, Martin, PE, Honeyman, G. and Keefer J. Gastrocnemius and soleus muscle length, velocity and EMG responses to changes in pedalling cadence. *J. Electromyography and Kinesiology* 2006; 16: 642–649. Reprinted with permission.)

muscles crossing another joint in the lower extremity system. In the consideration of the multijoint system, no joint can be taken in isolation (Kuo, 2001). Care must be given any interpretation of data on fatigue and its effect on individual muscle/joint systems as well as on the integrated function of the total lower extremity (Kuo, 1994).

Pedal Reaction Forces

Using a kinetic analysis of lower extremity function for the purpose of understanding skeletal muscle response to mechanical load requires knowledge of the interactive forces between the rider and the bicycle. Pedal reaction forces have been quantified using a variety of specially instrumented pedals (Gregor et al., 1991a). Three independent force components of the applied load to the pedal are illustrated in Figure 4.4a. Further, these components are used to calculate a center of pressure (Figure 4.4c, A_y and A_x) and a rotational moment about an axis orthogonal to the surface of the pedal (Figure 4.4b, M_z) (Broker & Gregor, 1990). Pedal reaction forces were first recorded in the late 1880s and it seems that regardless of the complexity of the instrumentation used to make the measurements all reaction force profiles in a standard seated position look quite similar (Gregor et al, 1991a).

While the magnitude of the pedal reaction force is important, vector orientation will markedly

influence how the leg musculature produces/ responds to various pedal loading demands (see Figure 4.1 on a frontal plane analysis). Regarding the sagittal plane, an exemplar pattern of the resultant pedal reaction force calculated at four intervals during the pedaling cycle is presented in Figure 4.5. While this type of presentation gives a general impression of the potential effects of these loads on the segments of the lower extremity, joint moments and reaction force components must be calculated using either inverse or forward dynamics simulations (Zajac, 2002). It is clear however, that changes in vector orientation during the pedaling cycle and hence the response of skeletal muscle to the different mechanical loads imposed during the pedaling cycle are affected by changes in rider orientation and bike geometry. This is true as the pedal and crank change orientation during one crank rotation as well as across a variety of bike setup conditions.

Another variable that has proven useful in understanding the analysis of muscle function, pedaling technique, cycling efficiency, and neural control of the lower extremity has been the quantification of the vector orientation of the load applied to the pedal and hence to the crank. This calculated outcome measure has been described primarily in the sagittal plane of the pedal using the force component orthogonal to the pedal surface and the anteroposterior shear force component in the pedal.

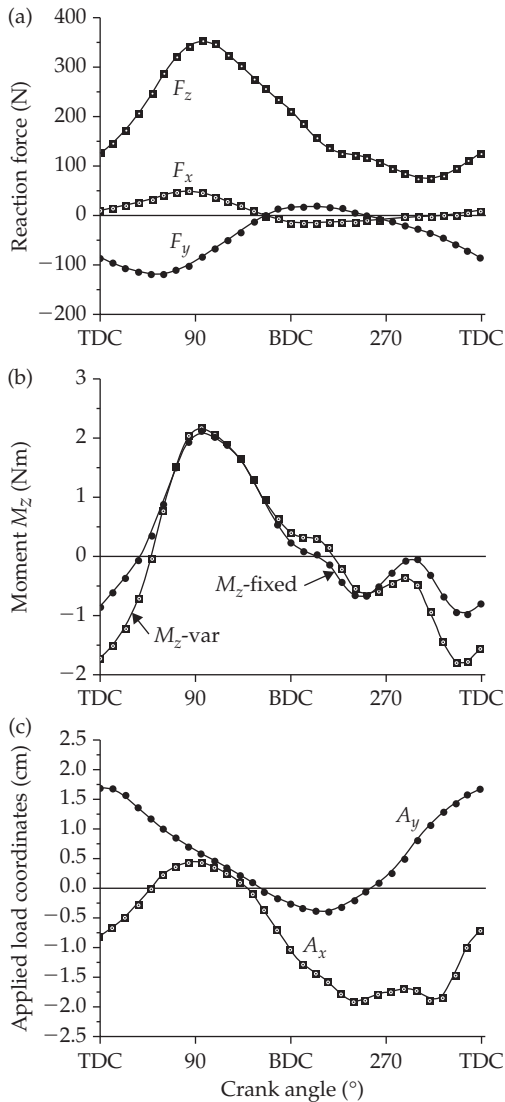


Figure 4.4 Mean patterns for five pedal revolutions for the pedal reaction force components, F_z orthogonal to the surface, F_y anteroposterior (AP) shear, F_x mediolateral (ML) shear (a), applied moment (b), and center of pressure patterns (c) during one pedaling cycle (top dead center [TDC] at 0° to TDC at 360°). (a) F_z is the component orthogonal to the pedal surface, F_y is AP shear, and F_x is the medial/lateral shear. (b) The applied moment (M_z) about an axis orthogonal to the pedal surface when this axis is through the center of pressure (M_{z-var}) or through the center of the pedal ($M_{z-fixed}$). (c) Coordinates of the applied load in the x (A_x) and y (A_y) directions. (Reprinted, with permission, from J.P. Broker and R.J. Gregor, 1990, 'A dual piezoelectric element force pedal for kinetic analysis of cycling,' *Journal of Applied Biomechanics* 6(4):393–403.)

(Note: Force components orthogonal to the pedal surface and the mediolateral shear force component were used to generate the data in Figure 4.1). Resolving these force components to the crank, the “effective force” acting on the crank, i.e., the component calculated orthogonal to the crank (LaFortune & Cavanagh, 1983), has been reported in the literature for many years. The utility of such a calculation in studying issues specific to the field of cycling mechanics, motor control, and muscle function is exemplified in studies where the direction of the applied load to the pedal is provided as feedback to the rider during a training or rehabilitation intervention. Broker et al. (1988, 1993), for example, examined the effect of visual feedback schedules on controlling force application to the pedal. Further, Hasson et al. (2008) used real-time feedback of the effective force pattern on the crank to study the coordination of one- and two-joint muscles and the reorganization of joint moments during learning of a complex task. Participants were instructed to direct pedal forces in a specified target direction, i.e., perpendicular to the crank or the “effective force” on the crank, during one-legged cycling. Results suggested that improved performance, i.e., learning a complex task, was accompanied by a reorganization of joint moment patterns and one- and two-joint muscle coordination.

A more detailed representation of “effective” and/or “ineffective” pedaling, however, was discussed by Kautz and Hull (1993) in which they separated a “fundamental” component of pedal loading from the measured pedal loads. The “fundamental” component is generated by the inertial and gravitational components acting on the pedal/lower limb because of the connection of the lower limb to the pedal, and represents the nonmuscular component of pedal loading. Standard computation of joint moments, reaction forces, and a complete description of the pedal and crank kinematics are required for the calculation of the inertial and gravitational components. Representation of these components from an elite cyclist pedaling at 400 W at 118 rpm is illustrated in Figure 4.6. A major observation presented in Figure 4.6 is that the muscular component, i.e., the component the cyclist can control, appears to be orthogonal to the crank

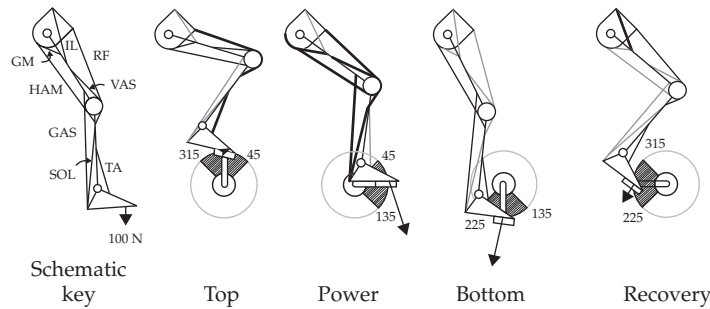


Figure 4.5 Schematic of the lower limb showing representative muscle activity of the lower limb, direction and magnitude of the force at the pedal, and limb positions in the four different quadrants of the pedal stroke. Muscle activity within each quadrant is indicated by the thickness and shade of the lines. Muscles may be very active (thick black), moderately active (thin black), or not active (thin gray). Numbers around the pedal arch denote the start and stop of the respective quadrant in crank degrees relative to TDC. Magnitude of the force vector corresponds to its length defined by the schematic key. Values for limb orientation, muscle activation and force production are derived from experimental data on an intact cyclist operating at 200W at 90 rpm. (GM, gluteus maximus; HAM, hamstrings; GAS, gastrocnemius; SOL, Soleus; TA, tibialis anterior; VAS, vastii; RF, rectus femoris.) (From Childers WL, Kistenberg RS, Gregor RJ: The biomechanics of cycling with a transtibial amputation: Recommendations for prosthetic design and direction for future research. *Pros Orthot Int* 2009; 33: 256–271. Copyright 2009 informa healthcare. Reprinted with permission.)

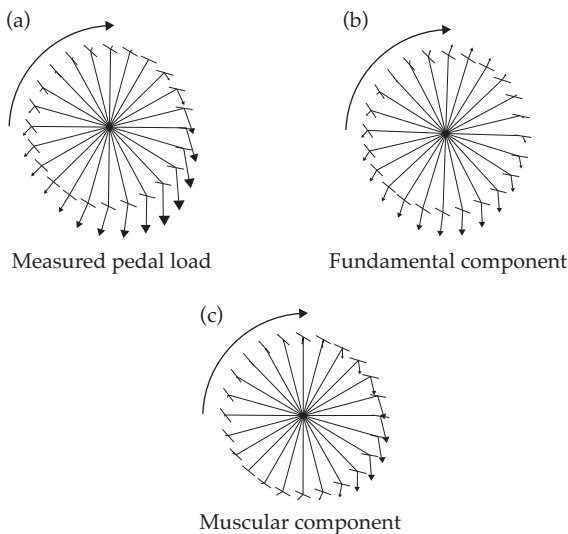


Figure 4.6 Clock diagrams illustrating the measured (a), fundamental (nonmuscular) (b), and the muscular (c) components of the pedal loading for an elite US national team cyclist. The muscular and fundamental components, which sum to produce the measure force pattern, illustrate the separate effect of natural dynamics and active muscular contractions on pedal loading, respectively. Reprinted, with permission, from J.P. Broker and R.J. Gregor, 1996, *Cycling biomechanics*. In *High-tech cycling*, edited by E.R. Burke (Champaign, IL: Human Kinetics).

and positive throughout the most of the pedaling cycle. This decomposition process seems more useful as it allows the investigator to study the interaction of the muscular component with the inertial and gravitational components in evaluation of limb control during cycling. The primary concern in these calculations however, is how the inertial component is evaluated; an aspect of this approach that remains open to debate.

Pulling up on the pedal during the recovery phase has received a great deal of attention as well in the literature with respect to mechanical effectiveness and metabolic efficiency (Korff et al., 2007) and in most cases calculated joint moment patterns are the primary interest because the patterns of EMG activity and the coordination of joint moments reveal a great deal more insight into muscle function, metabolic cost, and neural control.

Initial insights into these issues begins with the direction of the applied load to the pedal but eventually is brought to the level of muscular coordination. More recently, Mornieux et al. (2008) studied the effects of pedal type on the pull-up action during cycling and while pedal type did not influence cycling technique during submaximal exercise, an active pulling up action did improve pedaling effectiveness.

Regarding more clinical applications in limb control, Perell et al. (2000) used visual feedback of the effective force presented to stroke patients, i.e., unilateral cerebrovascular accidents (CVAs), for the purpose of enhancing gait symmetry. More recently Rogers et al. (2004) studied the effect of stroke on the control of foot force while subjects performed pushing efforts against “translationally” fixed and moving pedals. They reported that the direction of the vector component due to muscle activity (F_m) remained constant with increased effort in both pedal conditions but that the orientation of that vector component was different, bilaterally. These data suggest that despite the shifts observed in the orientation of F_m in the paretic limb in relation to the nonparetic limb, there might be a trigger for relative muscle activation based on posture and that lower limb force might be organized by magnitude and direction and that these are differentially affected by stroke. The aspects of the decomposition of the pedal reaction force into muscle, inertial, and gravitational components and the relevance of these measures to understanding the control of limb function will be further discussed in the section on Neural Control.

Muscle Moments

Muscle moment patterns represent the net kinetic demands experienced at each joint and are calculated using a linked segment model and equations of motion with kinematic, pedal reaction force, center of pressure, and anthropometry data used as input. Gregor (1976) (Figure 4.7) first reported flexor moments at the knee during the propulsion phase, e.g., a change from an extensor moment to a flexor moment at about 90° in the pedaling cycle, and that the muscle moments at the hip, knee, and

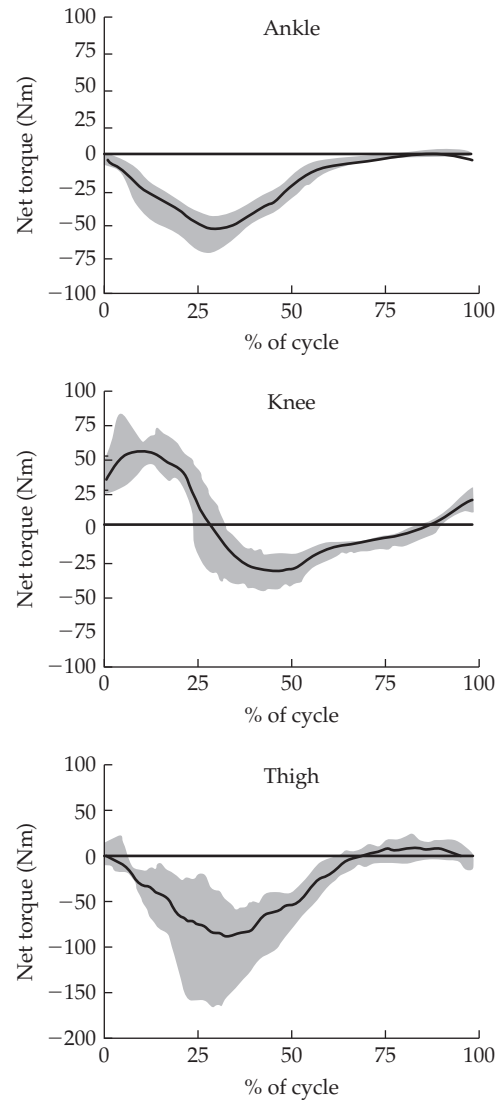


Figure 4.7 Muscle moment patterns for the ankle, knee, and thigh (hip) during the pedaling cycle. Patterns represent the average of 25 pedaling cycles (five subjects, five trials) at approximately 260 W and 84 rpm. (From Gregor et al., 1985. Reprinted with permission.)

ankle have *fairly repeatable patterns*. A large hip extensor moment has been reported during the propulsive phase while the knee extensor moment has been reported as the primary source of energy used to rotate the crank. During recovery, flexor moments appear at the hip and knee (see earlier

text related to “pulling up” on the pedal during recovery) and may actually serve to unload the pedal (e.g., unload the pedal by attempting to pull up on the crank during the recovery phase), potentially minimizing resistance to the contralateral propulsive limb (see section on Neural Control, Ting et al., 1998, 2000). There appears to be a plantar flexor moment at the ankle throughout almost the entire pedaling cycle. Magnitudes of the muscle moments appear to increase in response to increased load (Ericson et al., 1986) and are inversely proportional to changes in cadence (Redfield & Hull, 1986). However, despite variations in loading conditions, subject population, and bike setup, the general moment patterns remain essentially the same (Gregor et al., 1991a).

Zajac (2002) and his colleagues have used forward dynamics simulations in analyzing the cycling task and for the most part focused their efforts on muscle function, neural control, and rehabilitation (see section on Neural Control). While these investigators have made major advances in describing 3D mechanics during cycling, the vast majority of data reported by other groups in the literature remains focused on patterns calculated in the sagittal plane with some reports presenting data for specific movements in the frontal plane (McCoy & Gregor, 1989). Varus/valgus moments have been studied with respect to knee injury with general conclusions focused on knee rehabilitation (McCoy & Gregor, 1989; Ruby et al., 1992; Gregersen et al., 2006).

Of major interest to biomechanists, neuroscientists, and clinical scientists is the fact the hip and knee appear to perform very different actions during the propulsive phase of cycling, i.e., the hip consistently produces an extensor moment, while the knee produces an extensor and then flexor moment during the power phase. These observations appear consistently in the literature (Gregor et al., 1985; Andrews, 1987; Jorge & Hull, 1984; Redfield & Hull, 1986). Gregor et al. (1985), for example, discussed the muscle moment pattern at the knee in light of the paradoxical behavior of biarticular muscles, i.e., a two-joint muscle may act as an extensor of the joint of which it is a flexor (Lombard, 1903). Andrews (1987) also addressed

this “paradoxical” behavior in light of the method of classification employed in the study while van Ingen Schenau (1989) essentially concluded that the uniarticular muscles were “power producers” and the biarticular muscles were “power distributors” in the coordinated action of the lower extremity segments during the cycling task. More recently, however, Kuo (2001) argued that these conclusions could be misleading because the manner and degree to which a muscle contributes to a task such as cycling ultimately depends on the redundant nature of the musculoskeletal system and the synergism of the muscles involved in responding to the specific task. The significance of these statements rests in the investigation of how the CNS programs a system of muscles, aware of the architectural properties of individual muscle and the more global interdependence of these muscles in the entire lower extremity. So while individual muscle properties are important, how each acts/reacts in the system is where the focus should be.

Mechanical Work and Power

The purpose of this section is limited to the general power profiles of each joint in the lower extremity that have been presented in the literature (Broker & Gregor, 1994) (Figure 4.8). The rationale for this approach to these calculations rests in the fact that mechanical work and power have been calculated and reported in the literature using essentially two different methods, e.g., sources and fractions, revealing very different results (Zatsiorsky & Gregor, 2000). The reader is cautioned when evaluating literature on this topic to understand the methods employed that led to the conclusions drawn.

In an effort to address load sharing among the hip, knee, and ankle joints during cycling as previously discussed by Kuo (2001), Broker and Gregor (1994) used a deterministic model to study the management of mechanical energy in the lower extremities during the cycling task. Newtonian equations were used in a sagittal plane model to calculate joint muscle powers (Figure 4.8) and the hip joint force power across a range of loads and speeds. It has been suggested that more than 80% of the energy generated at the joints in the lower

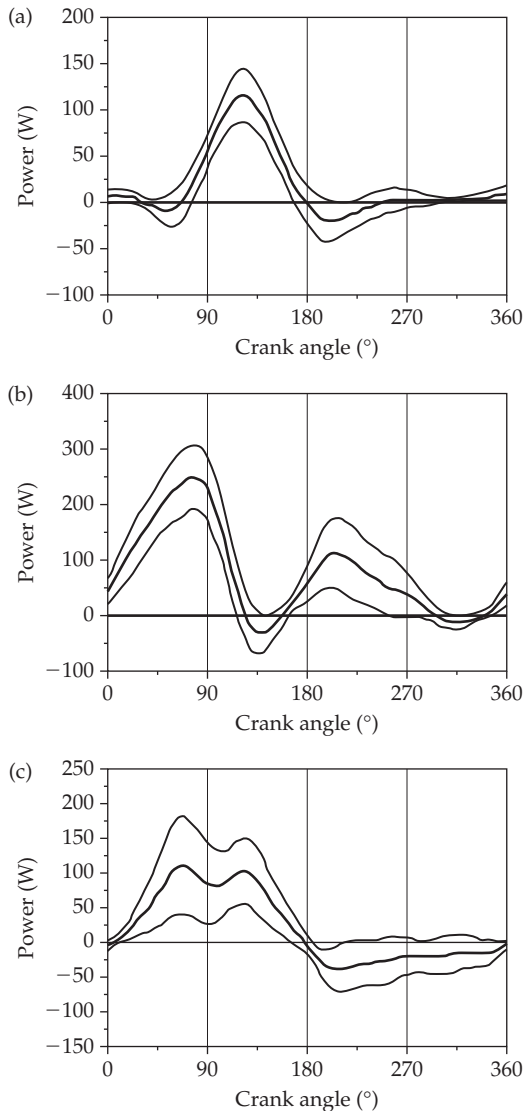


Figure 4.8 Mean (+1 SD) patterns of muscle power (calculated as the product of the muscle moment and angular velocity for each joint) during the pedaling cycle for 12 subjects riding at 250 W and 100 rpm. (a) the ankle, (b) the knee, and (c) the hip. (From Broker & Gregor, 1994. Reprinted with permission.)

extremity can be delivered to the pedals as useful energy to drive the bicycle (van Ingen Schenau, 1989; Ericson, 1988). This is in contrast to the results of similar analyses on running and walking.

Using three energy models to study the issue of energy production and transmission during cycling, Broker and Gregor (1994) studied how single-joint muscles might be energy producers and biarticular muscles might be able to transfer, or distribute, energy from one joint to another. Two models ranged from one in which only hypothetical single-joint muscles acted (no transfer) to one in which multijoint muscles acted and permitted unlimited transfer. A third model, having a sound basis in anatomic function and one consistent with reported EMG patterns (see section on Muscle Function), apportioned joint muscle powers to single- and two-joint muscles and muscle groups in accordance with minimum energy expenditure criteria. Using this model resulted in the appropriate transfer of energy across two-joint muscles and substantially limited the energy dissipated to nontransferable sources. These findings, however, should be considered again in light of some more recent thinking regarding system performance (Kuo, 2001). In a systems analysis of the lower extremity during cycling, the differences between muscles appear to be a matter of degree rather than type. Joint moments are calculated quantities not known to the nervous system and to some degree limiting in their analysis of system output. Energy can be transferred; single-joint muscles do affect more than one joint and muscle agonists and antagonists take on a more functional definition than an anatomical one. It is the system performance that is critical to the efficient energizing of the bike through appropriate end point forces imparted to the crank (Kuo, 2001).

Muscle Mechanics

Muscle Activity Patterns

Knowledge of muscle activation patterns (considering intensity, duration, and timing in a movement sequence) is significant to our understanding of how muscles are controlled by the nervous system to meet the demands of a given task. Muscle is the end organ of the control system, i.e., the nervous system, a force producer activated by the nervous system and a sensor providing information to the

CNS regarding length, velocity, and force. Skeletal muscle appears to be a “mutable actuator” at the interface between the CNS and the environment and affected both by central activation and environmental perturbations. Specific to the cycling task, activation intensity in major muscles used in propulsion should be optimized at the appropriate time during the pedaling cycle and minimized in muscles whose action might be counterproductive to propulsion, the major objective of the task. Muscle activity patterns have been reported for a variety of lower extremity muscles using both surface and fine-wire EMG electrodes (Ryan & Gregor, 1992; Juker et al., 1998).

EMG patterns are commonly described relative to the angle of the crank with the greatest magnitudes observed during propulsion, i.e. 0°, TDC, top dead center to 180°, BDC, bottom dead center. In contrast, EMG magnitudes are generally lower during recovery, with some reports showing significant activity in the flexors (e.g., hamstring muscles) suggesting these muscles might assist net forward propulsion supporting the contralateral propulsion limb. (See earlier discussion on the direction of the pedal reaction force and the section on Neural Control).

A review of many published reports shows the pattern of muscle activation among the major flexors and extensors in the lower extremity to be fairly consistent, i.e., repeatable, across consecutive pedaling cycles. Representative data published from 18 experienced cyclists riding at 90rpm and 250W on their own bicycles are shown in Figure 4.9 (Ryan & Gregor, 1992). Comparing these data to the muscle moment patterns previously described also shows some consistency between EMG timing and the moment patterns at each joint. For example, there is a hip extensor moment in propulsion and activity in the gluteus maximus during the same period. There is a plantar flexor moment during propulsion consistent with the presence of the triceps surae activity. And, the cessation of single-joint knee extensors at about 90° in the cycle together with the more prolonged hamstring activity beyond 90° supports, to a first approximation, the pattern of the muscle moment at the knee during propulsion. Direct links cannot be made, of course, between the EMG and the muscle moments because of the

integrated nature of the moment calculation, the electromechanical delay, and the fact muscles have an affect on more than one joint. Despite these limitations, however, there does seem to be some consistency in both moment and EMG patterns during seated cycling.

In contrast, there are some reports showing EMG patterns to be affected by changes, for example, in body position, i.e., “bike fit,” cadence, load, and cycling ability (Juker et al., 1998). Studying the effects of five different stationary cycling positions, these investigators concluded that position on the bike influenced the timing and magnitude of the muscles under study. In their investigation of single-joint hip flexor muscles, Juker et al. (1998) presented data on the activity patterns of the lumbar psoas, erector spinae (ES), and three different layers of the abdominal wall muscles during five different styles of cycling. These data, while difficult to obtain, are valuable to the understanding of hip and low back function during cycling during both propulsion and recovery. Hip function and stabilization can have a major impact on muscle function in the lower extremity.

Additionally, Savelberg et al. (2003) addressed the issue of upper body position by manipulating trunk angle during ergometer cycling, and directed their attention to recumbent cycling finding a dramatic effect of trunk inclination on muscle activity patterns. Chapman et al. (2008a), in contrast, suggested that upper body position had no substantial effect on muscle activation but they did report greater coactivation and poor EMG modulation in the aerodynamic position in novice versus elite triathletes. These findings suggest that elite cyclists may have better control over muscle activation as a consequence of repeated training. Further to their reports on body position, Chapman et al. (2008a) suggest that changes in cadence affect certain aspects of muscle activation. Their novice riders displayed greater variability, greater coactivation, and higher magnitudes of EMG than the highly skilled cyclists (Chapman et al., 2008a,b).

In contrast to the more complex mechanics of the lower extremity in the low back, pelvis, and thigh regions, musculature in the leg is more accessible and as a result has been studied more

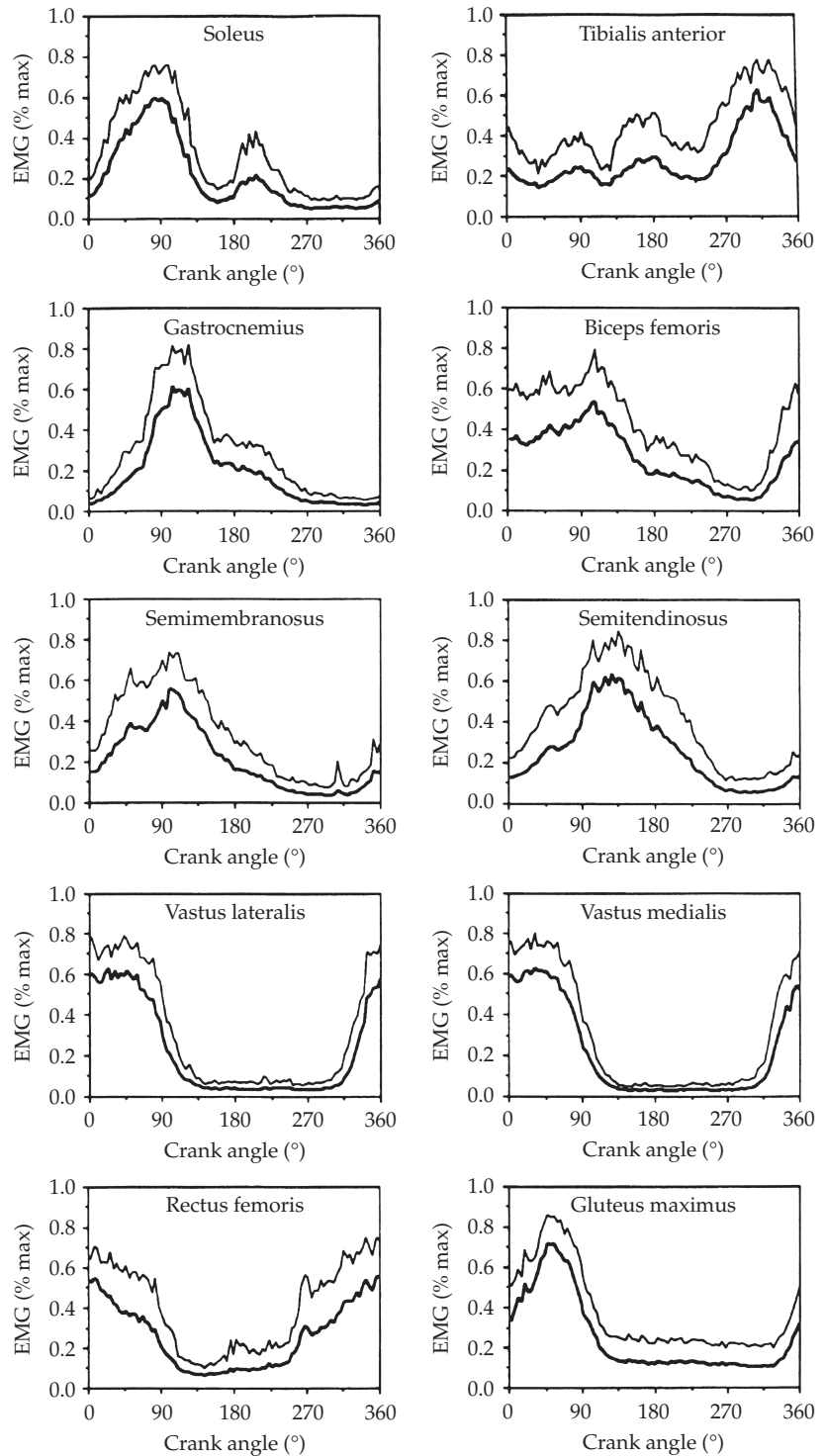


Figure 4.9 Mean patterns of muscle activation during the pedaling cycle for 10 muscles in the lower extremity. The dark lower curve is the average pattern from 15 pedaling cycles across 18 subjects (270 cycles), and the lighter upper curve is one SD above the mean. Magnitudes are normalized to maximal activation recorded within the trial. (From Ryan & Gregor, 1992. Reprinted with permission.)

extensively. Major ankle extensors are active during propulsion and although they are not considered major power producers they do provide an important link between the foot and pedal and they do provide evidence by their behavior regarding how two anatomical synergists work together to achieve a common goal, i.e., delivering power to the crank. Fregly and Zajac (1996) and Raasch et al. (1997) suggested the primary function of the ankle musculature was to transmit forces generated by more proximal muscles to the pedal. Both muscles are active before TDC but onset and peak SOL activity occur before onset and peak GAST activity. Reasons for this phase shift can be related to two factors; activity in both muscles seems to occur near peak stretch of the muscle-tendon-unit (MTU) with peak stretch occurring later in the GAST than in the SOL (Figures 4.10 and 4.11); and, a delayed peak in the activation of the GAST (Figure 4.11) would help contribute to the knee flexor moment consistently observed after 90° in the pedal cycle (Figure 4.7). These results are supported in a report by Duchateau et al. (1986) indicating the delay in onset of GAST activity after SOL onset remains but decreases (Figure 4.12), as cadence increased with near synchronized onset at very high cadences, e.g., 140rpm. There was no statistical difference across loads (10–70 N) in the decrease in this delay as cadence increased.

Duchateau et al. (1986) did not report MTU length changes.

It seems then that the nervous system regulating the onset of these two muscles may not be “hard

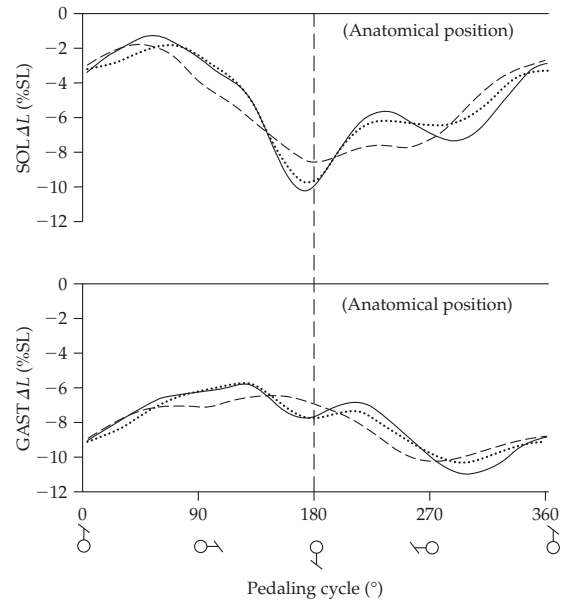


Figure 4.10 Average MTU length changes, calculated from joint kinematics, for the soleus (SOL) and gastrocnemius (GAST) muscles during the pedaling cycle at 90 W (—), 180 W (---), and 270 W (.....). (From Gregor et al., 1991b. Reprinted with permission.)

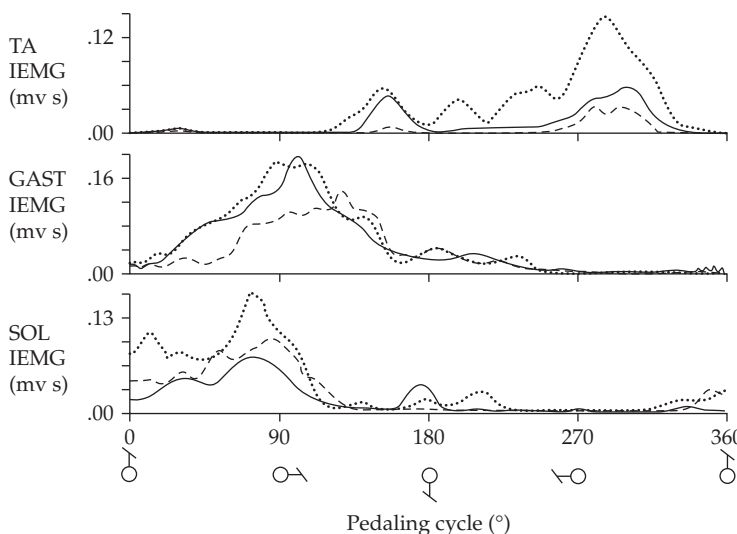


Figure 4.11 Average IEMG patterns for the tibialis anterior (TA), gastrocnemius (GAST), and soleus (SOL) muscles during the pedaling cycle at 90 W (—), 180 W (---), and 270 W (.....). (From Gregor et al., 1991b. Reprinted with permission.)

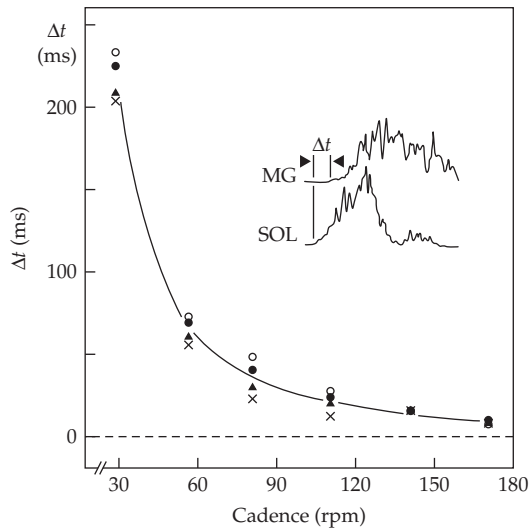


Figure 4.12 Illustration of the decrease in the delay (Δt) between onsets of soleus (SOL) and medial gastrocnemius (MG) EMGs as pedaling rate increases. Loads range from 10 N (o), to 30 N (.), to 50 N (dark triangles) to 70 N (x). Differences between loads were not statistically different, i.e., one curve was fit to all experimental results. (With kind permission from Springer Science+Business Media: Duchateau, J., Le Bozec, S. & Hainaut, K. (1986) Contributions of slow and fast muscles of triceps surae to cyclic movement. *European Journal of Applied Physiology* 55, 476–481.)

wired” as output demands change but is responsive to changes in power output requirements fully aware of the basic properties available to the nervous system through each muscle. Patterns of SOL and GAST activity, e.g., integrated EMG (IEMG), clearly show the GAST assuming a major role over the declining SOL in power transmission as power demands increase (Figure 4.13).

MTU and Muscle Fascicle Length and Velocity Changes

Further to the EMG and kinematically calculated MTU length changes, Gregor et al. (1987, 1991b) reported forces produced by the whole triceps surae group (Figure 4.14a and b), MTU length changes for the GAST and SOL muscles, EMG for the GAST, SOL, and tibialis muscles (Figure 4.14b), the muscle moment calculated using Achilles tendon forces (ATFs) with triceps surae moment arms and the net muscle moment calculated at the

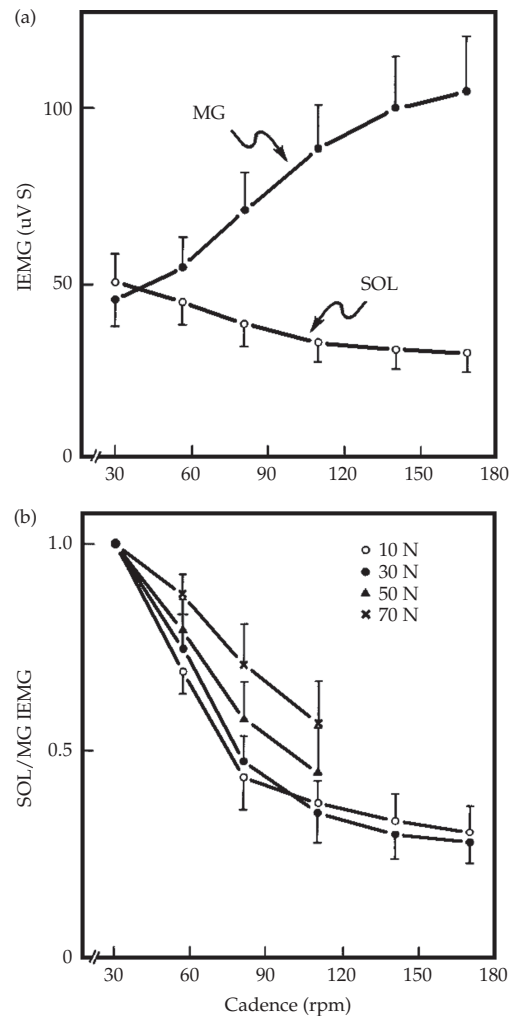


Figure 4.13 (a) Comparison of soleus (SOL) (o) and medial gastrocnemius (MG) (.) IEMG contributions to the movement of pedaling at different speeds against a load of 30 N. (b) Comparison for different loads (10–70 N) of the relative contributions of SOL and MG to pedaling at increasing speed. This figure clearly shows that the relative contribution of the SOL decreases as the speed of movement increases. Values are means \pm SE for eight subjects. (With kind permission from Springer Science+Business Media: Duchateau, J., Le Bozec, S. & Hainaut, K. (1986) Contributions of slow and fast muscles of triceps surae to cyclic movement. *European Journal of Applied Physiology* 55, 476–481.)

ankle through inverse dynamics across a range of power outputs, cadence, and load (Figure 4.14c). These data, while focused at the level of whole muscle mechanics and limb dynamics, provide

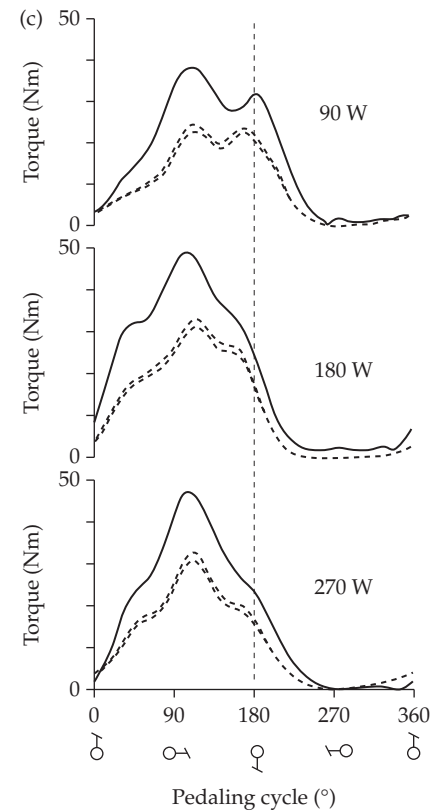
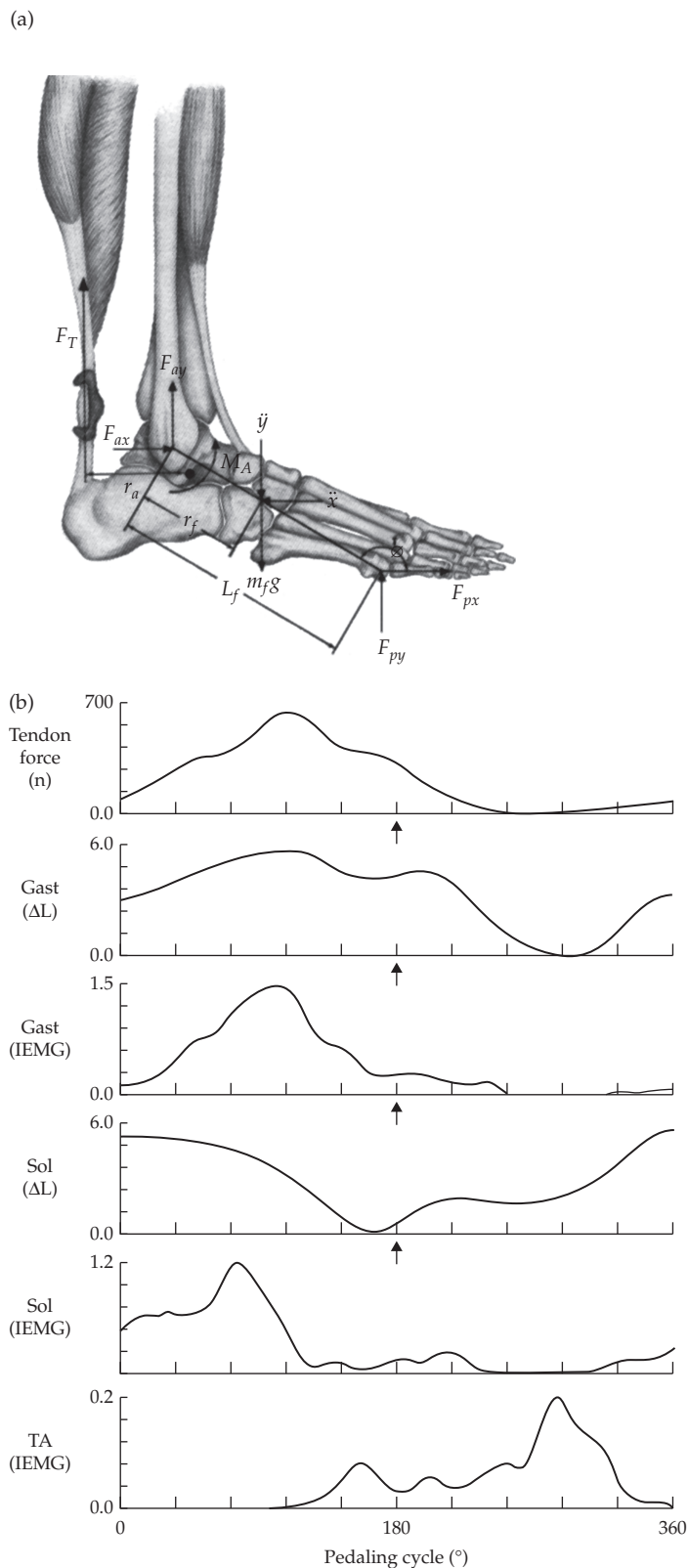


Figure 4.14 (a) Free body diagram for a rigid link model of the right leg and foot for the inverse solution and sketch of buckle placement, center of rotation location in the talus and location of AT moment arm (r_a). (From Gregor et al., 1991b. Reprinted with permission.) (b) ATF pattern, GAST, and SOL MTU length change patterns, and soleus (SOL), gastrocnemius (GAST), and tibialis anterior (TA) IEMG patterns for one subject (average of five pedaling cycles) at 265 W and 90 rpm. Arrow indicates BDC. (From Gregor et al., 1987. Reprinted with permission.) (c) Average muscle moment calculated using inverse dynamics (—), and the triceps surae moment using a fixed center of rotation (---) and moving center of rotation (...) at 90 W and 90 rpm, 180 W and 60 rpm, and 270 W and 90 rpm (From Gregor et al., 1991b. Reprinted with permission.)

a unique opportunity to compare the muscle moment produced by the common Achilles tendon (AT) and the net muscle moment at the ankle calculated using inverse dynamics. Results suggest that (1) the triceps surae impulse is about 65% of the impulse calculated using inverse dynamics and (2) that the shape of the impulses for each calculation is strikingly similar across all conditions of load and speed and across two different estimates of moment arm values. As a general reference, other investigators have reported individual muscle forces in animal models (Walmsley et al., 1978) and group muscle forces using fiber optic technology in the human AT (Arndt et al., 1998).

As previously stated, direct relationships between EMG, force, and the muscle moment are difficult to obtain (note: the activity of the tibialis anterior [TA] during the propulsion phase of cycling) but knowledge, in the human model, of each muscle's activity pattern and MTU length change pattern, calculated from kinematic data, can provide useful information. Previous reports have shown comparisons between individual muscle moments and the net muscle moment at the ankle during quadruped locomotion in a feline model (Fowler et al., 1993). In the human model, understanding individual muscle function rests on obtaining information about individual muscle activity patterns and length changes in whole muscle and muscle fibers. From these data, it can be further shown that an SSC exists in the GAST and SOL muscles during the cycling task (Figures 4.10, 4.11, and 4.14b) and that this may have some effect on muscle force production as observed by measurements from the AT. While seated cycling has smaller loads than walking, two points to keep in mind are (1) the above-mentioned SSC could potentially enhance each muscle's contribution to the force measured at the AT (Gregor et al., 1988) and (2) that a phase lag in the contribution of each muscle to the total force output appears to change with pedaling cadence which will also influence the timing of the contribution of the individual muscles to the net force at the tendon.

Pursuing the effects of cadence on the performance of specific leg muscles, Sanderson et al. (2006) reported interesting differences in the way the GAST and SOL muscles responded to changes in

cadence (Figure 4.15). It seems the peak magnitude of SOL EMG was not markedly affected by cadence, a finding supported by Gregor et al. (1991b) but not by Duchateau et al. (1986) who reported a decline in SOL IEMG as cadence increased. In contrast, the GAST muscle increased dramatically as cadence increased, supported by Gregor et al. (1991a,b) and Duchateau et al. (1986), and while having a second smaller peak in early recovery at lower cadences, this was lost at the higher cadences. Sanderson et al. (2006) attributed these differences, in part, to the higher shortening velocities experienced by the GAST MTU relative to the SOL as cadence increased; a hypothesis supported by the findings of Wakeling et al. (2006) showing higher muscle fascicle strains in the GAST as pedaling cadence increased. Further, Sanderson and Amoroso (2009) showed a significant decrease in SOL and MG EMG at the lowest seat height condition (92% trochanteric height) and that changes in ankle range of motion (ROM) affected EMG magnitudes and MTU lengths more than ROM changes at the knee as a function of seat height condition. In short, while muscles do seem to modify their activation patterns in response to changes in cadence and seat height, these changes seem to be specific to the muscle and are most likely influenced by muscle architecture, fiber type, and location in the limb. It may also be the case that the nervous system responds to the changes in power requirements by allowing length, velocity, and force feedback to modify the control pattern which is manifested, in part, by the decrease in differential onset of activity in a slow and fast ankle extensor (Figure 4.12).

Changes in limb kinematics will naturally affect the length-tension and F-V profiles, in vivo, of separate MTUs, as well as the performance of muscle groups (e.g., quadriceps and hamstrings). Sanderson and Amoroso (in press), for example, studied the effect of seat height on EMG, MTU length, and MTU velocity for muscles in the triceps surae during constant cadence (80rpm) and load (200W) and reported a decrease in both SOL and MG EMG with a decrease in seat height. This decrease in EMG was coupled with a decrease in lengthening and shortening velocities for the SOL but no significant effect was observed in the GAST as seat height changed.

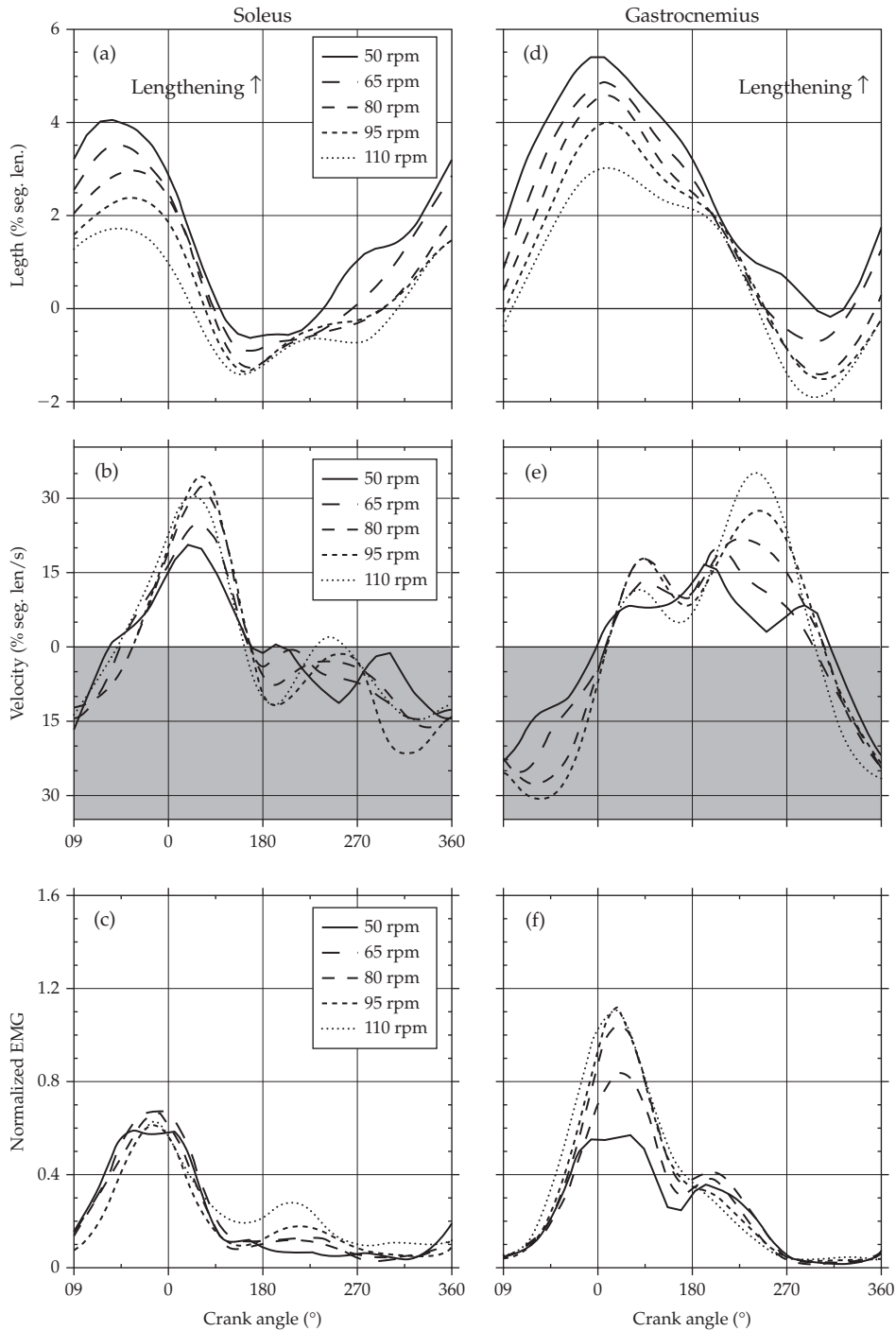


Figure 4.15 Mean length, velocity, and EMG for the soleus muscle (left panel), and the gastrocnemius muscle (right panel). Panels (a) and (d) are length change, (b) and (e) are muscle velocity, and (c) and (f) are the normalized EMG data. In panels (b) and (e), the shaded portion indicates lengthening velocity. (From Sanderson DJ, Martin PE, Honeyman G, Keefer J: Gastrocnemius and soleus muscle length, velocity, and EMG responses to changes in pedaling cadence. *J Electromyogr and Kinesiol* 2006; 16:642–649. Reprinted with permission.)

There are several issues here related to muscle architecture, one versus two joint muscles, fiber type, etc., that may influence the differential contributions of these two muscles to cycling performance and several options were presented in this report.

Because position on the bike will affect muscle mechanics, an important issue to consider at this point is defining the relationship between MTU mechanics and actual changes in muscle fiber length and velocity; changes in light of the rhythmic activity observed in all muscles during the pedaling cycle. Knowledge of muscle fascicle length and velocity would take us closer to muscle fiber behavior, cells responsible for force production, the cells in series with connective tissue and muscle force sensing transducers, i.e., GTOs, and the cells in parallel with muscle spindles responsible for providing sensory information on length and velocity changes within the muscle.

Comparisons between muscle fascicle behavior and MTU behavior during movement have been reported in a feline model (Maas et al., 2009) during overground walking on uneven terrain and results suggest that architecture plays a significant role in comparing the differences between MTU and fascicle length changes in the GAST and SOL during locomotion. For example, MTU length and velocity profiles more closely represent the profiles of fascicle behavior in the more parallel fibered feline SOL muscle than in the more pinnate fibered GAST muscles. These data on quadrupeds (Maas et al., 2005) were then associated with the local mechanical demands placed on muscle during locomotion and the potential relationship to muscle force and length sensory feedback to the CNS. Data on fascicle behavior in this animal model were obtained using implanted crystals, i.e., sonomicrometry, a technique not used in a human model.

Muraoka et al. (2001) reported muscle fiber and tendon length changes in the human VL during slow pedaling. Capitalizing on the already developed methods in ultrasound (US) (Fukanaga et al., 1997), Muraoka et al. (2001) focused on the interaction between muscle and tendon and the interdependence of their tissue properties on the integrated delivery of force generated in the muscle fibers and transmitted through the tendon to

the bone. Tendon and muscle elasticity, stiffness and mechanical stress, the SSC and movement control, energy storage and return, and enhancement of movement efficiency are all issues relevant to the study of muscle and tendon behavior during movement. Muraoka et al. (2001) suggested that the muscle fascicles operated on the descending limb of the F–L curve during the cycling task (note: the pedaling rate used in this study was 40rpm), and that the average muscle fascicle shortening velocities was less than those of the MTU during the first half of knee extension but greater during the second half of knee extension. Discrepancies were suggested to result from the nonlinear interactions between tendon and muscle and the fact that tendon enabled the VL muscle fascicles to remain closer to their optimal length and at lower shortening velocities during slow pedaling.

Finally, results from a series of studies on muscle function in humans, i.e., Wakeling et al. (2006), Higham et al. (2008), and Wakeling (2008), have been reported. Specific to cycling, Wakeling et al. (2006) used US, wavelet, and principle components analysis to investigate the contractile conditions in muscle fibers in the medial and lateral GAST and the SOL muscle during stationary cycling. Length and velocity changes in muscle fascicles were quantified using US while MTU length and velocity changes were quantified using essentially the same kinematic approach reported earlier (Sanderson et al., 2006; Gregor et al., 1991b). Whole muscle EMG patterns were obtained using surface electrodes placed on the skin overlying the SOL, medial, and lateral GAST muscles. Experimental conditions varied from 6.4Nm at 60–140rpm to 60rpm at 12–44Nm. These conditions were similar to the range of experimental perturbations discussed previously from Duchateau et al. (1986) and Sanderson et al. (2006). The basic premise for these experiments was to better understand the involvement of fast and slow muscle fibers within each muscle as mechanical demands on them changed. While the specific recruitment of different motor unit types cannot be specifically obtained using the wavelet technique and principle components analysis (Farina, 2008), information obtained in this study may provide some cautious insight to our understanding of possible shifts in fast and slow

motor unit populations involved in the control of the cycling task.

The findings from Wakeling et al. (2006) show that for any given power requirement, EMG output in the low torque/high cadence conditions was greater than the EMG output in the high torque/low cadence conditions. While EMG in all muscles increased with increased torque and cadence, SOL EMG increased more than the GAST muscle in response to increased torque; a finding similar in part to the data presented by Duchateau et al. (1986). In addition, EMG intensity for both the medial and lateral GAST muscles increased more in response to increased cadence, a finding similar to that reported by Sanderson et al. (2006). US data showed no significant effect of pedal cadence on either mean fascicle strain rate or total fascicle strain for all three muscles. Muscle fascicle strain rates were similar to MTU strain rates at the low torque conditions. Muscle fascicle strain rates significantly increased with load and cadence but cadence had a more profound effect on the total range of strain rates than did increases in torque. The highest strain rate values were recorded in the SOL muscle followed by the lateral head and finally the medial head of the GAST.

Of particular interest in these studies, e.g., Wakeling et al. (2006), is the EMG data. Despite limitations of the wavelet and principle components analysis, one of the principle findings here was the shift to higher frequency components in the MG EMG together with the higher muscle fascicle strain rates. In general, there seemed to be a "strain rate-dependent shift in frequency content of EMG that occurred independent of changes in load." So at the same load and EMG intensity, the frequency content of the EMG signal shifted to higher frequencies and this was coupled to the increases in fascicle strain rate. The investigators suggested that "in some circumstances, motor unit involvement can match the contractile properties of the muscle fibers to the mechanical demands of the contraction."

Collectively, these more recent investigations illustrate attempts at more focused analyses of muscle properties and behavior and how the motor units, the currency of the nervous system, might be

used to control the cycling task. And, this seems to be the case despite the limitations of the wavelet and principle components analysis (Farina, 2008) and the fact these studies focused on only a few more distal, more accessible muscles in the lower extremity.

Neural Control

The purpose of this final section is to review the use of the cycling task in its application to the study of the neural control of movement. While the literature base in this area is extensive, e.g., there is a growing number of studies in clinical rehabilitation, and muscle function, the studies reviewed here focus on issues in a much larger context. Muscle coordination is a primary factor in the performance of a skilled motor task. Some define it as the distribution of muscle activation, or force, among individual muscles to produce a selected combination of joint moments required by the task (Prilutsky, 2000). Some consider the dynamical interaction among the different body segments (Zajac, 1993), others consider a single-joint analysis and still others consider the multidimensional, multijoint system in a dynamical systems approach (Zajac, 1993; Kuo, 1994). A continuum of perspectives on the analysis of movement control will be presented.

The end point effect of coordinated muscle action in a cycling task is the application and control of the applied load at the pedal. In a previous section of this chapter, i.e., pedal reaction forces, a review of selected research findings was presented in which the magnitude and orientation of the reaction force vector was used to investigate cycling technique, metabolic efficiency, and mechanical effectiveness (Korff et al., 2007). Further to that, a decomposition analysis (Kautz & Hull, 1993) was presented isolating the muscle component contribution to the force applied to the pedal and crank. In a more recent series of studies, Gruben and colleagues (Gruben & Lopez-Ortiz, 2000; Gruben et al., 2003a,b,c) suggested that (1) pedal force magnitude was strongly dependent on force direction for pushing against both a fixed and moving pedal, (2) generating a large effective force at selected crank positions was

limited both by anatomical and physiological constraints, and (3) the observation of a simple force path geometry for two different tasks with dissimilar characteristics, i.e., a quasi-static pedal and a moving pedal, suggests that this aspect of foot control, i.e., the more simple force path, might be common to a range of lower limb tasks and reflect a mechanism by which the nervous system organized the control of foot force at the pedal.

As reported by Loras et al. (2009) in the process of calculating force effectiveness, it seems that by subtracting the contribution made by inertial forces to the pedal reaction force, i.e., the inertial part of the fundamental component previously discussed, the decrease in force effectiveness as pedaling rate increased was eliminated. To arrive at the magnitude of the inertial term, however, data were obtained during “unloaded” cycling, i.e., the leg did not have to overcome any external power. In this “unloaded” condition, it was assumed that any muscular activity recorded was attributed to overcoming internal energy losses and any forces recorded were caused only by the rotating mass of the lower limb. While this could be the case, a more accurate approach in calculating the inertial component would be to externally drive the “passive” limb and evaluate the inertial component under those conditions. The message here is to be cautious when interpreting data from this decomposition process and accurately estimating the actual muscular component when addressing questions in motor control. Isolating the muscular component, the one primarily controlled by the nervous system, is difficult and the process remains open to debate.

In an investigation of coordination strategies during cycling, Prilutsky and Gregor (2000) developed a 2D model of the lower extremity to predict muscle force patterns using two techniques, i.e., pushing and pulling the pedal. Using different optimization criteria to compare these predictions with EMG patterns, some major findings included (1) EMG linear envelopes, muscle moment arms, and estimates of maximum muscle force accounted for more than 80% of the variation of joint moments obtained from inverse dynamics, (2) changes in the relationship of EMG-muscle force due to muscle length and velocity changes during the conditions

employed in this study were relatively small, and (3) the optimization criterion that minimized the sum of muscle stresses cubed demonstrated the best performance in predicting the relative magnitude and patterns of muscle activation. The authors went on to suggest that the functional significance of muscle coordination strategy in cycling might be the minimization of fatigue and/or perceived exertion, certainly the two phenomena also studied in exercise physiology and motor control. Hence, the use of the rhythmic cyclic movements in the cycling task, controlling for speed and load at the crank and position of the rider with respect to the load, allows the investigators to study a variety of questions related to musculoskeletal biomechanics and neural control.

In a more general reference to muscle coordination, Prilutsky (2000) presented a conceptual model of connections among motor neuron pools of one- and two-joint muscles in the human lower extremity (Figure 4.16). This model, in concept, is similar to the approach taken by Eccles et al. (1957), Eccles and Lundberg (1958), and Grillner (1981) regarding the potential interconnections among muscles in the cat hindlimb regarding excitatory and inhibitory effects among force and length sensors and their influence on the strategies developed in response to external mechanical demands. With respect to the data presented by Duchateau et al. (1986), for example, the conceptual model proposed by Prilutsky (2000) may help explain the shift in influence of the SOL and GAST on the applied load at the pedal as load and cadence increase. GAST force increases as cadence increases, actually an increase observed in EMG intensity, and Duchateau (1986) shows a decrease in SOL EMG and hence the potential for a decrease in force. As shown in Figure 4.16, ankle extension provides a force-dependent excitation to the GAST and SOL muscles. It is also shown, however, that as the GAST force increases it provides a force-dependent inhibition on the SOL muscle. In part, this may explain the decrease in SOL excitation observed in reports on cycling. These data are to some extent similar to findings previously reported in the feline model (Fowler et al., 1993; Gregor et al., 2006). It must be remembered, however, that the hindlimb system and the

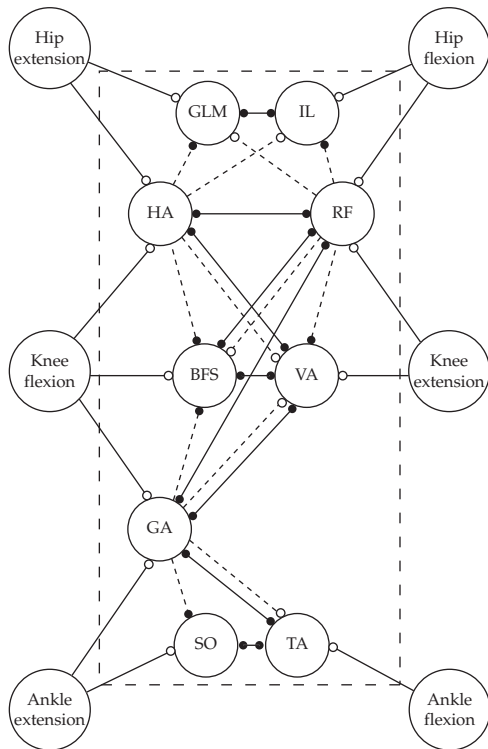


Figure 4.16 A conceptual model of connections among motor neuron pools of one- and two-joint muscles of the human lower extremity. Motor neuron pools of one- and two-joint muscles receive excitation inputs (open knobs and solid lines) from flexion and extension joint centers (large circles). The distribution of excitation inputs to synergistic muscles crossing a given joint corresponds to the following rules: More excitation is allocated to the muscles with a longer moment arm and a larger physiological cross-sectional area (PSCA). Two-joint muscles can receive excitation from two joint centers. Both one- and two-joint antagonistic muscles inhibit each other through reciprocal inhibition (closed knobs solid lines). At high forces, two-joint muscles can inhibit their one-joint synergists (force-dependent inhibition, closed knobs dotted lines) and excite their one-joint antagonists (force-dependent excitation, open knobs and dotted lines). The activation of each motor neuron pool is assumed to be proportional to the sum of all positive (excitatory) and negative (inhibitory) inputs to the pool. This model can potentially account for the observed coordination between one- and two-joint muscles at different combinations of joint moments. (GLM, gluteus maximus; IL, iliopsoas; HA, hamstrings; RF, rectus femoris; BFS, biceps femoris short head; VA, vastii; GA, gastrocnemius; SO, soleus; TA, tibialis anterior.) (From Prilutsky 2000. Reprinted with permission.)

human lower extremity system are not simple and there are other interactions shown in Figure 4.16 as well as interactions not shown, e.g., joint receptors, that can influence the results reported by for example, Duchateau (1986), Fowler et al. (1993).

Several reports have been made showing the effects of pedaling cadence on muscle coordination during cycling using dynamical systems analysis (Raasch & Zajac, 1999; Zajac, 2002). Raasch et al. (1997), for example, utilized an optimal control algorithm to understand fundamental muscle coordination principles associated with delivery of energy to the crank, i.e., in one- and two-joint muscle coordination, in the clearly defined task of maximum speed pedaling. Results suggest that a simple neural control strategy, i.e., partitioning muscles into alternating functional groups, while not a unique concept (see earlier), can produce the fundamental coordination needed to pedal effectively. The proposal that a strategy using two phase-controlled signals with all muscles partitioned into two functional groups finds support in the literature and is related to centrally generated primitives, e.g., central pattern generators (Grillner, 1981) modulated by a sensorimotor control mechanism to address any perturbations in crank loads. More recently, Zehr (2005) proposed that all rhythmic motor patterns, e.g., walking, cycling, swimming, may share a common neural control mechanism. This idea has been supported with indirect evidence that walking, leg and arm cycling as well as recumbent stepping could share a “common core” of control (Zehr et al., 2007).

Neptune et al. (1997) extended the domain of the work related to maximum pedaling to the effect of submaximal pedaling rates on neuromuscular coordination. Varying cadence from 45 to 120 rpm at a constant power of 250 W shows that muscle “on time” advances in the pedaling cycle as pedaling rate increases except for the slow SOL muscle which shifted to later in the pedaling cycle. The advance in timing of the MG in this report is consistent with the findings of Duchateau et al. (1986), but the shift of SOL onset to later in the pedaling cycle with increased cadence is in contrast to the report by Duchateau (1986) who showed the SOL to also advance in the cycle as cadence increased (Note: the

range of cadences in both studies had a great deal of overlap.) Neptune et al. (1997) further reported EMG intensity to be muscle specific with respect to increases in IEMG with increased cadence but that their “phase-controlled functional group analysis” indicated the functional contribution of each muscle, across the range of cadences, remained very similar (see Raasch & Zajac, 1999, for a detailed discussion of the phase-controlled functional group analysis). Finally, the data from Neptune et al. (1997) suggest that all muscles, except for the RF and SOL, were activated for a single functional role. The RF and SOL were excited for two functional phases; the RF as a “top transition-extensor muscle” and the SOL as an “extensor bottom transition muscle.” The former result is somewhat intuitive but the latter is in stark contrast to several other reports in the literature on the role of the SOL muscle in the cycling task, e.g., Ryan and Gregor (1992).

Because cycling is a bilateral task, questions related to how one limb affects the control system of the other limb becomes important; indeed, the control system regulates both legs and each limb has an effect on the control of the contralateral limb. The lower extremities are mechanically coupled by the crank as the foot is secured to the pedal and anatomically/mechanically coupled by the pelvis at the hip. While this coupling is an important part of this task, the effect one limb has on the other has received attention only recently. For example, one of the first reports on this issue of coupling and neural control using a human model was by Boylls et al. (1984). Influenced by literature using animal models (Kulagin & Shik, 1970), Boylls and colleagues evaluated “neural constraints” of the bilaterally paired lower extremities using EMG of four muscles and a stationary bicycle (Monark) modified to allow angular velocity ratios other than 1:1 at the two cranks. Bilateral limb movements in humans have been associated with “neurally mediated restrictions” on inhibitory crosscoupling, force scaling, and movement synchronization. Because the EMG timing patterns displayed little variability outside that observed with normal coupling at the crank, it was suggested that the trajectory of each leg is actively regulated during pedaling and interlimb neural constraints were absent (Boylls et al., 1984).

Ting et al. (1998) continued this work investigating the sensorimotor state of the contralateral leg and its effect on ipsilateral muscle coordination during cycling. These experiments were designed to determine whether independent central pattern generators, central interlimb neural pathways, as opposed to the use of mechanical coupling and ipsilateral feedback, were used to control the legs during cycling. Two-legged and one-legged cycling tasks were designed and matched mechanically to ensure loading on the single limb during one-legged cycling was identical to that imposed on that limb during two-legged cycling. Data from these experiments show that during limb flexion, i.e., primarily during recovery, major flexors increased their EMG activity and reduced the negative loading on the crank during flexion by 86%. These data support the notion that inhibitory pathways exist from the contralateral extensors onto ipsilateral flexors and that these pathways operate during two-legged cycling but not during one-legged cycling. While practice seemed to allow the cyclist to overcome these centrally mediated coupling programs, the investigators concluded that sensorimotor control of the “unipedal” task was affected by interlimb neural pathways and that the limbs do communicate seemingly coupled mechanically as well as through centrally mediated interlimb neural pathways.

Ting et al. (2000) introduced a series of perturbations, i.e., during one-legged cycling, to the contralateral limb that included 1) it generating an extensor torque, 2) being relaxed and held in a stationary position and 3) moving in antiphase to the pedaling limb, i.e., the ipsilateral limb, by a servomotor. These conditions extended the previous experimental paradigm and allowed a more extensive evaluation of interlimb neural coupling. Consistent with the previous report, during the presence of a contralateral extensor force, muscle activity in the pedaling limb, especially in the biceps femoris (BF) and semimembranosus (SM), was reduced, e.g., 25–30%, regardless of the amplitude of contralateral force or whether the limb was moving or not. In contrast, the TA and the RF activity were reduced only when the contralateral limb generated a high rhythmic force during leg movement. Differences between these two muscle

pairs have been previously addressed (Neptune et al., 1997; Raasch et al., 1997) and focused on the fact that the TA and RF accelerated the limb anteriorly and the BF and SM accelerate it posteriorly. These data are also consistent with reports on contralateral feedforward and feedback mechanisms controlling hamstring activity and RF and TA activity, respectively, during flexion (Ting et al., 2000). While several details in both studies (Ting et al., 1998, 2000) are important, it is safe to say that the sensorimotor state of the contralateral limb during cycling is important in shaping the motor pattern during the flexion phase of the pedaling cycle.

These conclusions were further substantiated in a report by Kautz et al. (2002) in which “uncoupling” of the two limbs was achieved using a servomotor system on the contralateral limb. The primary focus of that work was to determine whether rhythmic force generation from the contralateral limb substantially affected the bifunctional muscle activity in the ipsilateral limb during the limb transition phases of the pedaling cycle, i.e., flexion-to-extension around TDC and extension-to-flexion around BDC. It has been suggested in previous reports (Ting et al., 1998; Raasch & Zajac, 1999) that bifunctional muscles, i.e., two-joint muscles, are dominant contributors to the smooth transition around TDC and BDC in the pedaling cycle. Sampling EMG from four major thigh muscles results of these experiments suggest that neural interlimb coordinating mechanisms “fine-tune” bifunctional muscle activity during cycling to ensure the transitions mentioned earlier are executed effectively (Kautz, 2002).

The thread through most of these experiments in neural control focuses on the evaluation of muscle function within and between limbs in the control of external work during cycling. Knowledge of muscle properties, EMG patterns, and limb dynamics are important but only tools in support of the general hypotheses related to neural control and how the cycling task can be used in those studies.

Cycling in Challenged Populations

Cycling with a lower limb amputation presents a new set of challenges in that the prosthesis introduces an additional mechanical interface that the

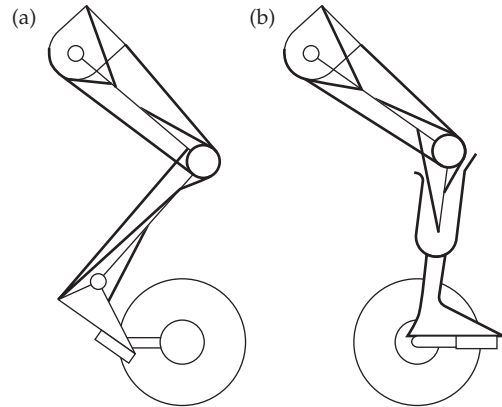


Figure 4.17 Schematic of the lower limb showing an intact limb (a) and a limb with a transtibial amputation (b). Major muscle groups are shown as thick lines. Note the gastrocnemius muscle has been altered by the amputation to a single-joint knee flexor.

cyclist must utilize to effectively apply and direct forces to propel the bicycle. For example, a unilateral transtibial amputee has lost the structure of the ankle joint as well as the surrounding musculature and the sensory information the amputated tissue previously provided. The person must now interact with their environment via a prosthesis on one limb and a fully intact limb on the contralateral side (Figure 4.17). Similar to the studies discussed earlier on intact cyclists, the cycling task also provides an excellent method to study how this asymmetric system is able to perform this task. The coupled nature of cycling also allows for transfer of energy between limbs via the crank and the pelvis so that one limb may compensate for the other (presumably deficient) limb. This compensation between the amputated and nonamputated limbs has been reported as asymmetric work production at the crank (Childers et al., 2009a). Results from Childers et al. (2009a) suggest that (1) work asymmetries within cyclists with transtibial amputation (CTA) are higher than the intact population (i.e., approximately 30% vs. 4% respectively), (2) the inertial and strength differences between limbs cannot account for the increase in work asymmetry, and (3) possible deficiencies in the amputated limb’s ability to direct force required the compensation (and thus increase in asymmetry) observed in the CTA group.

A similar trend has been seen in cyclists with a transfemoral amputation (CTFA). CTFA have had the ankle and knee joint removed from one limb and perform with work asymmetries between 50% and 60% (unpublished data).

An amputation through the tibia (transtibia) results in the alteration of the GAST muscle from a two-joint knee flexor/ankle extensor to a single-joint knee flexor. Likewise, an amputation through the femur (transfemoral) results in the alteration of the RF from a two-joint hip flexor/knee extensor to a single-joint hip flexor, whereas the other, more proximal muscles have not been surgically affected by the amputation and retain their original origin/insertion points. Childers et al. (2009a,b) reported a shift in peak activation in the GAST muscle (Figure 4.18) to much later in the crank cycle (107–178°) for three of the six CTAs tested and suggested this shift in activation reflected the muscle's change in function from a two-joint knee flexor/ankle extensor to a single-joint knee flexor. Additional unpublished data on one CTFA indicates peak activation of the RF shifts from 30° after TDC to 59° before TDC (Figure 4.18) and resembles data reported by Juker et al. (1998) on the psoas muscle thereby reflecting its new functional role as a single-joint hip flexor.

Conclusions

The cycling task is widely used in the fields of exercise, rehabilitation science, musculoskeletal mechanics, and neural control. In fact, in the development of rehabilitation programs cycling is used extensively in both neurorehabilitation and rehabilitation in orthopedics. This is primarily because it offers an intervention that can be controlled by the user in aspects related to load and speed in different body positions. Just as elite competitive cyclists match the bicycle to their specific morphology and strengths, the clinician, exercise specialist, and physical therapist must “match” the bicycle to the rider or patient to meet their specific strengths and weaknesses. The riders' physical interface with the bicycle, e.g., “bike fit,” as well as the load and cadence can be regulated by the therapist, exercise specialist, musculoskeletal biomechanist, or scientist in neuromechanics. The objective of this chapter is to convey the idea that cycling is a very useful task with wide ranging applications

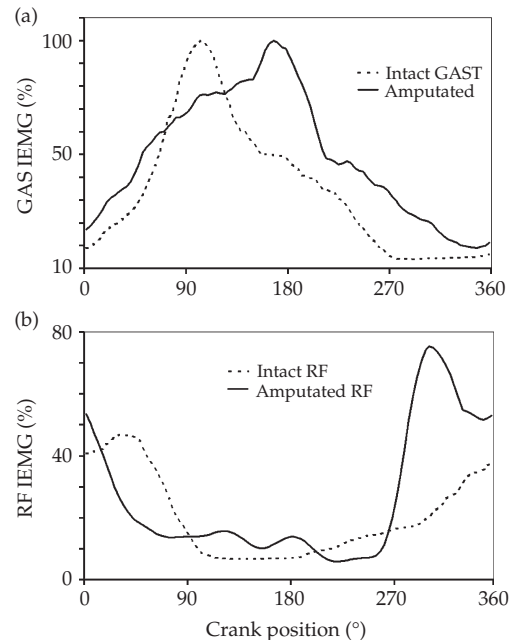


Figure 4.18 (a) Average IEMG of the gastrocnemius (GAS) muscle from the intact (dashed) and amputated (solid) limbs showing a shift in activation to later in the crank cycle for the amputated GAS. (From Childers WL, Kistenberg RS, Gregor RJ: The biomechanics of cycling with a transtibial amputation: Recommendations for prosthetic design and direction for future research. *Pros Orthot Int* 2009; 33: 256–271. Copyright 2009 informa healthcare. Reprinted with permission.) (b) Average IEMG of the rectus femoris (RF) muscle from the intact (dashed) and amputated (solid) limbs showing a shift in activation to earlier in the crank cycle for the amputated RF (unpublished data).

in the movement sciences and a task that can be used to study a variety of issues in human movement control. We began with a description of cycling output mechanics, discussed muscle, and the properties of skeletal muscle considered when evaluating how, as an end organ to the nervous system, it can be used to control movement, and finally a discussion of issues in neural control, e.g., central pattern generators, and the mechanical and neural coupling within and between limbs. The kinematics and kinetics of the task from the level of the motor unit to the whole lower extremity have been presented and applications to individuals with special needs discussed. The movement scientist can use this information to continue to develop unique applications of this task to improve human performance.

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Chapter 5

Kinetics and Muscular Function in Alpine Skiing

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Introduction

In many regions in the world, alpine downhill skiing is one of the most popular and most frequently practiced form of winter sport. In alpine regions, this fact is of high significance above all from economic, social, and health aspects. Within the last 20 years, the ski industry created a very successful innovation with the development of the carving ski system. In ski racing as well as in recreational skiing, skis have become much shorter, their side cut has increased to a great extent and binding plates (risers) have been fixed between the ski and the binding. In addition the stiffness of the ski has also changed. This evolution has, of course, also changed the movement patterns of performing ski turns; yet it might also have changed the risk of sustaining injuries. This chapter will give an overview on the dynamics of downhill skiing techniques. This will be followed by a comparison of biomechanical aspects of traditional and carving turning techniques in recreational skiing. Finally, some new insights into muscle fatigue during recreational skiing will be given.

The Dynamics of Downhill Skiing

In the following paragraph, an overview of the external forces working on the skier's body is

given. We differentiate it into the three important skiing techniques, gliding straight down the fall line, gliding straight across the slope, and performing a turn (Müller et al., 2008).

Straight Gliding Down the Fall Line

At the skier-ski system the gravitational force \vec{F}_G acts at the CoG. This force can be divided into two force components, \vec{F}_S , the force parallel to the slope plane that causes the acceleration of the skier and \vec{F}_N , the normal or perpendicular force to the slope (Figure 5.1).

The size of each of these forces depends on the angle α of the slope:

$$\vec{F}_S = \vec{F}_G \times \sin(\alpha)$$

$$\vec{F}_N = \vec{F}_G \times \cos(\alpha)$$

The force \vec{F}_{reac} represents the reaction of the snow to the ski. It is the sum of the distributed forces due to the snow pressure on the bottom of the ski. \vec{F}_{reac} works in the opposite direction of \vec{F}_N . (Müller, 1991; Howe, 2001; Lind & Sanders, 2004). The forces \vec{F}_N and \vec{F}_{reac} cause a torque (\vec{M}), when the lines of action of those forces do not coincide.

The skier is decelerated by the snow friction force \vec{F}_f and the air frictional force \vec{F}_D , which are directed opposite to the skier's velocity. In a simplified model, the snow friction force that acts along the contact area between ski and snow depends on

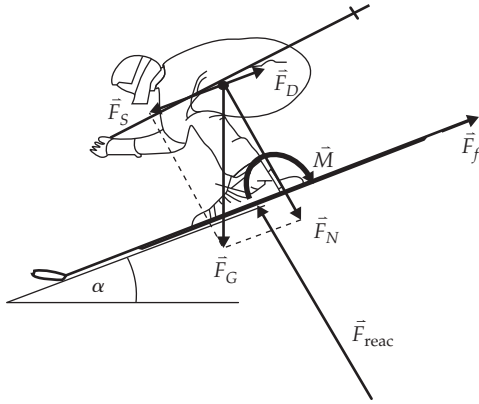


Figure 5.1 Force diagram of a skier gliding down the fall line (Müller et al., 2008).

\vec{F}_N and the composition of the contact surfaces of the ski and snow (μ) (Müller, 1991).

$$\vec{F}_f = \mu \times \vec{F}_N = \mu \times \vec{F}_G \times \cos(\alpha)$$

where μ is the sliding friction coefficient.

The air frictional force \vec{F}_D emerges from the resistance between the skier-ski system with the air. \vec{F}_D depends on the air density, the magnitude of the frontal area of the body, the square of the velocity of the skier, and a drag coefficient:

$$\vec{F}_D = C_D \times A_p \times \delta \times \frac{v^2}{2}$$

where C_D is the drag coefficient, A_p the frontal area of the body, δ the air density, v the velocity.

The drag coefficient (C_D) depends on the aerodynamic shape of the skier. An aerodynamic lift force \vec{F}_L is also present and depends on the velocity of the skier. This force works in the opposite direction of \vec{F}_N , reducing the snow friction force (Müller, 1991; Van Ingen Schenau et al., 1998; Howe, 2001; Lind & Sanders, 2004). In recreational skiing, this force is negligible.

Gliding Across the Slope (Traverse)

A slope is tilted at an angle α with a skier crossing the slope at the traverse angle γ , relative to the horizontal, as shown in Figure 5.2.

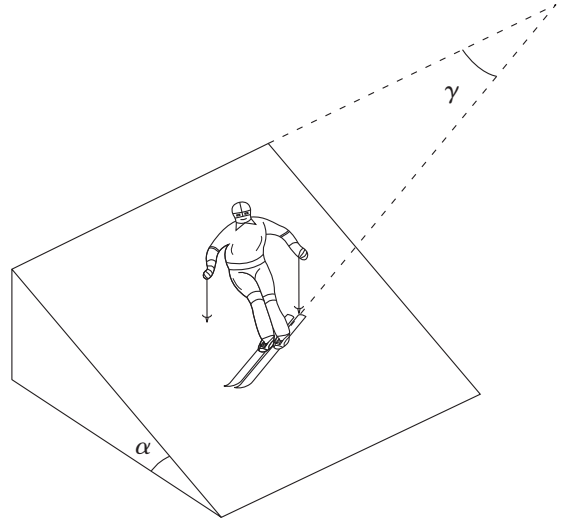


Figure 5.2 Geometry of gliding across the slope (Müller et al., 2008).

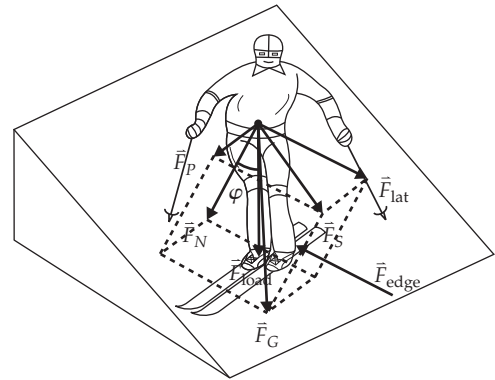


Figure 5.3 Force diagram of a skier gliding across the slope (Müller et al., 2008).

When skiing across the slope, the gravitational force is divided into the two force components \vec{F}_S and \vec{F}_N . \vec{F}_S is further separated into the two force components, \vec{F}_p , the force parallel to the ski track and \vec{F}_{lat} , the force perpendicular to the ski track. \vec{F}_p is the only force component that accelerates the skier (Figure 5.3).

The magnitude of both \vec{F}_p and \vec{F}_{lat} depends on the angle of slope α and the angle of the

direction of motion γ (Müller, 1991; Lind & Sanders, 2004):

$$\bar{F}_p = \bar{F}_G \times \sin(\alpha) \times \sin(\beta)$$

$$\bar{F}_{lat} = \bar{F}_G \times \sin(\alpha) \times \cos(\beta)$$

To avoid skidding caused by \bar{F}_{lat} , the skier has to produce an equal and opposite reaction force. The force \bar{F}_{load} bends the ski and pushes it perpendicularly into the snow surface. The edges carve into the surface with an edging angle φ and provide the equal and opposite force \bar{F}_{edge} to counteract \bar{F}_{lat} . The force \bar{F}_{load} is defined as (Lind & Sanders, 2004):

$$\bar{F}_{load} = F_G \times \sqrt{(\cos^2(\alpha) + \sin^2(\alpha) \times \cos^2(\gamma))}$$

Forces While Skiing a Turn

Figure 5.4 shows the main forces that are working on a skier while performing a turn. In addition to

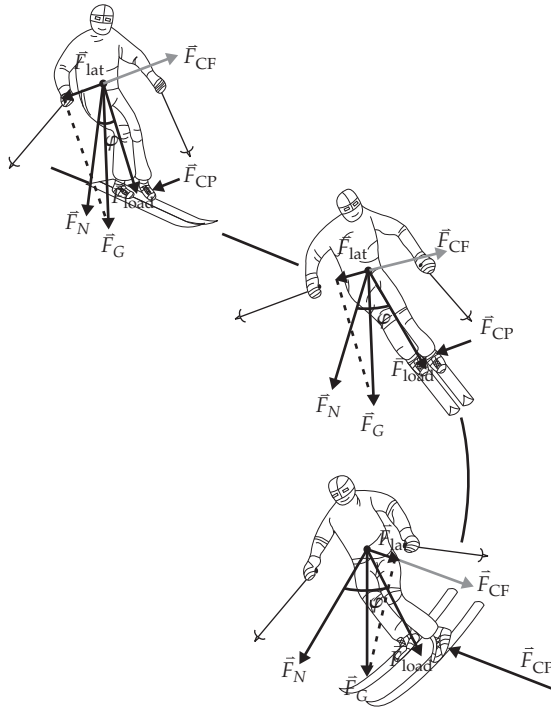


Figure 5.4 Force diagram of a skier performing a turn (Müller et al., 2008).

these forces, the forces shown in Figures 5.2 and 5.3 are also working, but they are less important for the turning technique.

The turning radius is mainly determined not only by the geometry of the edged and flexed ski in contact with the slope, but also by other factors like snow conditions, torsional stiffness of the ski, and preparation of the slope (Howe, 2001).

To ski a turn, the ski has to be placed on the edge with an edging angle φ . The snow reaction force, \bar{F}_{reac} working at the edges of the ski can be divided in two components. One component is working vertically against the gravitational force \bar{F}_G , and the other component is the centripetal force \bar{F}_{CP} working toward the center of rotation. The centripetal force \bar{F}_{CP} causes a torque which pushes the skier to the outside of the turn. To stay in balance, the skier has to lean inward with an angle ε . The inward leaning angle ε depends on the skiing velocity (v), the radius of the CoG (r), the gradient of the slope, and the angle of the direction of motion γ . The sum of the forces working on the CoG (\bar{F}_{load}) acts precisely through the ski-snow contact area to balance the reaction force (Figure 5.5).

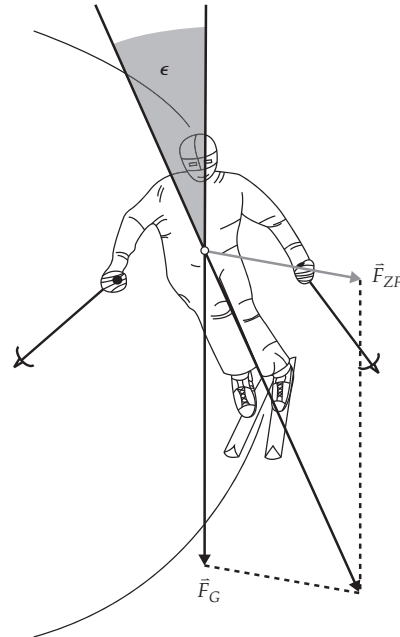


Figure 5.5 Dynamical balance in turning (Müller et al., 2008).

In this situation, \bar{F}_{load} is calculated from the force normal to the slope (\bar{F}_N), the force parallel to the direction of motion (\bar{F}_p), and the force perpendicular to the direction of motion (\bar{F}_{lat}). In the steering phase 1 (until the fall line), (\bar{F}_{lat}) acts toward the center of curvature. In steering phase 2 (from fall line until edge changing), (\bar{F}_{lat}) is pointing away from the center of curvature (Müller, 1991). The previous descriptions of the forces in the diagrams are regarding an inertial reference frame. However, it is also possible to describe turning in a moving reference frame with the skier as the frame of reference. Then, the circular motion of the turn generates a centrifugal force (\bar{F}_{CF}) (gray color in Figure 5.5). The centrifugal force acts on the CoG of the skier-ski system and is directed outward radially at each point of the circular path of the turn. The centrifugal force depends on the velocity (v), the mass (m) of the skier, and the radius (r) of the centre of mass (CM):

$$\bar{F}_{CF} = m \times \frac{v^2}{r}$$

When the skier is defined as the frame of reference, \bar{F}_{CF} is produced to allow the sum of the forces working on the CoG (F_{load}) to act precisely through the ski-snow contact area to balance the reaction force. In an inertial reference frame, it is the inward leaning angle ε maintaining this balance.

Biomechanics of Carving Techniques

Since the introduction of the new carving ski systems in ski racing as well as in recreational skiing,

the so-called “curved turn” is considered to be the ideal turning technique. In carving, steering the turn should be performed along the ski edges without any lateral skidding. The ideal curve radius during carved turns (CTs) is a function of ski waist, on-edge angle, and ski/snow flexion. The more strongly waisted the ski and the greater the on-edge angle the more strongly the ski must flex in order to maintain contact with the slope along the total length of the edge. The curve cut into the snow under full contact with the slope is designated the turn radius. Figure 5.6 shows the ideal curve cut radius as a function of the on-edge angle with variously waisted skis (Müller & Schwameder, 2003).

To compare the new carving technique with the traditional parallel steering technique, a complex analysis was done at the University of Salzburg using kinematic, kinetic, and EMG methods (Müller et al., 2005). An excellent carving skier and former World Cup ski racer performed eight test runs on a well-prepared piste with both carving (side cut radius: 14 m) and traditional giant slalom skis (side cut radius: 32 m). Kinematic analysis of the runs was done using three video cameras and the Peak 3D system. GRFs were measured by implementing bilateral mobile pedar insoles (Novel, Munich, Germany) into the boots of the subject. Simultaneously, it was possible using EMG to observe the activities of the gluteus maximus, vastus medialis (VM), VL, RF, BF, TA, and peroneus longus muscles (Biovision system, Frankfurt, Germany) (Figures 5.7 and 5.8).

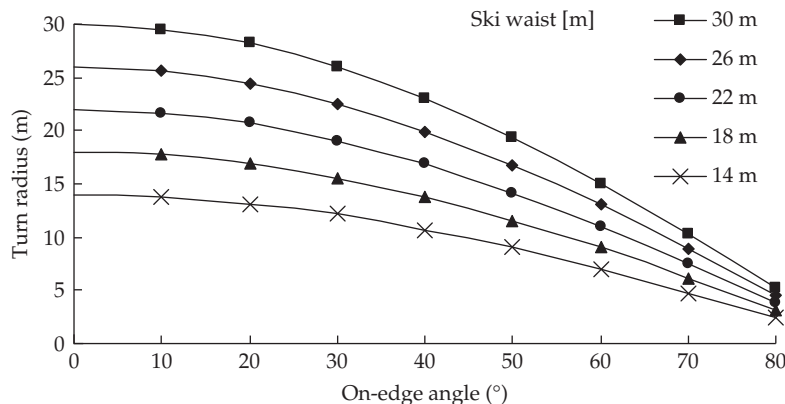


Figure 5.6 Turn radius as a function of on-edge angle for variously waisted skis (Müller et al., 2005).

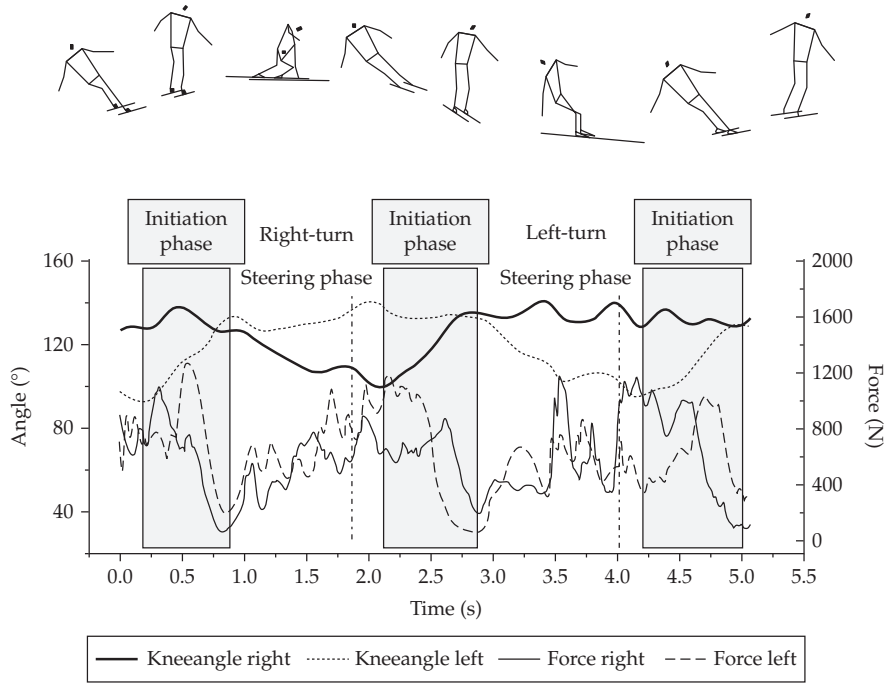


Figure 5.7 GRFs and knee angles of CTs (Müller et al., 2005).

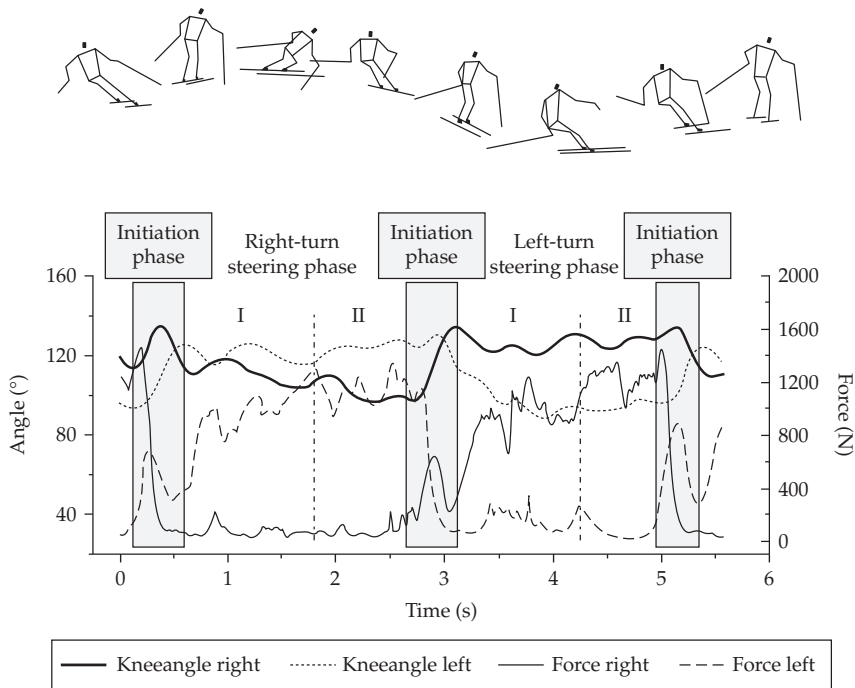


Figure 5.8 GRFs and knee angles of parallel (skidded) turns (Müller et al., 2005).

Summing up the results of the comparison between the traditional parallel and the new CT, the following aspects seem to be mostly relevant:

- In the traditional parallel turn (PT), during the steering phases, the predominant load is on the respective outer ski whereas we find intensive coloadings of the inner leg in all turning phases with the carving technique (Figures 5.7 and 5.9).
- The total loading forces are much higher in CTs. The higher loading is mainly a result of higher speed, smaller turning radii and therefore higher centrifugal inertial forces. But the total loading is almost equally distributed to both legs.
- The intensive coloadings of the inside leg (IL) results in a slow SSC (about 1300 ms) of the knee extensor muscles (VM) of the IL during the steering phase and the initiation phase (Figure 5.10).

Toward the end of the eccentric loading phase, the GRFs of the IL reach values of about 120% body weight with a knee angle of about 100°.

- The carving technique has a relatively short second steering phase (steering the skis out of the fall line) and a comparatively long initiation phase.
- The turn initiation phase of the carving technique is in structure very similar to that of the traditional PT. Up-unweighting in combination with edge changing takes place in both techniques.
- Turns with carving skis demand obviously better sagittal balance ability as well as an improved edge steering ability in order to be able to stay centrally positioned over the ski.
- The more waisted and more flexible skis together with the greater on-edge angles during the steering phase make the much smaller turning radii with the new carving technique possible.

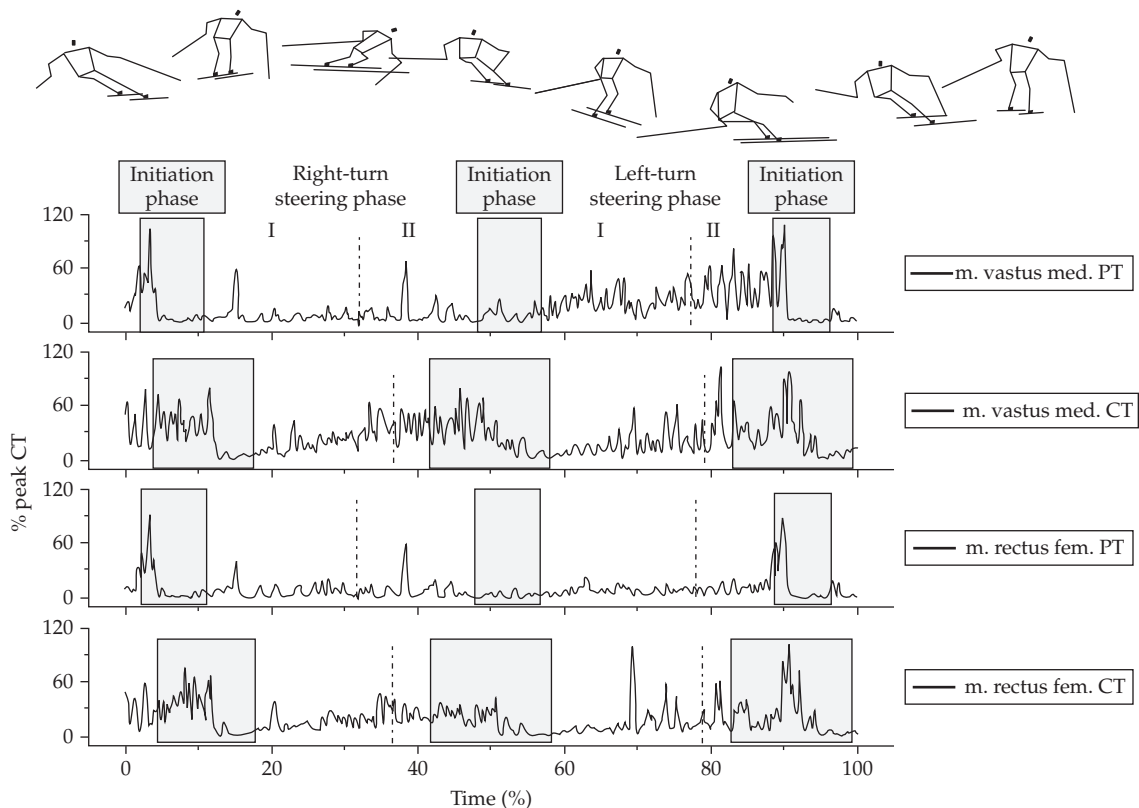


Figure 5.9 Comparison of EMG activities between traditional parallel turns (PTs) and carving turns (CTs) (Müller et al., 2005).

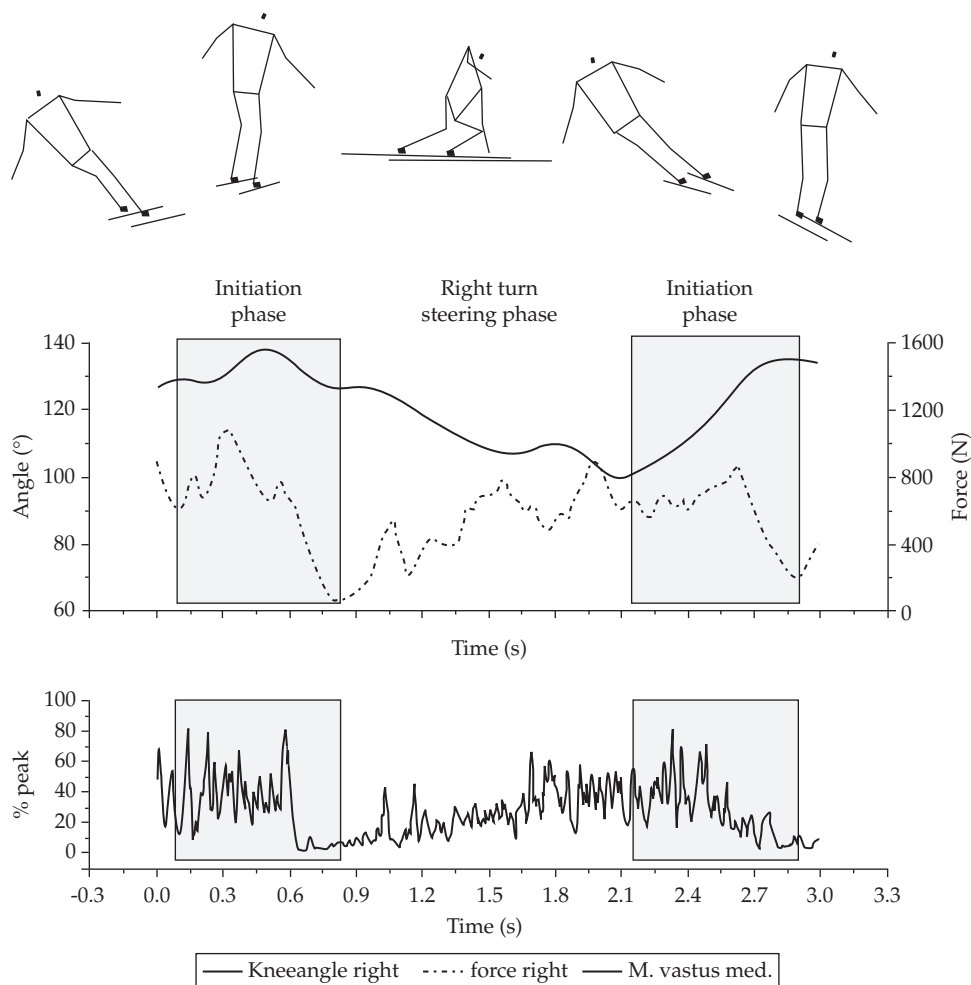


Figure 5.10 Skiing-specific SSC of *M. vastus medialis* of the IL performing a CT.

But even the most experienced skier is not able to carve the turn without any skidding component. This has already been described by Lind and Sanders (2004, p. 139) as follows: “Even the most purely CT must involve some skidding. In all actual CTs some parts of the ski’s edge will yaw under loading as the surface of the snow deforms and fails beneath it, and those parts of the edge will skid rather than carve.”

In a recently finished study (Wagner, 2006), the skidding angle β (angle of attack) was introduced in order to calculate the amount of skidding during a turn. As it is shown in Figures 5.11 and 5.12, β is defined as the angle between the line of the end

and of the tip of the ski and the velocity vector of the ankle of the skier’s leg. The smaller the angle β , the less skidding is performed during the turn.

In this 3D kinematic analysis, various typical ski turning techniques for recreational skiing were compared with each other focusing on the parameter angle β . One expert skier performed six different turning techniques with three trials of each technique. Figure 5.13 shows the angle(β)–time curve during a complete CT. Both turns are divided into three phases as described in Müller et al., 1998. Phase 1 represents the first steering phase. It starts with the beginning of edge changing and

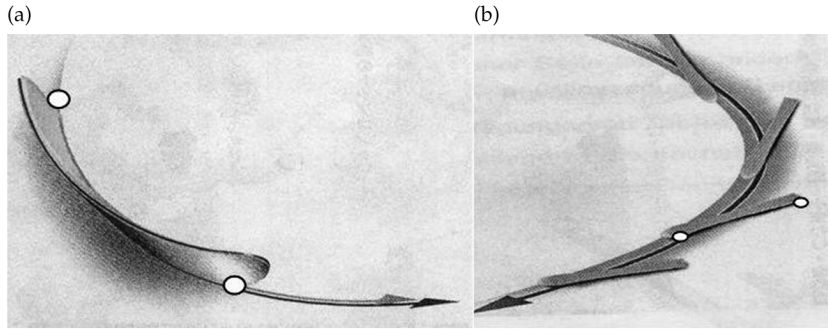


Figure 5.11 Representation of an ideal CT (a) and a skidded turn (b) (Müller et al., 2008).

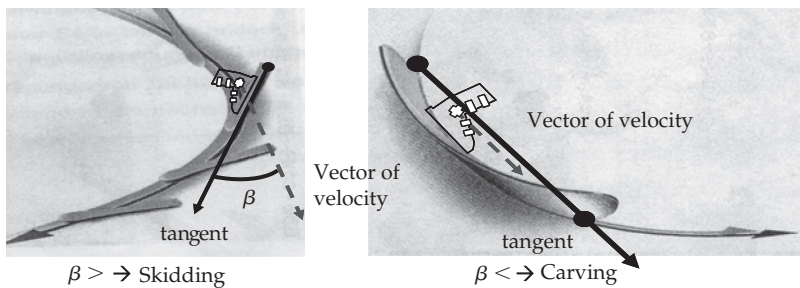


Figure 5.12 Definition of skidding angle β (angle of attack), a variable to define carving and skidding (Müller et al., 2008).

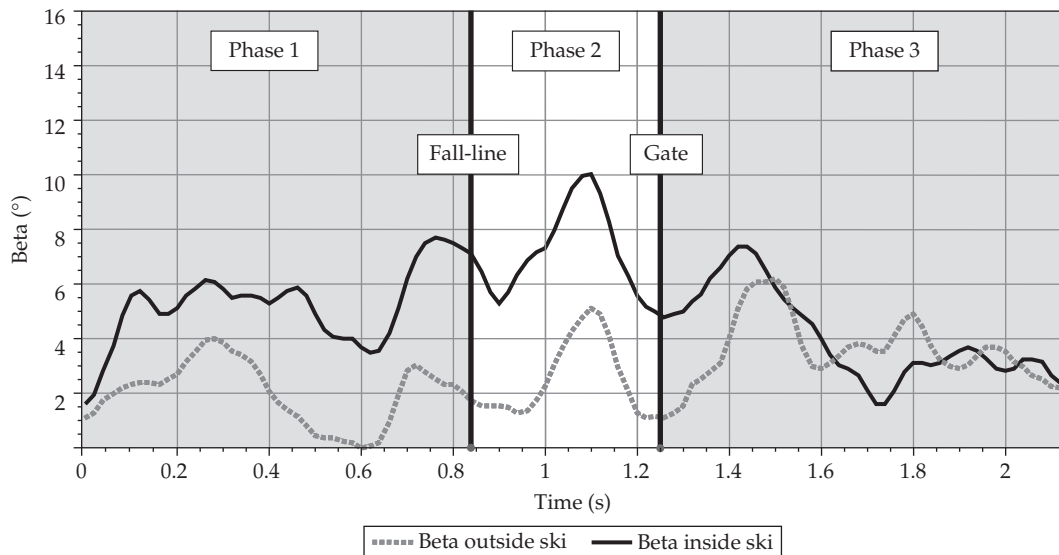


Figure 5.13 Angle β as a function of time in a CT (Müller et al., 2008).

lasts until the outside ski has reached the fall line. Phase 2 starts with the fall line and finishes when the gate has been passed. Phase 3 ends when the edge changing starts again. The angle β of the CT is indeed much smaller compared with the skidded

turn. Its range lies between 0° and 10° , the average value is 4.1° , whereas the range of the skidded turn lies between 5° and 25° with an average of 18° . In this study it was possible for the first time to quantify the amount of skidding or carving during

real-performed turns in skiing. It could be shown that CTs without any skidding hardly seems to be possible. But it could also be shown that the amount of skidding during turns differs a lot within the various turning techniques.

Muscle Functions and Muscle Fatigue in Recreational Skiing

Surface EMG is often used to evaluate muscle activity during movement. A review of EMG literature during alpine skiing indicates that amplitude and timing pattern of muscle activity have been analyzed along with the relative activity of different muscles (Berg et al., 1995, knee extensors). Due to the involvement of different muscle fiber types (slow- and fast-twitch fibers) and their different motor unit action potentials, the frequency of an EMG signal contains additional information about muscle fiber activation. When a muscle is active, the fast fibers can generate higher frequencies within the signal than slow fibers (Wakeling, 2009). Furthermore, muscle fatigue results in changes to both the amplitude/intensity and frequency contents of an EMG signal. Thus, to describe muscle activity during alpine skiing adequately, a method covering both aspects is essential.

The analysis of the frequency content by calculating the median power frequency (Kroell et al., 2005) resulted in the loss of timing information, which is essential in alpine skiing research. Developments in wavelet analyses for EMG permit the signal intensities to be simultaneously resolved in time and frequency, with time resolutions in the order of 20 ms (von Tscharner, 2000). The resulting intensities are a close approximation of the signal power contained within a given frequency band at each time point. This presents the possibility to visualize the EMG intensity pattern of a movement in time–frequency space during each cycle of a movement as it has been previously demonstrated for running and cycling (von Tscharner, 2002; Wakeling, 2004). Due to the primarily cyclical movement patterns in alpine skiing, which is comparable to running and cycling, there appears to be potential for a more in-depth analysis of muscle activity during alpine skiing by the use of wavelets.

In a study investigating a group of 10 recreational skiers (Kroell et al., 2008, 2009), subjects had to ski 24 runs through a standardized corridor on a slope with 300 m elevation with a medium inclination (21°). On average, 11 double turns (one right plus one left turn) per run were performed. Knee angle and EMG of RF and VL of the right leg were measured during runs 1, 2, 23, and 24. Knee angle and raw EMG were used to determine the start and the end of each double turn (first turn right leg = inside leg [IL]; second turn right leg = outside leg [OL]). The EMG signal from each run was resolved with a wavelet transformation into intensities for each point in time (10 center frequencies between 19.3 and 395.4 Hz; von Tscharner, 2000). Thereafter, the mean intensity spectrum was calculated for 10 evenly spaced windows (time windows 1–10) of each double turn. For each subject, all double turns of run 1 and run 2 were used to calculate a representative turn for the START of the skiing session. The same was done with run 23 and run 24 for the END of the skiing session. To get a representative result for the group, the same was done over all subjects for START and END. With those values, 3D intensity grid-plots for the overall group were created (Figure 5.14). Those plots represent wavelet 1 to wavelet 9 on the vertical axis represented by center frequencies (19–331 Hz) and the time information by time windows (1–10) on the horizontal axis. The intensity is represented by the shading, where bright means high intensity and dark denotes low intensity.

Differences in timing, intensity, and frequency content of RF and VL can be observed in the 3D plots (Figure 5.14). VL is active throughout the double turn with maximum intensities during time window 7–8 where the measured right leg is the OL. In contrast, RF shows a biphasic activity with two clear maxima on IL (TW3) and OL (TW8) and low activity during edge changing from IL to OL (TW5–6). Due to the fact that current skiing technique has changed to a more bilateral style (Mueller & Schwameder, 2003), one could assume that there would be higher activity during IL. Although the skiers performed a modern recreational carving technique, the predominant unilateral use (OL) of the one-joint knee extensor VL seems to be given.

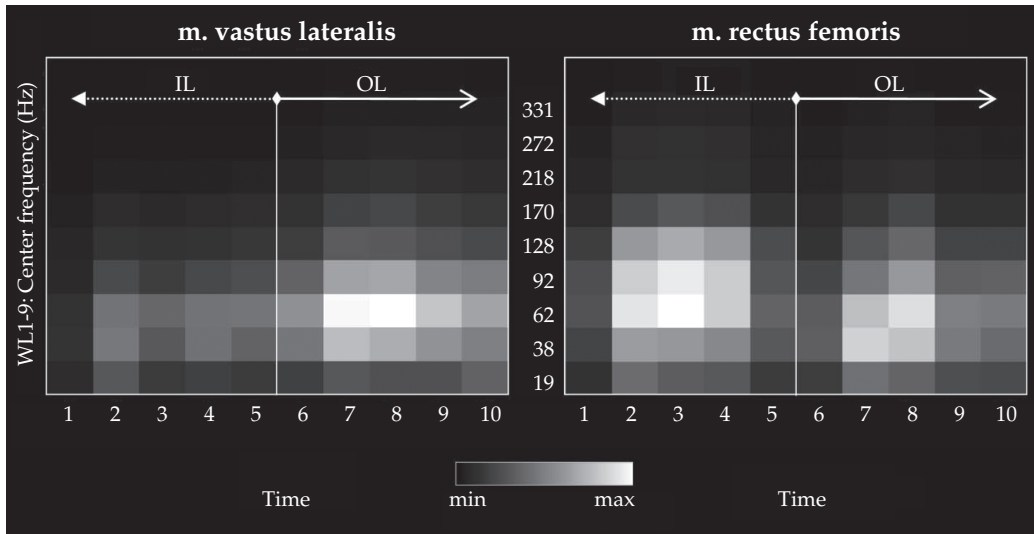


Figure 5.14 Intensity plot over time and frequency of vastus lateralis (VL) and rectus femoris (RF) for the overall group. IL, inside leg; OL, outside leg; WL, wavelet.

The contrasting biphasic patterns of RF suggest that this two-joint muscle works not only as knee extensor during OL, but also as a hip flexor and/or knee extensor during IL. We assume that there is currently a situation-dependent loading (RF as knee extensor) and unloading (RF as hip flexor) necessary which is reflected in the EMG activity.

According to the frequency content, one can observe on VL the greater involvement of higher frequencies during OL compared to IL. In contrast, the results of RF clearly indicate a more pronounced involvement of higher frequencies on IL compared to OL. Doud and Walsh (1995) reported almost a 1:1 ratio of muscle fiber length changes to change in frequency (20% increase in length/18% decrease in EMG frequency). It is very unlikely that the differences in frequencies between IL and OL are caused by changes of the fiber length. Especially, the muscle fiber length of the biarticular RF remains in a rather constant length during alpine skiing, due to the closely related movement of the hip and knee angle (Berg et al., 1995, minimum and maximum angle coincided). Hence, the fiber strains are too small to explain the changes in frequency and therefore the most likely explanation for the differences in frequency content are

from changes in recruitment. Those changes in recruitment of RF may provide additional information on the functional characteristics of the muscle during skiing. It is possible that RF recruits more fast fibers during IL (higher frequencies) compared to more slow fibers during OL. With the already mentioned functional skiing technique aspects, this would mean that RF recruitment, and specifically the fast components, is an important steering factor during IL. The ability to recruit fast RF fibers over a substantial time (hundreds of IL turns during a day) seems to be a crucial aspect in alpine skiing. The appropriate motor unit recruitment maybe necessary to obtain a good bilaterally loaded skiing technique through knee extension of the IL, or to reduce the loading of the IL through hip flexion. A potential consequence of high loading of the IL is a fall due to over edging.

Figure 5.15 represents the changes in muscle activity of the biarticular RF from the beginning to the end of a 3h skiing session. One can observe very specific changes in terms of timing, intensity, and frequency caused by the 3h skiing intervention. There is a slight increase in muscle activity (Figure 5.15 [+]) at the last part of IL (TW 4 and 5) and a clear increase at the last part of OL (TW 9 and 10).

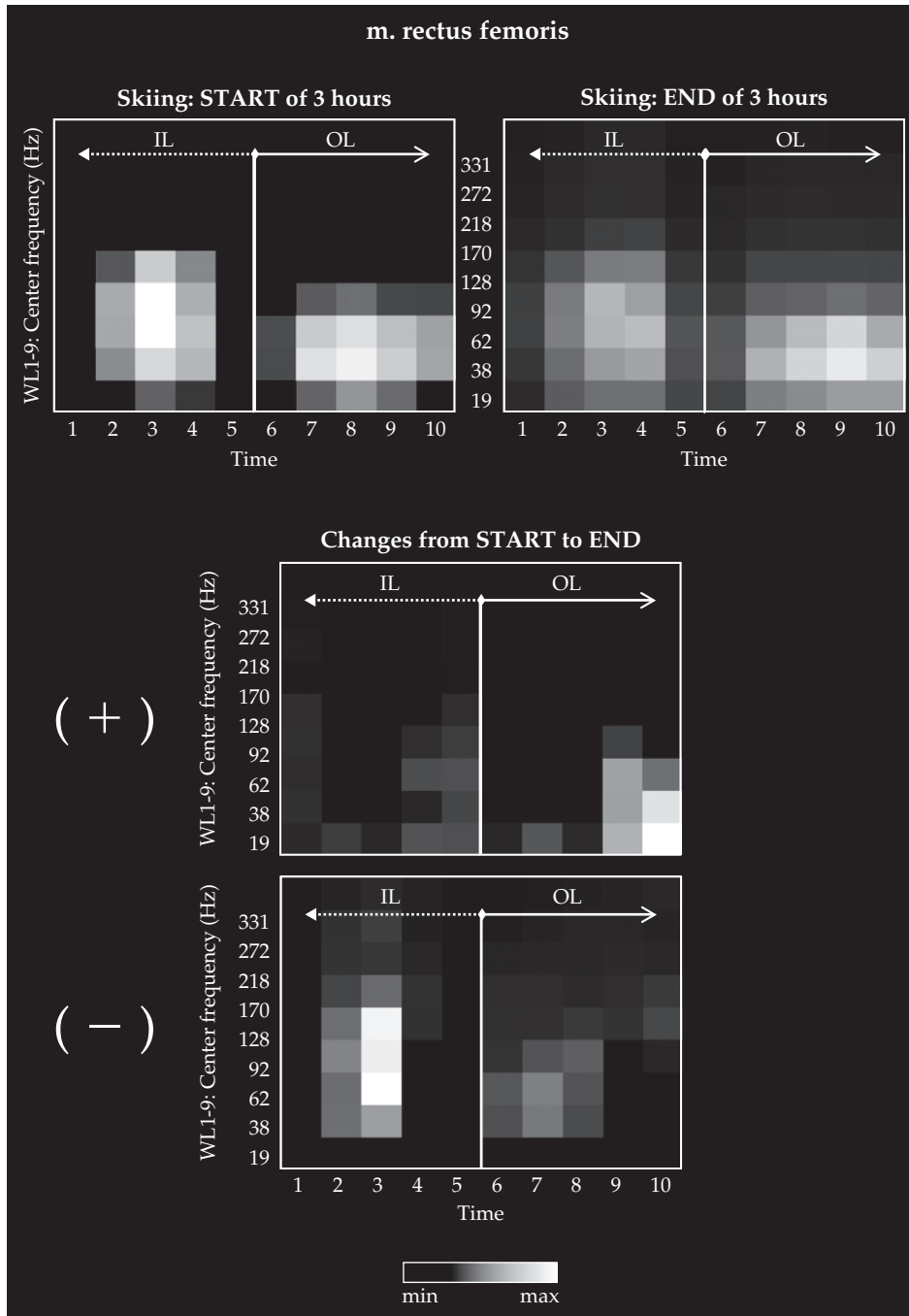


Figure 5.15 Changes of muscle activity during a 3h skiing session. (a) Intensity plot over time and frequency of rectus femoris (RF) for the overall group at START and END of the skiing session. (b) Plots of differences representing [+] as increasing intensities and [-] as decreasing intensities. IL, inside leg; OL, outside leg; WL, wavelet.

On the other hand, a decrease (Figure 5.15 [–]) during the earlier IL (TW 2 and 3) and OL (TW 6, 7, and 8) phases can be observed. According to the frequency content of the signal, one can observe that higher frequency components are decreased during IL (TW 2 and 3, Figure 5.15 [–]) and lower components are increased during OL (TW 9 and 10, Figure 5.15 [+]). Due to very similar knee angles at the START and END of the skiing session, the changes in the frequency content are again more recruitment related than muscle length. There seems to occur fatigue-related recruitment changes which are detrimental regarding the above-described importance of RF (specifically the fast components) as steering factor on IL. The reduction in high-frequency EMG may indicate that the proposed steering role of RF to IL is compromised at the end of a prolonged ski session. We suggest that OL therefore has to compensate somehow the limited function of IL. The result is a more pronounced activity at the last

part of the turn instead of a homogenous activity throughout the turn at the beginning of the skiing session. From a functional perspective, this could mean a potential risk of falls due to higher dynamic components at the end of the turn which increases the efforts for skiers to initialize the next turn.

Using the wavelet analysis gives the potential to extract conventional intensity and timing information as well as additional information about the frequency content of the signal. Due to this possibility, the principal differences of the two-joint RF and the one-joint VL were described with greater depth and detail than by the common methods used by Berg in 1995. Consequently, the results should be considered in the physical preparation of alpine skiers (high demand on the biarticular RF) and teaching/guiding concepts (ski instructors) of recreational skiers to reach improved IL steering by adequate loading in general, as well with increasing fatigue during a skiing day.

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Chapter 6

Kinetics and Muscular Function in Ski Jumping

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Introduction

Ski jumping is an exciting and primarily a competitive sport involving both ballistic and aerodynamic factors. The ballistic factors include release velocity and release position from the take-off table, whereas aerodynamic factors influence the gliding properties of the jumper/skis system (velocity, surface area, posture of the jumper/skis system, suit design, turbulence, and resisting and lifting forces) during the in-run, take-off, flight, and landing phases of the jump. The role of both ballistic and aerodynamic factors has undergone some changes during past years mainly due to changes in jumping hill profiles and other regulations concerning equipment and anthropometric characteristics of jumpers' body type. These changes will be discussed in more detail later in this chapter. Table 6.1 shows the importance of some selected factors to the length of the jump in ski jumping and it can be clearly seen that the in-run speed is the strongest factor influencing the jumping distance (see also Virmavirta et al., 2009). The table also shows that ski jumping performance is highly dependent on the aerodynamic forces acting on the jumper during the flight phase. Since the aerodynamic forces are proportional to the square of jumper's speed, the effect of these two factors is strongly linked together. However, the takeoff is still considered

to be the most important phase in ski jumping as it determines the initial conditions for the subsequent flight.

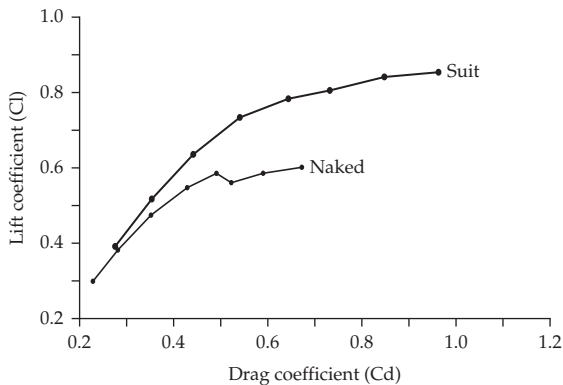
The overall development of ski jumping performance during past years has made FIS (Fédération Internationale de Ski) to modify the equipment regulations in order to control the progressive increase in jumping distances. After the dramatic effect of size of the modern jumping suit on the jump length was fully realized (Figure 6.1, Virmavirta & Kivekäs, 2009), many changes have been done to reduce its aerodynamic effect. However, the current regulations on suit design reduce utilization of this particular aerodynamic effect. Probably, the most important change was done in 2004 when the maximum ski length that a jumper is allowed to use in competition was related to the body mass index (BMI). A current rule states that the maximum ski length, 145% of the jumper's body height, is only allowed if the jumper's BMI is 20.5 or more. The rule was introduced to make the low body weight less important for successful ski jumping performance. The effect of body mass on jumping distance is well known (see Table 6.1 and Figure 6.2a) and the BMI rule was assumed to stop the tendency among ski jumpers toward extremely low body weight (Schmölzer & Müller, 2002; Müller et al., 2006; Müller, 2009). The possible effect of BMI rule on the body type of ski jumpers and furthermore to the final jump distance is well demonstrated in Figure 6.2b where the relationship between body mass and jumping distance of best jumpers in the Olympic ski jumping competitions is presented.

Table 6.1 The effect of different parameters on jumping distance as based on computer simulation

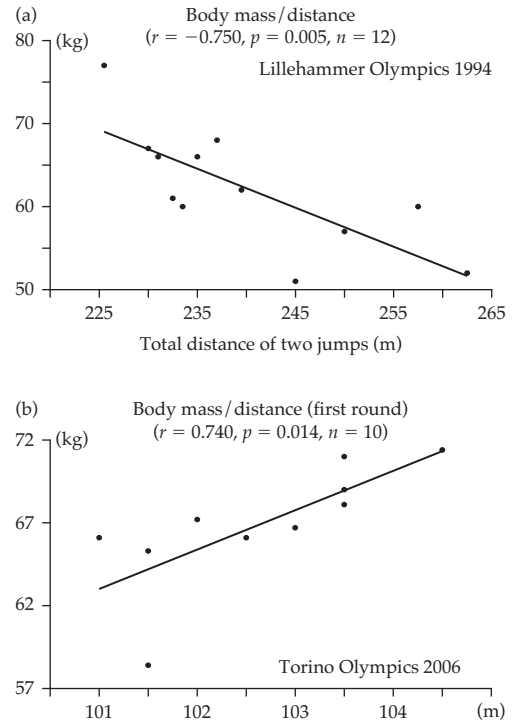
Parameter	Change	Distance (m)	Difference (m)
Reference jump		130.0	
In-run speed	+1%	134.5	4.5
Mass	-1 kg	132.1	2.1
C_l Flight	+1%	131.7	1.7
C_d Flight	-1%	130.8	0.8
Take-off force _{ave}	+1%	130.5	0.5
Friction	-1%	130.3	0.3

(J. Kivekäs, Whistler HS-140m jumping hill, Aquila Ski Jumping Simulator.)

C_l , lift coefficient; C_d , drag coefficient.

**Figure 6.1** The effect of jumping suit on the aerodynamic forces. The measurements were performed with the “doll” model without (naked) and with the jumping suit.

The above-mentioned changes added by the changes in jumping hill profiles certainly places special demands on the competition organizers when attempting to guarantee equal conditions for all jumpers with different performance levels even in the Olympic level (Virmavirta et al., 2005, 2009). Reducing aerodynamic effects is often compensated by increased in-run speed which inevitably results in some problems that ski jumping has faced in previous years when the best jumpers accidentally fly too far. Despite apparent easiness, the modern ski jumping is consequently a very complex sport whose comprehensive understanding requires knowledge of several factors related to

**Figure 6.2** Correlation between body mass and jumping distance of the best jumpers in the 1994 (a) and 2006 (b) Winter Olympics.

jumping distance, including application of computer simulation.

This chapter updates the information presented by Komi and Virmavirta (2000) on the same topic and a special effort will be made to characterize kinematics and kinetics of the take-off performance. The presentation is based primarily on the published and unpublished material obtained in the various ski jumping research projects of the Neuromuscular Research Center, University of Jyväskylä. For a comprehensive review of biomechanical research in ski jumping, the reader is referred to the recent paper of Schwameder (2008) including more than 100 references.

Kinematic Characteristics of the Current Take-off Technique

Takeoff is the most crucial phase for the entire ski jumping performance since it determines the

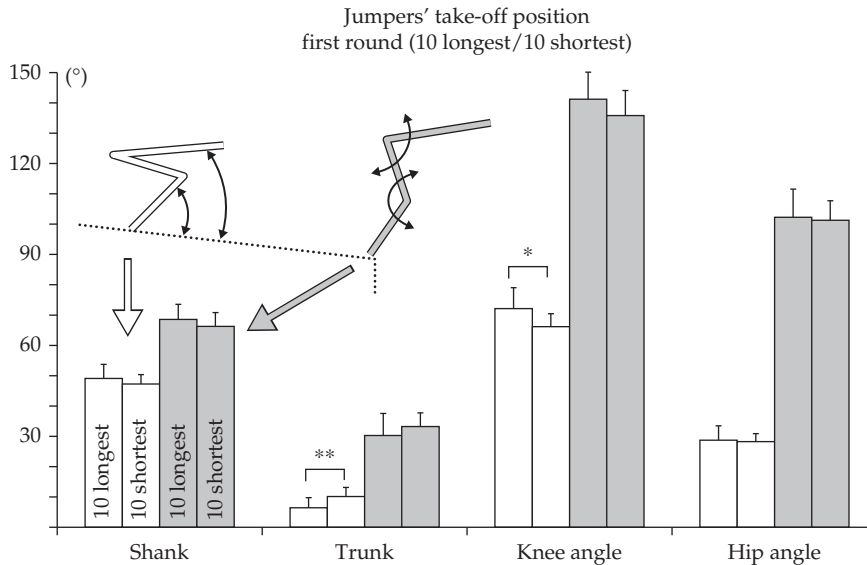


Figure 6.3 Comparison of the take-off position between the best (10 longest) and worst (10 shortest) jumpers in the Olympic ski jumping competition 2006. (From Virmavirta et al., 2009.)

initial conditions (velocity, angle of takeoff, angular momentum, and position of the jumper/ski system) for the subsequent flight. The purpose of the successful takeoff is to gain vertical velocity along with maintenance or even increase of the horizontal release velocity. Ski jumping takeoff is performed during a short time, ranging from 0.25 to 0.35 s and covering approximately 6–7.5 m from the take-off table. Thus, the first take-off movements are initiated during the transition phase from the end of the in-run curve to the flat table. This phase requires a good timing and coordination due to sudden disappearance of the centrifugal force at the end of the in-run curve (~6.5 m before the release). Understanding the importance of the take-off phase in ski jumping performance has tended to follow developments in flight phase aerodynamics (jumping suits, flight style, body weight, etc.). Improvement in ski jumping aerodynamics has increased the jumping distance so much that the precise effects of various take-off parameters on jumping distance may have been masked by other interacting factors (e.g., lift and drag forces).

The take-off analysis of the 2006 Olympic ski jumping competition (Virmavirta et al., 2009) describes well the kinematic characteristics of the current take-off technique used by ski jumpers. The rapid take-off movement can be characterized

by changes in two major angles: knee and hip. The knee angle displacement is, on average, from 70° to 140° (Arndt et al., 1995; Schwameder & Müller, 1995; Virmavirta et al., 2009) and it is not completed while on the take-off table. The knee extension velocity reaches a very high maximum value of over 12 rad/s (Virmavirta & Komi, 1993; Virmavirta et al., 2009), which is usually reached at the release instant from the take-off table. In a successful take-off, the hip extension velocity is also relatively high (>10 rad/s), which is caused mainly by the thigh movement but with a smaller upper body extension (Arndt et al., 1995; Virmavirta & Komi, 1994). The angular velocity of the hip joint of the best jumpers in the Olympic ski jumping competition 2006 correlated with the jumping distance ($r = 0.651$, $P < 0.05$, $n = 10$). Even though the differences between take-off positions of good and poor jumpers were small (Figure 6.3), the best jumpers exhibit the large variety of techniques and still achieved approximately the same distance (Figure 6.4).

Upper body movement is considered to be critical in the successful ski jumping takeoff because rapid hip extension is thought to cause high air resistance due to the upright position of the upper body. Ski jumpers can achieve a proper flight position only by producing sufficient forward-rotating angular momentum about the CoM during the takeoff. This

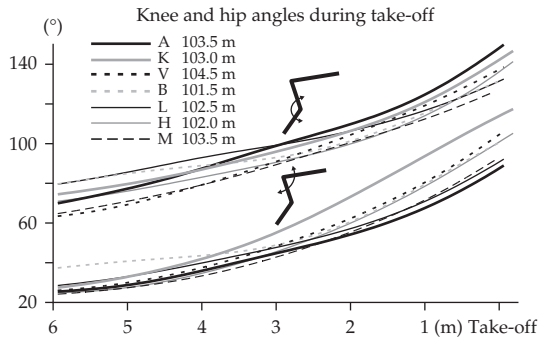


Figure 6.4 Knee and hip angles of the selected good jumpers during the takeoff in the Olympic ski jumping competition 2006.

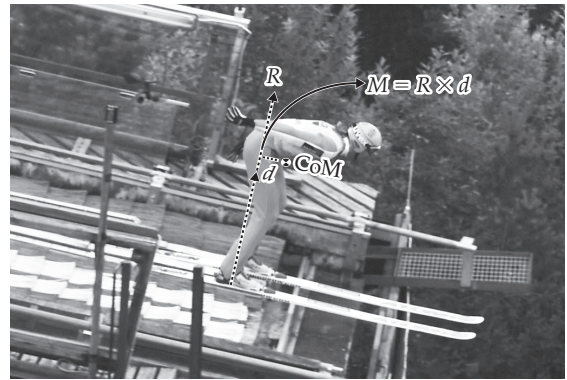


Figure 6.5 Prerequisites for generation of angular momentum about the CoM during the takeoff.

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Figure 6.6 Schematic illustration of angular momenta of a ski jumper during takeoff and flight. (Modified from Schwameder, 2009.)

is possible if the GRF (take-off force) vector passes behind the CoM and creates a needed moment arm (see Figure 6.5). The average forward-rotating moment has been found to be $66 \pm 10 \text{ Nm}$ and the final angular momentum at the release instant is $19 \pm 3 \text{ Nms}$ (Schwameder & Müller, 1995). The opposite backward-rotating angular momentum is produced by aerodynamic drag force acting on the jumper/skis system and it is highly dependent on the jumper's upper body movement. However, it seems that the upper body movement does not necessarily result in shorter jumps if the jumpers are able to create sufficient forward angular momentum during the takeoff and therefore to prevent the exposure time to high air resistance from being

too long. The good balance between these opposite moments of force is a major challenge for the jumper's successful performance (Figure 6.6).

In Figure 6.7 two different take-off techniques, both providing a good result, are presented. The behavior of the shank and upper body movements during the takeoff showed very different patterns between these jumpers (change in shank angle: 28° vs. 14° and upper body: 7° vs. 28° for jumpers A and K, respectively). It is known that by keeping the shank angle the same, especially during the early stages of takeoff (jumper K in Figure 6.7), the jumpers are better able to produce force against the surface and move their upper body forward (Virmavirta & Komi, 1994). The shank

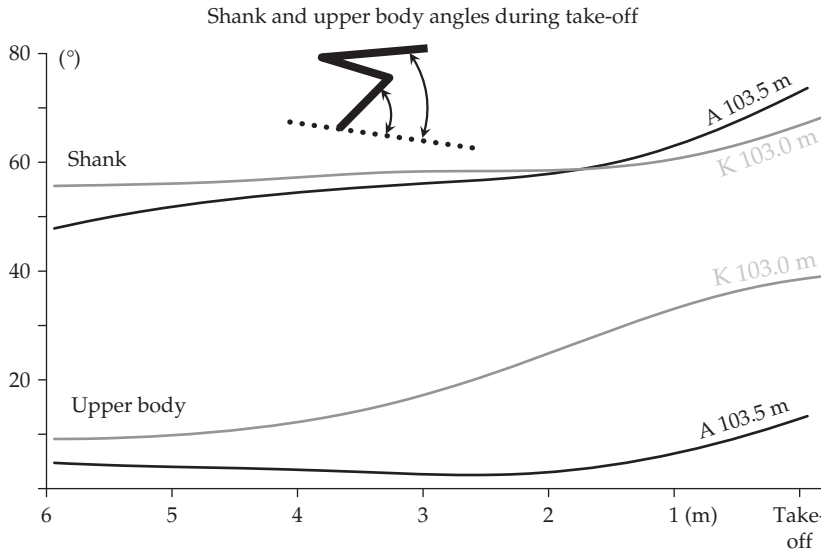


Figure 6.7 Comparison of the shank and upper body angles of two jumpers (A and K) in the same round in the 2006 Olympic competition at HS-106 m hill. (From Virmavirta et al., 2009.)

segment serves as a support for the thigh segment to rotate forward around the knee joint. However, excessive forward motion of the shoulders (relative to the knee/ankle) is very often compensated by the increase of the upper body angle relative to the direction of motion (more upright position), which tends to produce the necessary backward-rotating angular momentum. When the upper body position remains the same, as occurs for jumper A in Figure 6.7, the shank angle must increase in order to balance the forward motion during the rapid knee extension. This is a very crucial phase in the ski jumping takeoff when the take-off forces should be produced against the surface.

Optimal Aerodynamic Position After the Takeoff

Since the first comprehensive studies of Straumann (1927, 1955), considerable scientific effort has been devoted to studying optimal aerodynamic position of a ski jumper during the flight phase. Straumann's (1955) most favorable flight position angles measured in the wind tunnel showed values which are surprisingly close to the positions used by ski jumpers today (Figure 6.8). Figure 6.8 demonstrates flight positions of the best jumpers during the different flight phases in the Ski-Flying World

Championships of 1994 and Olympic Winter Games 2002 (Müller et al., 1996; Schmölzer & Müller, 2005). More detailed analysis of the early flight phase in the 2002 Olympic ski jumping competition (Figure 6.9) shows that jumpers' steady flight position is almost completed within 0.5 s (Virmavirta et al., 2005). During this short period of the flight phase (~15 m), also called transition phase, jumpers are most likely not able to do any intentional adjustments and therefore a close causal relationship between the take-off and the transition phase determines jumpers' steady flight position. Transition phase involves an important balancing of the angular momentum (nose up/down) created by jumper's take-off action and aerodynamic force (Hildebrandt et al., 2009; Schwameder, 2008; Virmavirta et al., 2009). A crucial factor of the ski jumping performance is to achieve optimal position of jumper/skis system for the steady flight phase as soon as possible. Mistakes during takeoff cannot be corrected during the flight phase, but the benefits of a successful take-off action can be destroyed by mistakes during the flight.

Kinetics of the Take-off Performance

Figure 6.10 demonstrates all the forces acting on the jumper during the inrun and takeoff as well

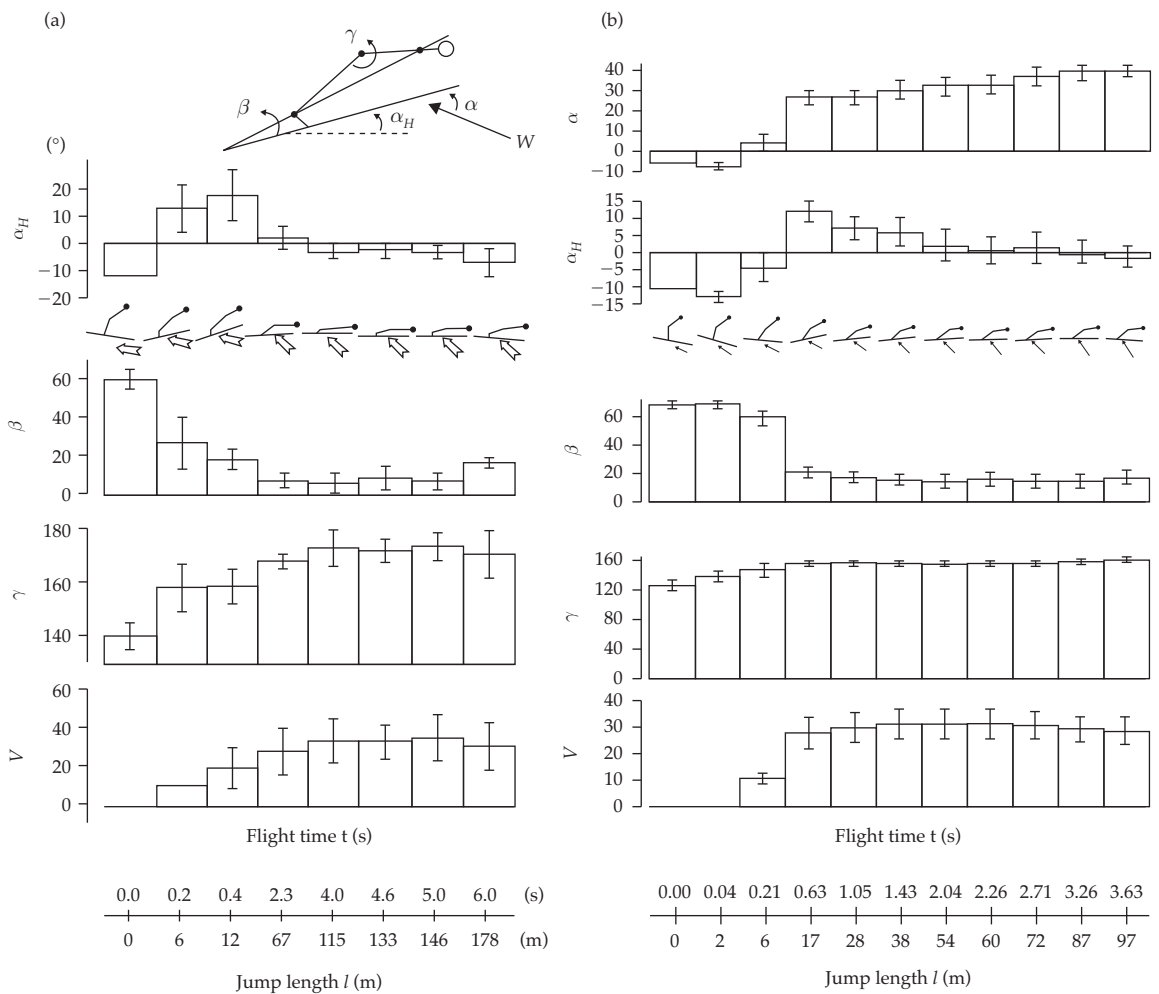


Figure 6.8 Position angles of the best jumpers in the Ski-Flying World Championships (a) and Olympic Winter Games 2002 (b). (Modified from Müller et al., 1996 and Schmölzter & Müller, 2005.)

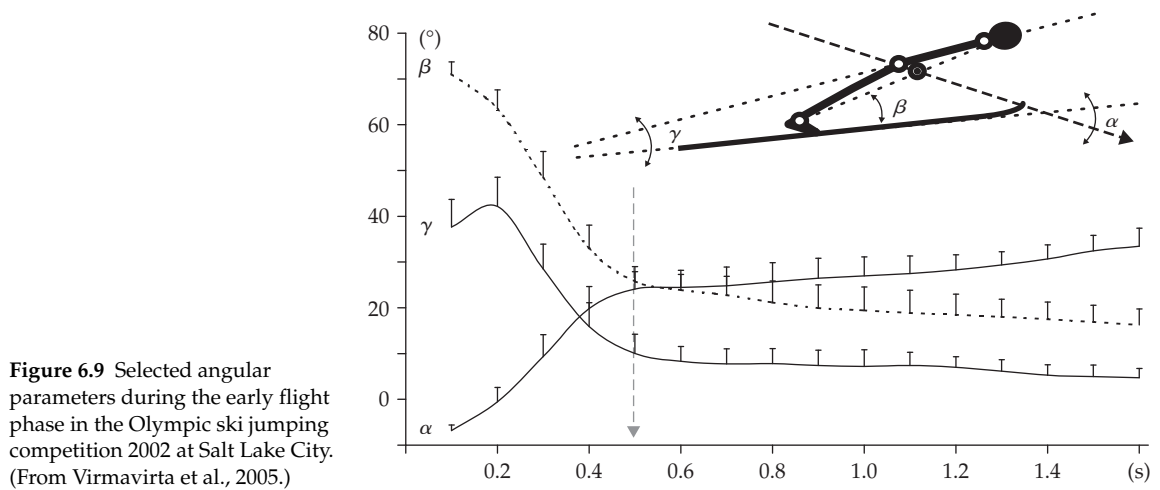


Figure 6.9 Selected angular parameters during the early flight phase in the Olympic ski jumping competition 2002 at Salt Lake City. (From Virnava et al., 2005.)

as the prerequisites for the creation of the angular momentum during the takeoff. During the straight part of the inrun, the force perpendicular to the track is equal to the normal component of the weight force and a component resulting from the centrifugal force will be added as the jumper

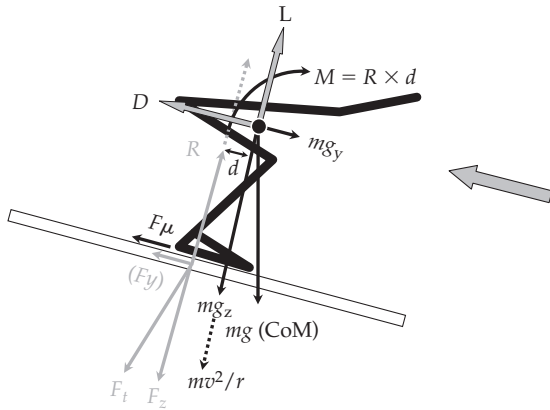


Figure 6.10 All the forces acting on ski jumper during the inrun and takeoff. CoM, center of mass.

enters the in-run curve. The extra pressure during the in-run curve is well demonstrated in Figure 6.11 where plantar pressures and EMG activities are measured from the entire ski jumping performance. The centrifugal force (mv^2/r) is typically more than 0.6G (~400–450N). This phase is very crucial for the timing and coordination of the take-off movements due to sudden disappearance of the centrifugal force at the end of the in-run curve. Therefore, the first take-off movements are initiated during the transition phase from the end of the in-run curve to the flat take-off table. The determination of the exact take-off time/distance in ski jumping can be done only by measuring take-off forces and its components with the suitable force measuring system. Figure 6.12 shows the typical force measurement of the simulated ski jumping takeoff (imitation) in the laboratory conditions and it demonstrates well the problems in the estimation of the true take-off time.

Since the number of trials in one training session performed in actual jumping hill conditions is

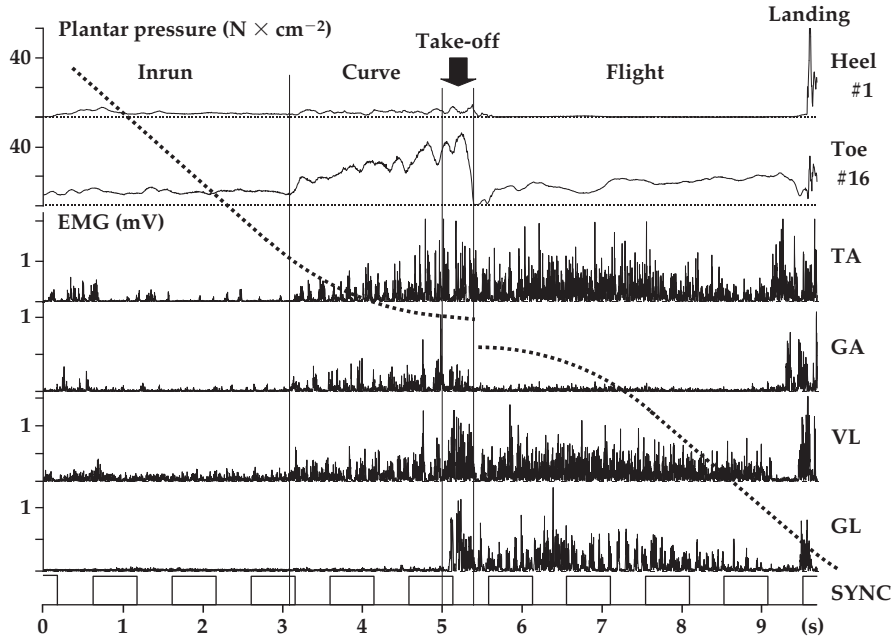


Figure 6.11 Selected pressure and EMG signals from one jump (in-run speed 23.2m/s and length of jump 93m) of an international level ski jumper. EMG, electromyography; TA, tibialis anterior; GA, gastrocnemius; VL, vastus lateralis; GL, Gluteus; SYNC, synchronizing pulse. (Unpublished data from the Neuromuscular Research Center, University of Jyväskylä.)

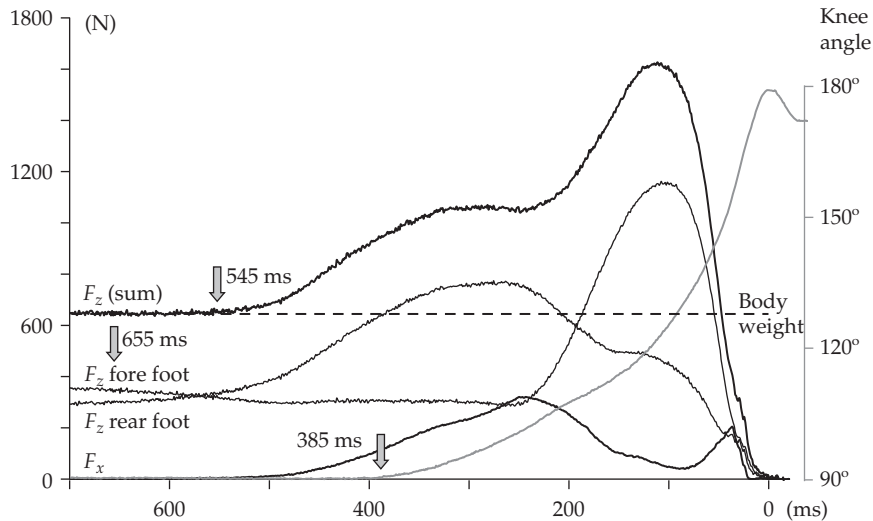


Figure 6.12 Recordings of the vertical (with its components under the fore and rear foot areas) and horizontal force as well as the knee angle of the simulated ski jumping takeoff (imitation) in the laboratory conditions. (Unpublished data from the Neuromuscular Research Center, University of Jyväskylä.)

fairly limited and does not provide much time for specific training, ski jumpers use *simulated takeoffs* (“dry land” exercises) for emphasizing their take-off training. The conditions (friction, aerodynamics, etc.) between the hill jumps and simulated jumps differ substantially and therefore it has been important to examine the differences between these two conditions (Virmavirta & Komi, 2001). The results of these studies give some insight into the comparison of the ski jumping takeoff with other jumping activities (i.e., vertical jumps). In actual ski jumping conditions, ski jumpers typically reach velocities of approximately 2–3 m/s perpendicular to the take-off table at the release instant (average 2.60 ± 0.15 m/s for 10 best jumpers in the Olympic final 2006 corresponding to the jump height of 0.35 m for the CoM in the vertical jump). Figure 6.13 shows the comparison between the vertical force and power production of the simulated ski jumping takeoff and the maximum vertical SJ of one excellent jumper with strong take-off capacity. This jumper utilized 88% of his full vertical take-off capacity (take-off velocity) in the simulated takeoff and the percentage is most likely to be less in the actual conditions (Vaverka et al., 1993). The

importance of the takeoff for the entire ski jumping performance cannot be estimated correctly, if only computer simulation is applied (Table 6.1). In the example of Table 6.1, the increase of 1% in the average take-off force of the reference jump results in only 0.5 m increase in the jumping distance. This increase of force production corresponds to less than 1 cm increase in jump height of the vertical jump. This improvement is fairly easy to achieve by training alone. However, a 5% increase in the average force results in increase of 3 m in jump length and this gain of 10 cm in vertical jump is not easy to obtain by training only. In the jumping hill profile used in the computer simulation of Table 6.1, the theoretical jump length without any vertical force production would be only 30 m. These simulations show that the take-off force component perpendicular to the tracks is important not only for the rise of the path of the CoM but also for creation of the angular momentum during the takeoff.

The creation of the *angular momentum* during the ski jumping takeoff is well discussed by Schwameder (2008, Figure 6.14). The vector analysis presented by Schwameder clearly shows that the change of location of the jumpers’ CoM relative

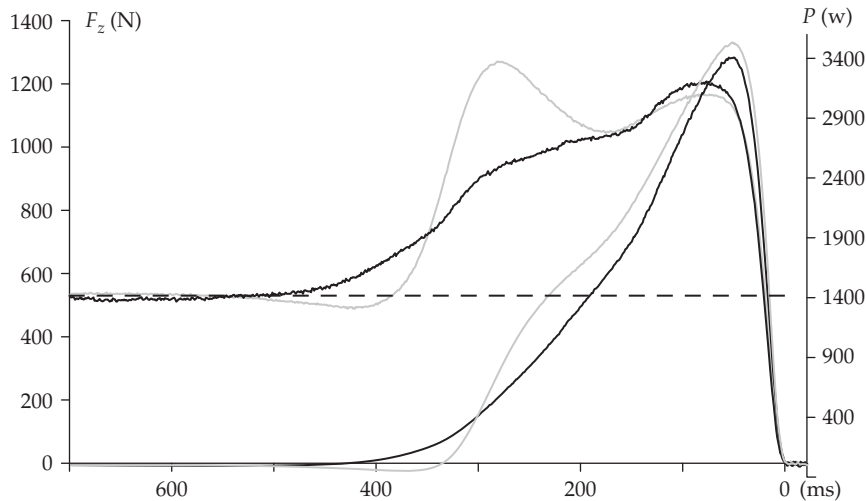


Figure 6.13 Vertical force (F_z) and power production (P) of the simulated ski jumping takeoff (black) and the maximum vertical SJ (gray) of one excellent jumper with strong take-off capacity. (Unpublished data from the Neuromuscular Research Center, University of Jyväskylä.)

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Figure 6.14 Resultant force (F) and its translational (F_t) and rotational (F_r) component during takeoff with different relative positions of the CoM. a and d are the moment arms of F_r and F , respectively. (From Schwameder, 2008).

to the resultant GRF vector has much more effect on the magnitude of rotational component (responsible for the angular momentum) than on the translational component (determines the gain in vertical velocity) during the takeoff. This interrelation is highly linked to the subsequent flight phase and places special demands on the ski jumpers' coordination skills during the takeoff. Therefore without any vertical force production a ski jumper is not able to achieve proper flight position.

The duration of the takeoff has been found to differ a lot between the actual (250–300 ms) and simulated jumps (>500 ms). According to Schwameder (2008), this difference can be explained by preparation for the takeoff in simulated jumps (~50 ms), incomplete knee extension at release instant in actual hill conditions (65–80 ms), different footwear (training shoes versus jumping boots, 20 ms), and aerodynamic lift in hill jumps (30–60 ms). The preparation for the takeoff is well demonstrated in Figures 6.12 and 6.15 as the force distribution under the feet changes and TA muscle activates. The difference in knee extension between the actual and simulated conditions (~140° vs. 180°, respectively) can be accomplished in 50 ms due to very high maximum angular velocity (~15 rad/s) measured at the knee joint in simulated jumps. The effect of aerodynamic forces on the F–T characteristics of the simulated ski jumping takeoff has been examined in a wind tunnel.

Virmavirta et al. (2001) did a series of *wind tunnel experiments* where the jumpers performed takeoffs (also with skis) in nonwind conditions and in various wind conditions and GRFs were recorded with a force plate installed under the tunnel floor. The aerodynamic quality of the jumpers' initial take-off

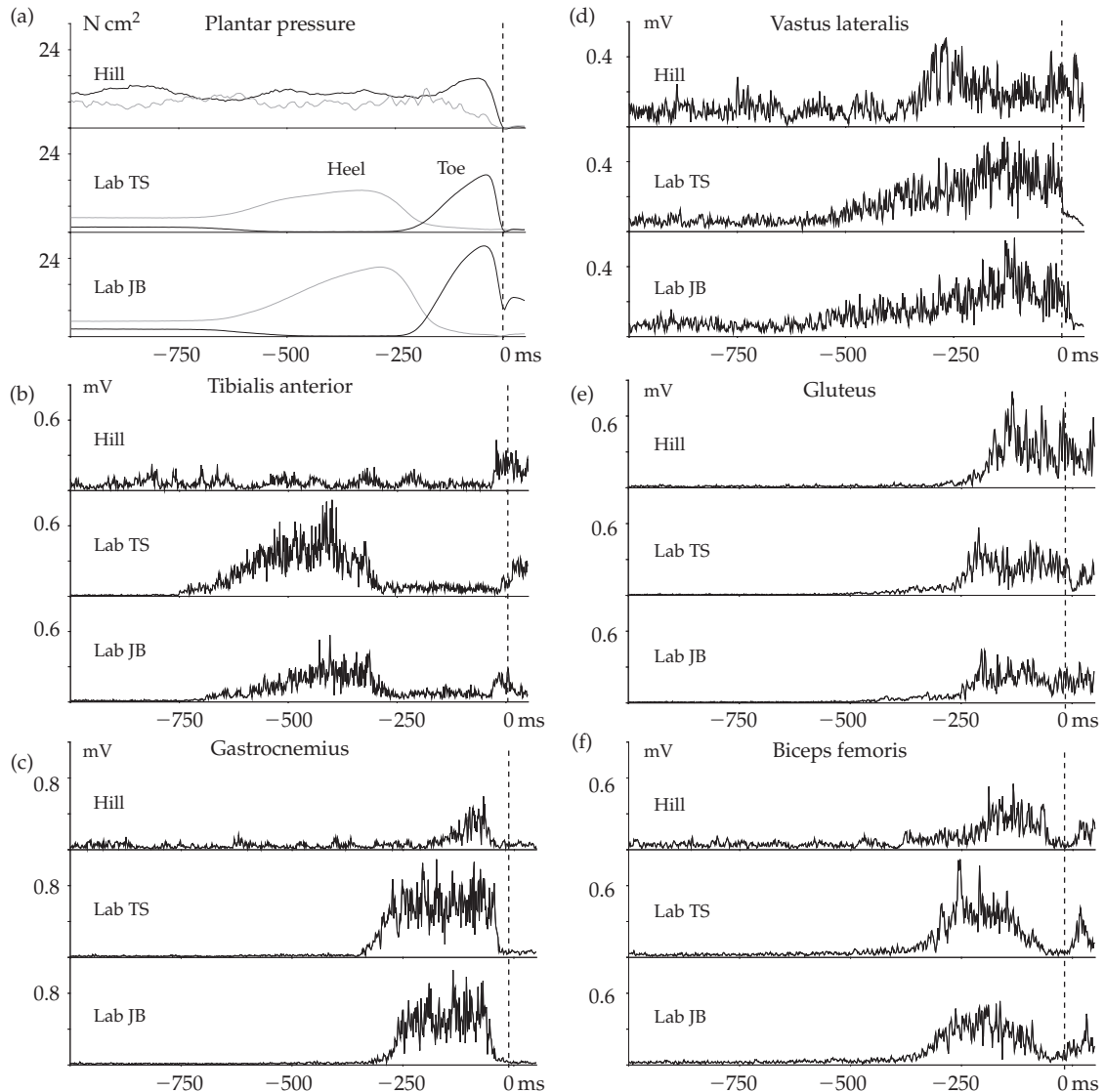


Figure 6.15 (a)–(f) Average plantar pressure and EMG activities of several trials during the takeoff of one excellent jumper in three different conditions (hill = actual jumping hill conditions, lab TS and lab JB = simulated laboratory conditions with training shoes and jumping boots, respectively). Zero in the timescale indicates the release instant from the take-off table under hill conditions and the toe-off instant under lab conditions. (Modified from Virmavirta & Komi, 2001.)

position is presented in Figure 6.16, which shows that high air resistance also creates an unfavorable large lift force before the takeoff. The lift is generated when the air goes under the upper body in an unfavorable in-run position. The jumpers' different

ability to utilize aerodynamic lift during takeoff is probably caused by the behavior of the air stream around the upper body before and during takeoff. The dramatic decrease in take-off time (Figure 6.17) found in all jumpers in wind conditions was

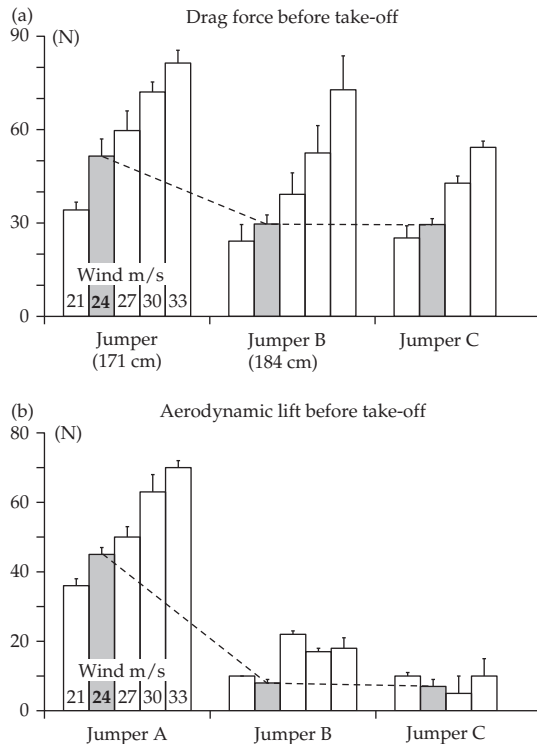


Figure 6.16 The aerodynamic drag (a) and lift (b) forces acting on three different jumpers (A, B, and C) with different wind speeds in a wind tunnel. Gray bars (broken line) indicate a typical in-run speed used in actual jumping conditions. (Unpublished data from the Neuromuscular Research Center, University of Jyväskylä.)

interpreted to be the result of the influence of aerodynamic lift force. It was concluded that the aerodynamic lift caused by wind brings the simulated ski jumping takeoff closer to field jumping conditions and helps the jumpers to perform takeoff in the limited time on the take-off table more effectively than has been assumed.

Conclusions

The material presented in this chapter has emphasized that the ski jumping performance is dependent on both aerodynamic and neuromuscular

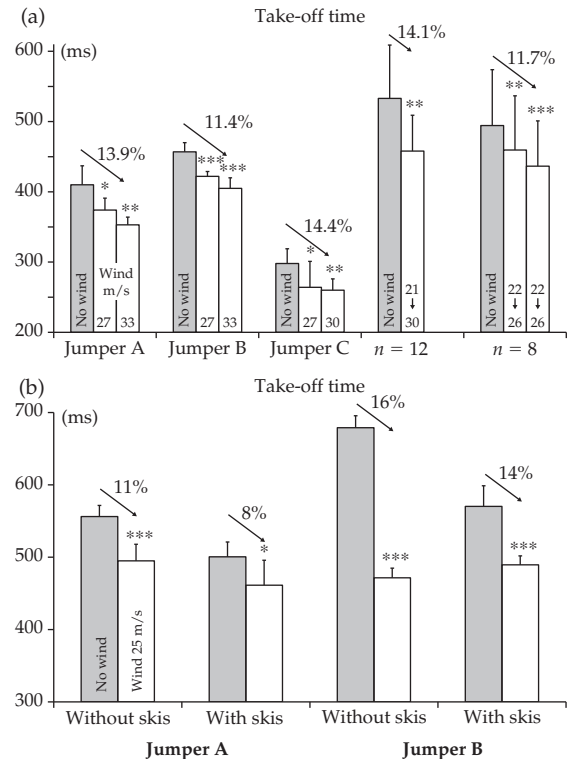


Figure 6.17 Take-off time of ski jumpers in nonwind and wind conditions without (a) and with (b) skis. The results of two groups ($n = 8$ and $n = 12$, a) shows the average values measured in different wind conditions. (Unpublished data from the Neuromuscular Research Center, University of Jyväskylä.)

factors. The presentation did not, however, consider the influence of varying wind conditions during the jump. FIS is presently planning to use compensation factors, which could then “correct” the unequal wind conditions between jumpers. The influence of the wind as such, especially its inconsistency, is not at all proven and studied experimentally. It is therefore recommended that should FIS continue to develop these compensation rules further and especially for the standard use, research is urgently needed to make the performance in this competitive sport of ski jumping evaluated objectively and fairly for every athlete.

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Chapter 7

Mechanical Efficiency of SSC Exercise

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Introduction

In most human motions, skeletal muscles act through an SSC (Norman & Komi, 1979), which has been described in detail in Chapters 1 and 2. The major conclusion from the various studies, both human and animal, is that SSC muscle action produces muscle outputs which can be very different from the conditions of isolated preparations, where activation levels are held constant and storage of strain energy is limited. Furthermore, it can be stated that, if the mechanical outputs of the muscle are enhanced in SSC action, the logical consequence should be that the work efficiency is enhanced as well.

To start the discussion on ME of SSC actions, various definitions with relevance to ME need to be addressed briefly. In principle, the question deals with the problems of efficient and economical movement, both of which have been defined in a variety of ways (Cavanagh & Kram, 1985). For example, *muscular efficiency* has been defined by Stainsby et al. (1989) as the ratio of the mechanical work to the metabolic energy expended. *Gross efficiency* and ME have been used as synonyms (Goldspink, 1977; Kaneko et al., 1984), consisting of two processes, phosphorylation coupling and contraction coupling, in converting energy from one form to another. Efficiency of phosphorylation coupling is about 60% and its calculation involves

dividing the conserved free energy of adenosinetriphosphate (ATP) by free energy of oxidized foodstuff. In contraction coupling, the energy stored in phosphates is converted into tension with an efficiency of about 49% (Whipp & Wasserman, 1969). Therefore, in isolated situation, the muscular efficiency is about 29% in maximum. Experiments, conducted primarily during the last three decades, have shed doubt on such a relatively low level of ME which varied only slightly. It is now known that the different forms of muscle action have different MEs and that the velocity of shortening and stretching influences its value (Margaria, 1968; Kaneko et al., 1984; Aura & Komi, 1986). In addition, SSC alone may introduce very different loading conditions and subsequently different ME values.

The term “effectiveness” is defined as the interaction of skilled movement and energy cost (Cavanagh & Kram, 1985). *Net efficiency*, *work efficiency*, or *apparent efficiency* mean the energy expended above that at rest, while *delta efficiency* is defined as “the average gradient of the energy expended vs. work done curves for a specified limits of the working time” (Cavanagh & Kram, 1985). The term “economy” means oxygen consumption and/or energy expenditure at a given workload. In human movement, the economy is the better the lower the submaximal oxygen uptake is (Cavanagh & Kram, 1985). Although mechanical work is missing in determination of the term economy, submaximal oxygen uptake per unit body mass required to perform a certain task is widely accepted as the physiological criterion for efficient movement.

ME of Various Human Movements

Relevant Background Studies for ME

When attempts are made to compare ME values between different isolated muscle actions, one should keep in mind the basic differences of mechanical output and the associated activation profiles. They should basically determine the difference in ME between eccentric (negative work) and concentric (positive work) actions. Firstly, as shown already in the 1920s by Levin and Wyman (1928), the maximum force produced by eccentric actions exceeds considerably that of the concentric action. While the maximum EMG activity recorded in these two action types is roughly the same (Komi, 1973), the obvious conclusion could be that for the same neural input (muscle activation), the produced forces are different between these two isolated muscle action types. This difference becomes even more obvious, when one plots the EMG levels against the force output measured at various submaximal loads. Figure 7.1a demonstrates this early finding of several authors, such as Asmussen (1953), Bigland and Lippold (1954), and Komi (1973) among others.

Similarly, it has been well demonstrated that maximal oxygen uptake increases with increasing mechanical work or power output. Figure 7.1 implies therefore that the eccentric exercise is more economical as compared to the respective concentric exercise. This may lead to two important

consequences: (1) the same absolute force can be produced with much less motor unit activation in eccentric action, and/or (2) for the same EMG level, the force output is much smaller in concentric as compared to the eccentric action. In order to predict still further how the differences in ME between these isolated action types should look, one could find the work of Bigland-Ritchie and Woods (1976) very useful. Figure 7.1b demonstrates their findings that the energy expenditure as measured by the O_2 consumption at certain submaximal power outputs is much smaller in eccentric as compared to the concentric action. These basic relationships have then led to the finding of different MEs between positive and negative work as shown in Figure 7.1c (Margaria, 1968). This observation has been regarded as a classical one and the author has suggested that while the two action types demonstrate obvious differences in ME, the values of ME are quite constant over reasonable speeds of shortening and lengthening actions, being approximately 0.2 (20%) and -1.2 (120%) for positive and negative work, respectively. We know now that his conclusion is no longer valid, although the basic difference in ME exists between the two exercise types, but the notion of constancy of ME values cannot be supported.

Jyväskylä Studies in ME

Our interest in becoming involved in exploring MEs in locomotion was also prompted by the

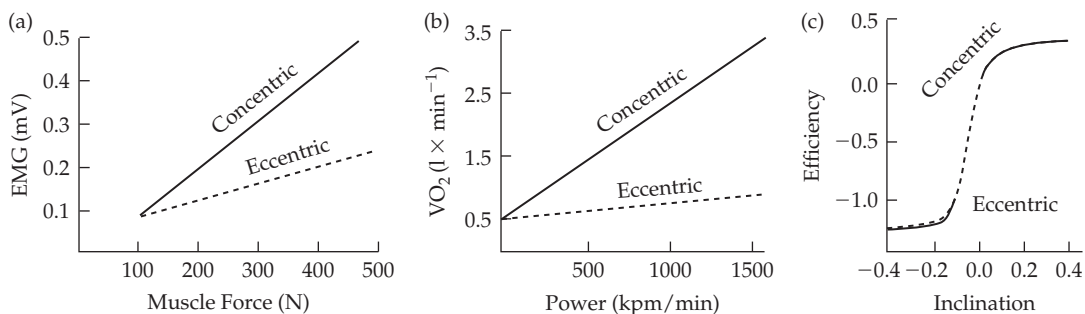


Figure 7.1 Comparison between pure concentric and eccentric muscle actions. (a) EMG–force relationship, (b) oxygen consumption (VO_2)–power relationship as measured at a selected constant speed of cycling in either the forward or reverse direction, and (c) relationship between efficiency and inclination of the ground during walking or running (Based on Komi, 1986).

basic observations in Figure 7.1a and b. As our earlier studies comparing eccentric and concentric actions either in fresh or fatigue situations involved rather small muscle groups, elbow flexors (Komi & Buskirk, 1972; Komi, 1973), we needed to construct another type of ergometer, in which larger muscle groups could be studied and different steady-state energy expenditure levels compared at various constant intensities of either concentric or eccentric actions. This then led to designing a sledge apparatus, the principle of which is shown in Figure 7.2. Accordingly, isolation of the pure negative work is accomplished by dropping the subject-sledge system from predetermined heights against the force plate. When the feet touch the force plate, the subject resists the imposed stretch in the braking phase until the predetermined knee flexion, which in most experiments was 90° . When this knee position is reached, two assistants pull the sledge-subject

system back up to the same energy level for the next negative work attempt. Constant time of 3 s was usually allowed before the next drop against the force plate. For the determination of ME, the drops were repeated usually 80 times per energy level (drop height). The expired air was collected before, during, and after the exercise period until the resting O_2 consumption was resumed. A range of dropping heights was chosen so that ME values could be obtained for different braking phase speeds as set by the dropping height.

The isolated positive work was performed so that the subject assumed the initial 90° knee flexion position on the force plate and pushed himself (and the sledge) to a predetermined energy level as set by the marker placed on the sledge rail. After reaching this level, the two assistants catch the sledge and lower it slowly to the original starting position for the next effort. Special care is taken that

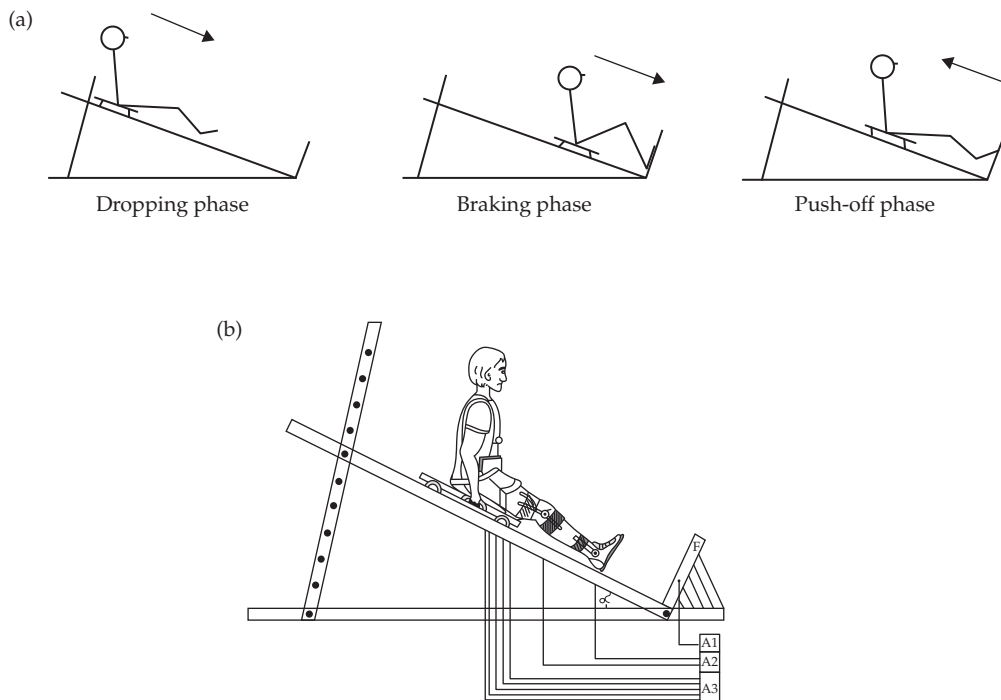


Figure 7.2 Different phases of the muscle actions (a): The braking phase refers to the eccentric action of the leg extensors, whereas the push-off phase refers to the concentric action of the same muscles. Diagram of the sledge apparatus (b): The subject was fixed with belts tightly on to the sledge. F and A1, force plate and its amplifier; A2, amplifiers of the electrical goniometers for the knee and for the ankle; A3, EMG amplifiers (Kyröläinen & Komi, 1995, modified from Kaneko et al., 1984).

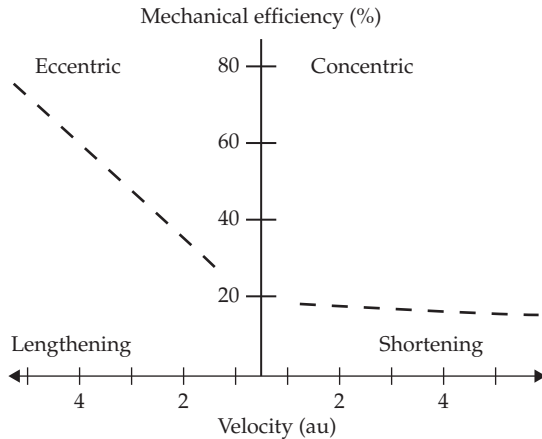


Figure 7.3 ME measured on the sledge apparatus in the isolated situations of lengthening (eccentric) or shortening (concentric) actions. The velocity axis gives the comparable lengthening and shortening speeds as obtained by varying the drop height in the eccentric mode and push-off height in the concentric mode, respectively (Based on Aura & Komi, 1988a).

no countermovement is allowed in propulsive push from the force plate. The different levels of positive work are performed by instructing the subjects to push themselves always to the set energy level.

Considering these experimental difficulties, the following major findings can be summarized (Figure 7.3). While the ME is definitely lower in the positive work (concentric exercise) as compared to negative work (eccentric exercise), no constant value could be given to either condition. In fact, for the positive work, the highest ME value of 18–19% was obtained at the lowest push-off speeds, and it was reduced linearly down to 14% at the highest concentric speeds. In the isolated negative work, when the leg extensor muscles were performing primarily eccentric exercise, ME increased as a function of increase in stretch speed from 25% (lowest stretch speed) up to 80% (highest speed). These observations and findings are expected considering the basic principle of Hill's F–V curve (Hill, 1938) as well as other background features depicted in Figure 7.1a and b.

How is ME of the positive work influenced when the preceding negative work phase is modified? In Chapter 2, it was emphasized that the mechanical

output of the push-off phase of the SSC action is primarily modified by the prestretch velocity and the coupling time between stretch and shortening. It could be logical to assume that the ME of the positive work could be influenced by these same attributes. The first attempts to answer these questions utilized Margaria's concept of the constant negative work phase ME of -1.2 in the formula to calculate the ME of the positive work. The negative work ME was not measured, but the constant value was assumed as a reliable one by many research groups. Our own studies (Ito et al., 1983; Bosco et al., 1982) were among these early attempts. We realized, however, quite soon that the use of a constant negative work ME of -1.2 was misleading. The results, shown in Figure 7.3, demonstrate this clearly. Consequently, we used the following formula to calculate the positive work phase ME in SSC (Aura & Komi, 1986):

$$\eta_+ = \frac{W_{\text{pos}}}{\Delta \text{En. exp} - \frac{W_{\text{neg}}}{\eta_-}} \times 100\%$$

where W_{pos} is the positive work of one action, W_{neg} the negative work of one action, $\Delta \text{En. exp}$ the energy expenditure above the resting level (sitting posture) of one action, and η_- the ME of the negative work, individually measured values.

In this formula, the eccentric phase was given a value obtained in an isolated situation which utilized the same braking phase speed as in the subsequent SSC condition. Figure 7.4a and b gives results of these experiments and they imply that by changing the conditions of the negative work phase, the obtained positive work efficiency is modified. More specifically, the following two findings are of importance: (1) with an increase in the prestretch velocity, ME of the constant positive work phase increases (Aura & Komi, 1986), and (2) this increase in ME of the positive work phase is a function between stretch and shortening so that the shorter coupling time results in enhanced ME in the positive work phase (Aura & Komi, 1986).

This method to measure ME of the positive work of SSC is quite time consuming, as it requires two

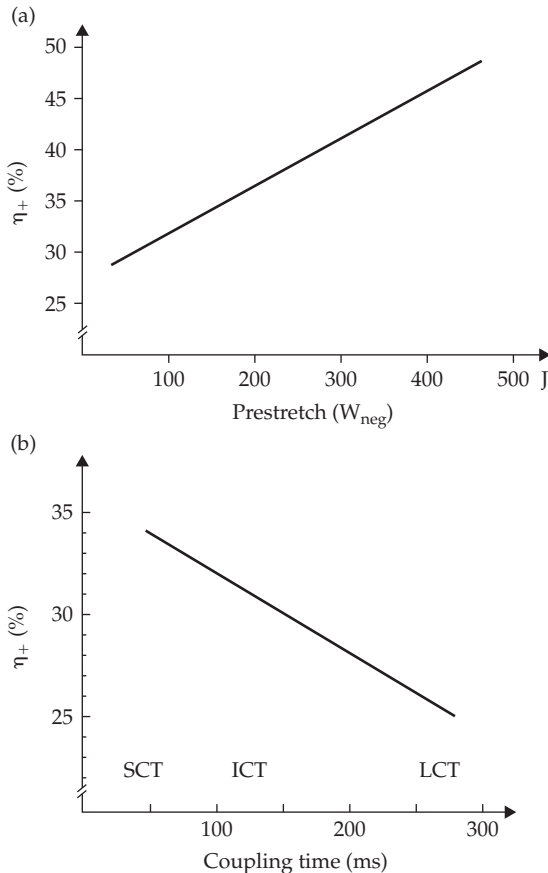


Figure 7.4 (a) Relationship between ME of the positive work (η_+) and prestretch intensity (W_{neg}) in SSC exercise (Based on Aura & Komi, 1986). (b) Relationship between η_+ and coupling time (Based on Aura & Komi, 1988b).

sets of measurements per condition; the ME of the eccentric phase has to be measured first with the various stretching velocities (drop heights) of the braking phase. The second part then utilizes these drop heights for various SSC conditions, in which the mechanical output (push-off height) is kept the same.

Margaria's (1968) approach has been criticized for two reasons: first that it gives unrealistic ME value of -1.2 for the negative work, and secondly that negative work efficiency is constant. Although our approach with the sledge apparatus sounds attractive, the procedure that we have adopted is still not completely accurate for giving exact ME for the

negative phase of SSC. The reason is simple: in the isolated eccentric exercise, even when the stretch is controlled while dropping the subject from the predetermined height, the activity profiles of these isolated eccentric actions differ from the one produced in the braking phase in the SSC. In the isolated eccentric actions with the sledge, the subject is instructed to stop the movement. The braking phase is then like in the landing type of performance. In the combined SSC action, the braking phase action is followed by immediate transition to the subsequent concentric (push-off) phase as shown in Figure 7.5. In the isolated eccentric exercises, the stretching velocity is not constant throughout the stretch. Consequently, the activation profiles of this isolated eccentric action may not be the same as the one produced during the SSC. Despite the same intention, the purpose of the SSC is to combine the stretching (eccentric) and shortening (concentric) parts with the smooth transition. Consequently, the EMG profiles show clear differences in two conditions as shown in Figure 7.5 (Aura & Komi, 1987). Thus, if the calculation of positive work of ME in SSC exercise is based on the assumption that the eccentric phase of SSC is the same as in the pure eccentric condition, the obtained values are misleading. The EMG analysis demonstrates this very clearly (Kyröläinen et al., 1990). In fact, in the isolated condition, the EMGs were much lower than in a comparable eccentric phase of SSC exercise. Thus, despite the apparently same mechanical work, the energy expenditures must have been different. Higher EMG values would imply higher energy expenditure when two types of eccentric phases, which have the same mechanical work, are compared with each other. This naturally introduces errors in the final calculations of ME in the positive work phase in SSC and the final result will probably be an overestimation (Kyröläinen et al., 1990).

ME of the Total SSC Exercise

Avoiding the difficulties in assigning correct ME values for the negative work phase part of the SSC exercise, the measurements of the ME during the entire SSC exercise can be obtained more realistically. Kyröläinen et al. (1990) measured the total

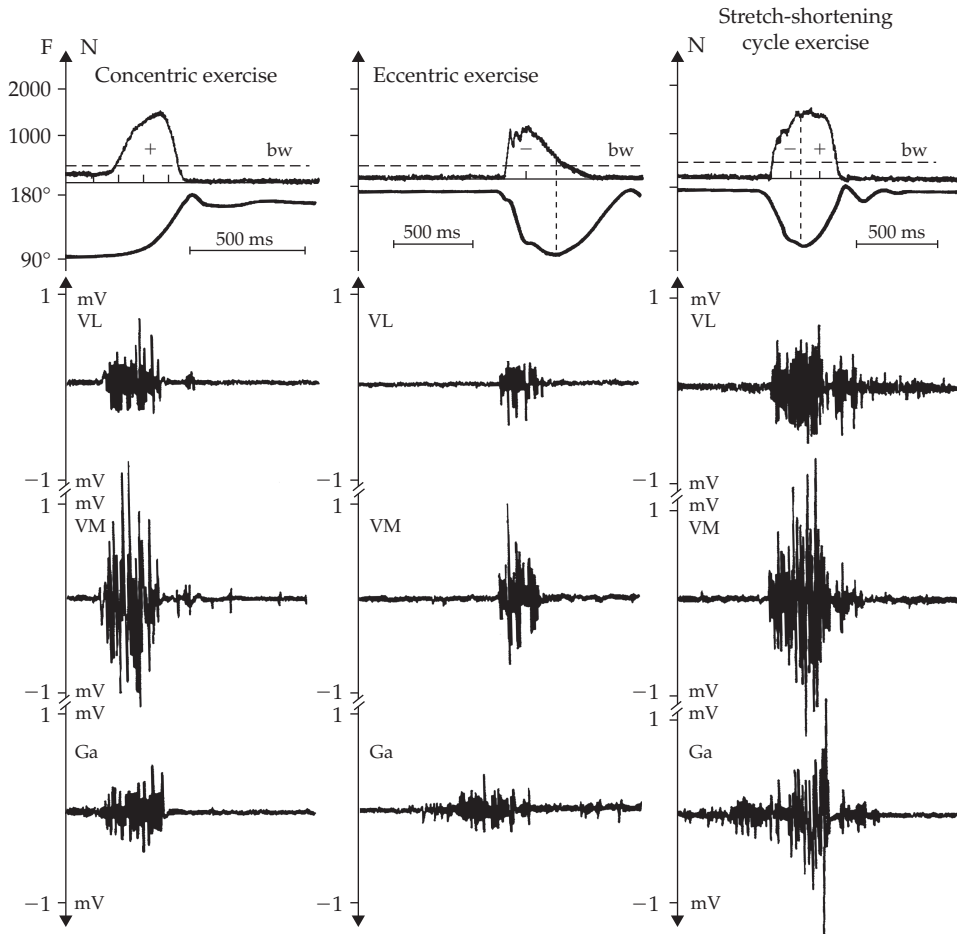


Figure 7.5 Ground reaction force (GRF), knee angle, raw EMG signals from the vastus lateralis (VL), vastus medialis (VM), and gastrocnemius (Ga) muscles recorded during the concentric, eccentric, and SSC exercises EMG, electromyography. (Modified from Aura & Komi, 1987).

ME in SSC by varying both prestretch intensity (dropping height) and subsequent positive work phase intensity.

During SSC exercise, the definition of ME follows the traditional computation in which the determinants of ME, external work, and energy expenditure must be estimated as accurately as possible. The methods employed have varied slightly depending on the exercise type and availability of equipment. It is often difficult to estimate the ME of human motion because of the uncertainties involved in defining and computing the external work.

Stretch-shortening cycle performance (sledge jump) starts when the assistants drop the subject from a predetermined dropping height causing differences in the stretch velocity of the leg extensor muscles. The initial braking (eccentric) phase follows immediately the push-off (concentric) phase of a predetermined intensity. The assistants select the required dropping height, and inform the subject of the height to which the sledge has been risen to. A force plate placed perpendicularly to the sliding surface is used to measure the reaction forces as well as the beginning and the end of the contact. The distance the sledge moved and its velocity are

measured by an optical encoder. Thereafter, the external work is calculated by the integral of the function $F(x)$ between the limit p_1 and p_2 as follows:

$$W_{\text{tot}} = \int_{p_1}^{p_2} F(x) dx$$

where F is the reaction force, x the displacement of the sledge, p_1 the beginning of the contact, and p_2 the end of the contact with the force plate (Kyröläinen & Komi, 1995).

The results were very conclusive: the different combinations of the SSC exercise produced higher total MEs as compared to pure concentric exercises. For example, the combination of low eccentric intensity (dropping height 20 cm) and high concentric intensity (90% from the maximum) produced total ME of 29.1%, while the respective ME value was 40.1% during the combination of high eccentric intensity (80 cm) followed by concentric action with the intensity of 60% from the maximum.

The EMG activities when measured from the VL, VM, and SOL muscles were potentiated in the SSC exercises during the eccentric phase of the take-off. These findings demonstrate how important role the stretch phases play as an increasing factor to potentiate performance, and consequently to increase ME. Chapter 2 discusses some of these underlying factors. Among them the reflex contribution combined with the elastic potentiation maybe involved. Thus, it seems quite clear that ME of the total SSC (e.g., hopping exercise) increases as a function of increase in the stretch velocity of the braking phase. The increase seems to be similar among power- and endurance-type athletes (Figure 7.6) and the ME values as high as 60% may not be uncommon. It is also noteworthy that in the cycling exercise, ME can exceed that of the isolated positive work, the value of which is less than 20% measured on the sledge (see Figure 7.3). This is most probably due to the finding of a small but important component of SSC in bicycling (Gregor et al., 1991 and Chapter 4) which would then increase ME in bicycling above 20%.

As mentioned earlier, in SSC exercises, such as jumping, the fast motor control of human movement

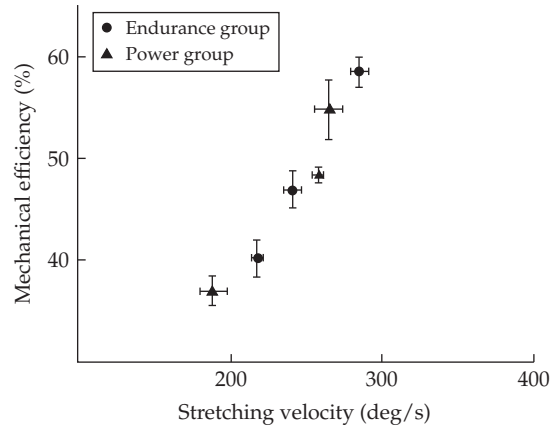


Figure 7.6 ME of the total SSC exercise increases as a function of increase in the braking phase (stretching) speed similarly among endurance and power-trained subjects. The stretching velocity was determined indirectly by the angular velocity of the knee joint during the sledge jumps (Kyröläinen & Komi, 1995).

is essential for powerful performance. Before the ground contact, the extensor muscles are activated under the influence of a central motor program (Melvill-Jones & Watt, 1971). The preactivity appears to be a preparatory requirement, both for the enhancement of EMG activity during the eccentric phase of the takeoff and for the timing of muscular action with respect to the ground contact (Moritani et al., 1991; Kyröläinen et al., 2005). The increase of preactivity with increasing running speed can be clearly seen in Figure 7.7. The result is in agreement with the similar finding observed for the GAST muscle (Komi et al., 1987).

Although the sledge jumps are after all quite convenient to use for measuring ME in total SSC, there maybe needs to study exercises that have more powerful braking phases. DJs from different heights are useful in this regard. These exercises were introduced in Chapter 2, but the original instructions to perform the jumps from different heights and their relevance to rebound (push-off) performance can be obtained from Komi and Bosco (1978). In classical DJ, the instruction is always given to rebound (take off) maximally after the braking phase. For the purpose of measuring ME at constant push-off performance, the situation is

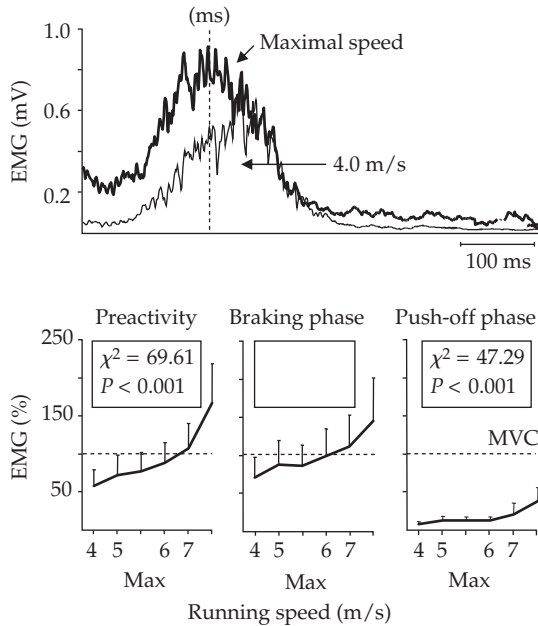


Figure 7.7 EMG activity patterns of the VL muscle at submaximal (4.0 m/s) and maximal speeds of running (above). Mean (\pm SD) relative EMG activities for three functional phases (below). The dashed horizontal lines indicate the maximum (100%) EMG measured during maximal voluntary isometric contractions (MVC). The dashed vertical line represents the beginning of the ground contact in running. Chi-squares indicate statistically significant changes with increasing running speed EMG, electromyography. (Kyröläinen et al., 2005).

different. When the subjects are dropped from a certain height, he is asked to maximally resist the downward movement during the braking phase, bend the knees minimally, and perform a submaximal push-off phase during the take-off. The subjects perform, for example, 60 muscle actions which last for a total of 3 min. In this case, the frequency is once every 3 s and is controlled by an audiosignal. When the subject leaves the ground contact, two assistants use a rope attached to the subject's vest and pull him up to the same energy level by a special pulley system for the next drop. The third assistant takes care of the subject's balance during this phase. For more details, the reader is referred to Kyröläinen and Komi (1995).

Simultaneously with the force recordings, angular displacements of the knee and ankle joints are measured by electrogoniometers. When the work intensity is low, on an aerobic level, energy expenditure (E) can be calculated by the values of oxygen uptake and respiratory ratio.

Finally, the ME of the total work (W_{tot}) is calculated as follows:

$$\text{ME} = \frac{W_{\text{tot}}}{\Delta E} \times 100 \%$$

In addition to the sledge apparatus for measuring ME, Belli et al. (1993) have introduced a kinematic arm, which is a device for 3D recording of human movement. It enables to study external mechanical work of subjects during running (Belli et al., 1993). It consists of four rigid bars linked together by three joints equipped with optical transducers. One end of the kinematic arm is connected to a fixed reference point while the other end is fixed to the back of the subject, near the CoG of the whole body, and can move freely in the three spatial directions (Figure 7.8a). For more details of this method, see Belli et al. (1993) and Kyröläinen et al. (1995). Mechanical cost measured with the kinematic arm correlates with the respective energy cost in a nonfatigued situation (Candau et al., 1998) (Figure 7.8b).

The methods discussed earlier give possibilities to study ME in a variety of ways in either repeated isolated muscle actions or during SSCs. There exist, however, many problems in any method of estimating ME and, especially, its determinant of mechanical work. Questions such as "how to take into account all distributions of potential (elastic and gravitational) and kinetic (linear and rotational) energy sources?" (Smith & Milburn, 1993) or "how to determine internal and external work?" (Winter, 1979) are constantly asked in this regard. In reference to the latter question, for example, Martin et al. (1993) have demonstrated that the total mechanical work (external work) provided a better explanation for the aerobic demands in locomotion than the segment-based model (external + internal work).

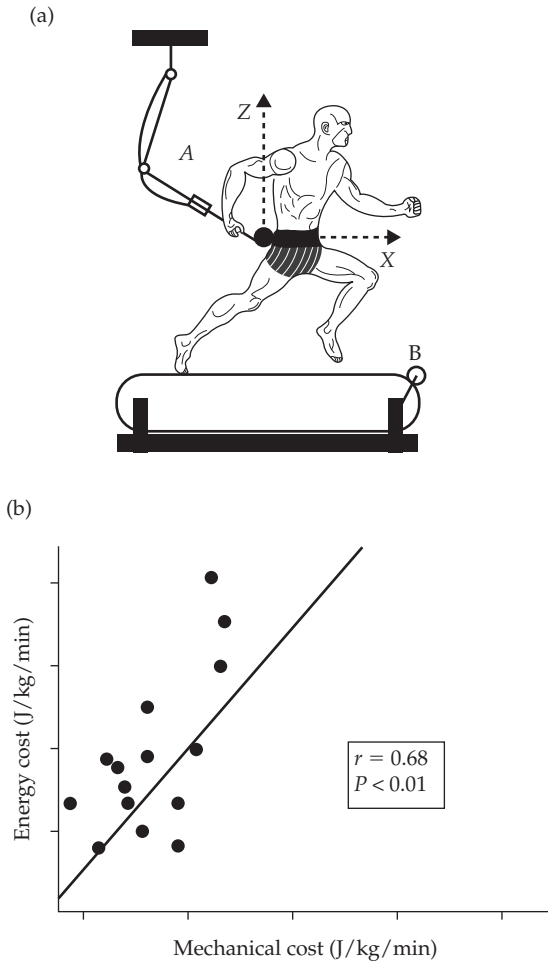


Figure 7.8 (a) Side view of the measurement with the kinematic arm. (a) and (b) are the kinematic arm and encoder wheel measuring the belt displacement of the CoG and belt, respectively (Belli et al., 1995). (b) Correlations between the energy cost and mechanical cost (measured with the kinematic arm) in running (Candau et al., 1998).

Training Adaptation of ME

As indicated in the introduction, adaptation of ME to special situations such as training has usually been examined as economy of performance. For example, this would refer to the concept of running economy where the energy expenditure is measured at selected constant speeds during a training

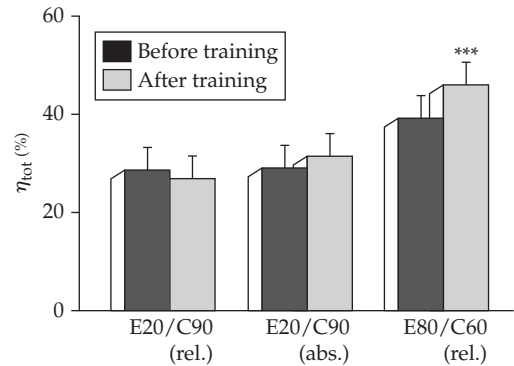


Figure 7.9 Mean (\pm SD) values of the total ME of SSC exercises before and after 16 weeks of training. E20 refers to the dropping height of 20 cm and C60/90 intensity of the concentric work (Kyröläinen et al., 1991).

period. Our approach has focused on the training, utilizing sledge exercise with different combinations of muscle actions. Figure 7.9 shows findings of such a study where subjects were trained 4 months, 3 times a week. The training mode of the SSC exercise consisted of power-type SSC jumps on the sledge as well as other plyometric jumps. All these exercises were performed with maximal intensity. The results indicated that the ME values, as measured in the sledge apparatus, improved from 40% to 46% during the training (Kyröläinen et al., 1991). These results were confirmed in another study with similar power-type training of 15 weeks, and the improvement of ME ranged from 37% to 47% (Kyröläinen et al., 2004). The overall performance improved by 8% measured in maximal jumping, which was accompanied by 24% reduction in energy expenditure of sledge jumps. The change in ME was interpreted by the improved overall neuromuscular performance, joint control strategy, and intramuscular coordination.

Consequently, the measurement of running economy such as those of Millet et al. (2002) and Storen et al. (2008) and the present calculations of ME are well in line with each other. This emphasizes improvement due to training-induced adaptation of the neuromuscular system and its function during repeated SSC exercises either in short- or long-term bases. The training has been shown to

influence, especially, the neural input to the muscle (Häkkinen & Komi, 1986; Kyröläinen et al., 1991). Subsequently, power-type strength training can improve the recoil characteristics of muscles causing increased ME (Kyröläinen et al., 1991). One should not be surprised to see similar effects from simple long-term running training alone. However, the true mechanisms of ME improvement is still very much in its infancy, especially with regard to direct measurements of the specific attributes. As obvious targets for more detailed research in the area of neuromuscular function and ME, the following aspects need to be explored: (1) distribution and adaptation of agonist activation profiles for the important phases of SSC—preactivation, braking, and push-off phases; (2) agonist/antagonist EMG profiles; (3) optimization of tendomuscular stiffness for different joints in question; (4) roles of facilitatory (e.g., stretch reflexes) and inhibitory (e.g., Golgi tendon reflexes) inputs to regulate and modify muscle function and ME; and (5) additionally, but not finally, further research should also reveal what roles the possible adaptation in fascicle-tendon interaction play in terms of ME of human locomotion, especially SSC exercise.

Fatigue

Neuromuscular fatigue is another concept, in which ME is expected to change, especially during the course of long-term SSC exercise, such as marathon running. As the present volume has a special and very comprehensive chapter on SSC fatigue, the present short discussion makes an attempt to characterize only briefly those features that are related to decrease in ME during marathon. Furthermore, our discussion is limited to aspects that deal with neuromuscular function.

Figure 7.10 implies that there is a continuous decrease in running economy in the cause of marathon run. This change was associated with the shift in the muscle activity patterns of the leg extensor muscles more to the push-off of the SSC cycle as was observed in our earlier study (Komi et al., 1986). This strongly suggests that maintenance of a

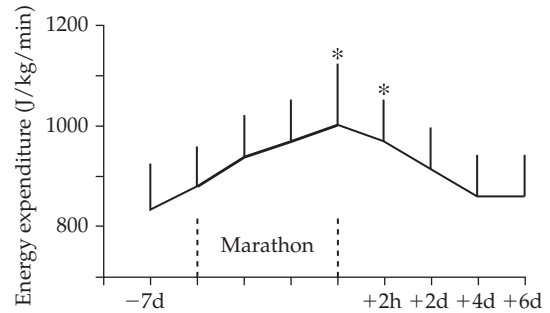


Figure 7.10 Mean (+SD) energy expenditure before, during, and after the marathon. * $P < 0.05$ (Kyröläinen et al., 2000).

constant running speed in the course of marathon is characterized by different factors: the motor unit activation is increased, and as the increase takes place mostly in the concentric phase, the ME of the exercise must decrease. The progressive shift in EMG activation toward the push-off phase means use of high energy taxing during concentric work. Loss of tolerance to repeated impact loads in the course of marathon results in a reduced utilization of elastic energy with a subsequent reliance more on concentric actions.

Kyröläinen et al. (2000) have demonstrated that after the marathon, oxygen consumption, energy expenditure (Figure 7.10), ventilation, and heart rate increase in a standardized 5 min submaximal running test, while the respiratory exchange ratio decreases simultaneously. Blood lactate may not necessarily change during the marathon while serum troponin and creatine kinase activity values increase at 2h and 2days after the marathon, respectively. In running kinematics, only minor increases in stride frequency and similar decreases in stride length can be observed. These changes are, however, so small that they are not sufficient to explain the weakened running economy. The postmarathon appearance of muscle damage/soreness processes are naturally events that may lead to changes in running mechanics and energy sources, and thus contributing to changes in running economy (Braun & Dutto, 2003). For further details, refer Chapter 11.

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Chapter 8

Transcranial Magnetic Stimulation as a Tool to Study the Role of the Motor Cortex in Human Muscle Function

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Introduction

The very first experiments regarding the effects of magnetic fields on humans date back to the end of the 18th century (d'Arsonval, 1896 in Pascual-Leone et al., 2002). Therefore, it is rather surprising that it was as long as 90 years later, when Barker et al. (1985) demonstrated that it is possible to activate the corticospinal pathway by a magnetic field (transcranial magnetic stimulation, TMS) applied over the motor cortex in human subjects. The authors recorded muscle action potentials from surface electrodes placed over *digitus minimi* on the contralateral side of the body. Later on, these responses became known as motor-evoked potentials (MEPs). Barker's experiments were published 5 years later than the first study using transcranial electrical stimulation (TES) for the same purpose (Merton & Morton, 1980).

An obvious advantage of TMS over TES is that it is painless and induces only slight, if any, discomfort. In addition, it has a good safety record, especially when following the safety guidelines for repetitive TMS (Wassermann, 1998). Basically, the only reported side effect of TMS is an inadvertent

seizure, which mainly applies to the use of repetitive TMS. In this regard, a history of epilepsy or intracortical abnormality, such as a previous stroke, maybe considered as risk factors. For single-pulse TMS, a muscle tension-type headache and slight discomfort at the site of stimulation are less serious but relatively common side effects (Wassermann, 2000).

Since the initial experiments done by Barker et al. (1985), interest in TMS has steadily increased, and a vast literature has accumulated in recent years. The ease and safety of TMS has made it a very popular tool to study human motor control, evaluate corticospinal transmission in patients with disorders, and perform functional mapping of cortical regions. In addition, TMS has been used to study and treat various neurobehavioral disorders (for review see Wassermann & Lisanby, 2001; Pascual-Leone et al., 2000) such as depression (for review see McNamara et al., 2001). The popularity of TMS is reflected in a large number of excellent reviews (Petersen et al., 2003; Di Lazzaro et al., 2004; Terao & Ugawa, 2002; Abbruzzese & Trompetto, 2002).

The aim of this chapter is to compile the most crucial information about this technique, mainly for the benefit of sport scientists, and thus to ease the first steps into the world of TMS. Although it is possible to apply series of pulses at a variety of frequencies with TMS (repetitive TMS), this chapter only highlights the single- and paired-pulse stimulation methods. Additionally, recent studies that

aimed to reveal cortical mechanisms and adaptations with respect to human muscle function serve as examples to clarify possible fields of application.

Technical Background for TMS

For magnetic stimulation, a powerful and rapidly changing current pulse is produced in a coil of wire (magnetic coil), which is placed over the scalp. The emerging magnetic field leads to an electric current that penetrates the brain and depolarizes neuron membranes, resulting in an excitatory or inhibitory postsynaptic potential (Rothwell, 1997; Hallett, 2000).

A magnetic stimulator may be looked at as a capacitor connected to a coil of wire, which has a certain inductance and resistance (Figure 8.1). The stimulator is capable of transferring energy from a bank of capacitors up to about 5 kV. During the discharge phase, the coil is then subjected to high currents of up to 10 kA. The waveform of the magnetic field is proportional to the discharge current, whereas the induced electric field waveform is proportional to the rate of change of the magnetic field

(Hess et al., 1987). Bone, fat, gray matter, and white matter present little or no impedance to a magnetic field of rapidly changing intensity. The direction of the current flow in the coil is opposite to the direction of the induced current in the nervous tissue, and it flows parallel to the surface of the coil (Weber & Eisen, 2002; Terao et al., 2000).

Present stimulators can create two different types of stimulus waveforms: monophasic and biphasic. In general, several single-pulse studies have shown that the effectiveness of these two waveforms is different (Di Lazzaro et al., 2001a; Arai et al., 2005). In the study of Arai et al. (2005), resting motor threshold (rMT) was significantly lower for biphasic TMS compared to monophasic TMS of the motor cortex. The reason might be that in biphasic TMS, the second phase of induced current will also induce neuronal voltage changes. In addition, since the second phase of induced current is always larger than the first, biphasic TMS should always be more effective than monophasic TMS with the same intensity output. Moreover, it has been shown that the most effective direction for the current flow is opposite between monophasic and biphasic TMS

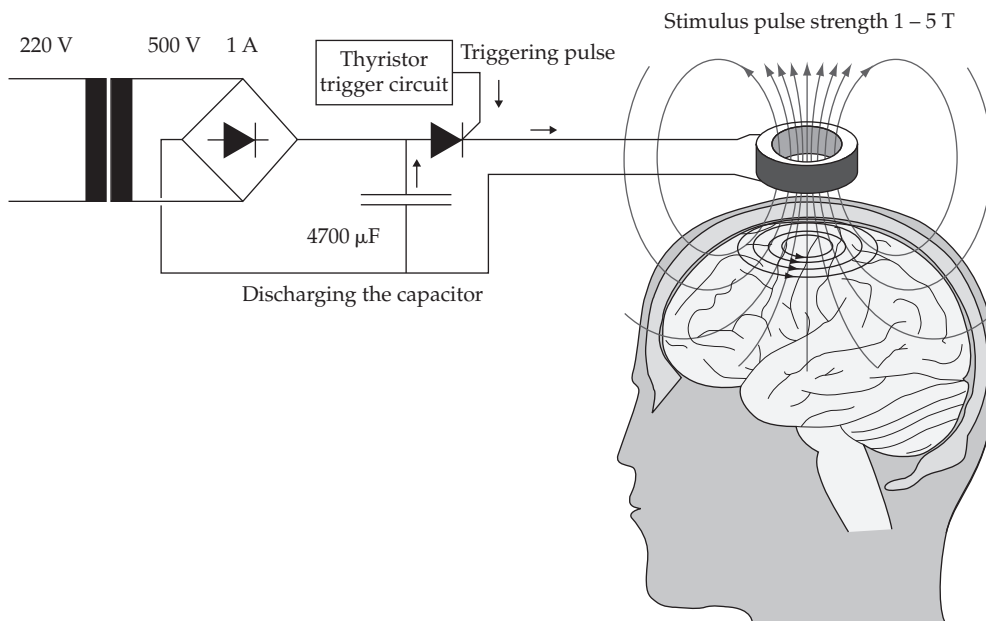


Figure 8.1 The principle of the magnetic stimulator. The magnetic field generated by the coil induces an intracortical electric field. (Modified from Malmivuo & Plonsey, 1995.)

(Kammer et al., 2001). Another important aspect when considering the differences between the two waveforms is the complexity of the stimulation. In this regard, it is known that the descending volleys evoked by biphasic stimulation are much less homogenous than those produced with monophasic stimulation (Di Lazzaro et al., 2001a).

Magnetic stimulation does not involve a small and predictable site of stimulation. Thus, the term "focal" stimulation, which is sometimes used, is misleading. This is because the physics of magnetic fields causes them to diverge after they leave their source. The geometry of the stimulating coil, together with the structural organization of the neuronal circuitry of the cortex, determines the amplitude and spatial distribution of the activating function, and thus the site of stimulation.

Magnetic field strength alone is not a suitable measure of magnetic stimulator performance. Magnetic field strength is defined as the magnetic flux density, and it does not indicate the total magnetic flux produced by the coil. Thus, in a small

coil, the magnetic flux is concentrated in a small area, and the magnetic field intensity is higher than with a larger coil. However, the field declines much more rapidly with the small coil (Hallett, 2007; Jalinous, 1991).

The first magnetic stimulator coils were circular in geometry. In such a coil, the electric field is zero under the coil center and maximal under the mean diameter of the winding (Figure 8.2a). Thus, the stimulation is likely to occur under the winding. Circular coils are considered to be capable of activating neurons that lie 1.5–2.0 cm below the scalp surface (Epstein et al., 1990). The primary disadvantage of the circular coil is the relative uncertainty regarding the exact site of stimulation (Pascual-Leone et al., 2002).

A figure-of-eight coil is composed of two circular coils, which are placed side by side, and connected so that the current in one coil rotates in the opposite direction to that in the other. This coil type has become more popular because it can stimulate more selectively with a spatial resolution of 5 mm.

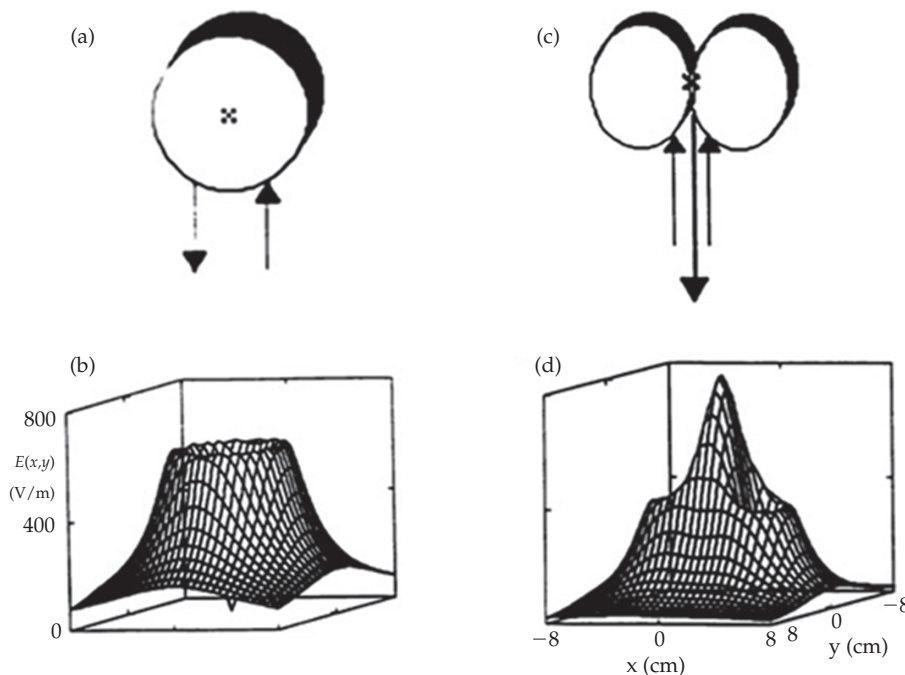


Figure 8.2 Circular coil (a) and its induced electric field (b). Figure-of-eight coil (c) and its induced electric field (d). (Modified from Hallett, 2007.)

The amplitude of both the magnetic and electric fields is highest at the intersection of the windings (Figure 8.2b). There are two advantages when stimulating with a figure-of-eight coil. Firstly, it stimulates the mesial surface of the frontal lobe more effectively, so the legs can be stimulated. Secondly, it is more comfortable for subjects because of the more focal area of stimulation (Terao et al., 2008; Jalinous, 1991).

A double-cone coil is also made out of two circular coils in a similar manner as the figure-of-eight coil. However, the coils are connected to each other at an angle of 90–100°, and their diameter is usually slightly larger (~12cm). A double-cone coil is able to create greater electrical field intensity and can thus penetrate deeper into the brain (3–4cm in depth) than other coil types. Therefore, it has been considered to be the best tool for leg motor area stimulation (Roth et al., 2002).

Physiological Background

Anatomy of the Corticomotoneuronal System

Several distinct yet interconnected anatomical regions are responsible for human motor function. They include the primary motor cortex, the premotor areas and supplementary motor cortex, basal ganglia, thalamus, cerebellum, brain stem, and reticular formation. The primary motor cortex is thicker and has a lower cell density than other parts of the cerebral cortex. There are six distinct layers of neurons in the motor cortex. The main output cells are large pyramidal cells in the fifth layer, and smaller cells in the third layer. The corticospinal pathway originates from these pyramidal cells, and is the only descending motor pathway that is known to make monosynaptic connections with the spinal motor neurons (Weber & Eisen, 2002).

Each cortical motor neuron synapses with various spinal motor neurons, and each spinal motor neuron receives inputs from many cortical motor neurons. Evidence to support a significant contribution of corticospinal projections to muscle responses comes from experiments in which the effect of TMS on the firing probability has been

investigated (Palmer & Ashby, 1992; Brouwer & Ashby, 1992). These studies were able to reveal a shorter rise time of the excitatory postsynaptic potential (EPSP) in the spinal alpha motor neurons evoked by TMS, which corresponded to increased firing probabilities. For polysynaptic EPSPs, rise times would have been longer (Petersen et al., 2003).

This complicated arrangement between cortical and spinal motor neurons affords humans their accurate movement control and ability to perform complex movements. This system regulates the control of force, grip accuracy, angulation, rate of change of movement, and muscle tension, and it may also be essential to the learning of new motor skills (Weber & Eisen, 2002). It has to be stressed explicitly that the MEP always reflect responsiveness of cortical and spinal neurons.

The pyramidal neurons are subjected to excitatory and inhibitory modulations from layers two to four. Inhibitory interneurons, basket and stellate cells, are located in the third and fourth layer. In addition, there are small corticocortical excitatory pyramidal neurons in layers two and three, which have preferentially oriented horizontal dendrites. Gamma-aminobutyric acid (GABA) is the inhibitory neurotransmitter, while glutamate is the main excitatory neurotransmitter. TMS can activate both types of interneurons, and thus the motor cortical output is based on the net effect (Weber & Eisen, 2002; Ziemann & Rothwell, 2000).

Physiology of TMS

Motor-Evoked Potential

A prerequisite for using TMS as a tool to evaluate cortical excitability is that TMS excites the aforementioned pyramidal neurons at or before the cell soma, and not the axons of the corticospinal cells directly. Over the past two decades, many studies have examined the exact site of TMS stimulation (Burke et al., 1993; Nielsen et al., 1995; Di Lazzaro et al., 2001b). The major point that can be extracted from these studies is that, in general, TMS does not seem to penetrate deep enough into the brain to be able to directly activate the corticospinal axons. Whether it activates some other neurons in

the brain that have synaptic input to the pyramidal cells seems to be not that important, since the evoked responses would be influenced by changes in cortical excitability in any case (Petersen et al., 2003).

The exact site of TMS stimulation has usually been studied by comparing the MEP latencies between TMS and TES of the motor cortex in animals and humans (Figure 8.3). In a classic study by Patton and Amassian (1954), it was shown that single electrical stimulus applied to the motor cortex of cats and monkeys induced multiple descending volleys in the corticospinal pathway. They were able to prove that the initial volley, which they called the direct wave (D-wave), was induced by direct stimulation of the corticospinal axons. In addition, they found evidence that the latter volleys originated from indirect, synaptic activation of the corticospinal cells, and thus they called them indirect waves (I-waves).

It has been shown that the latency of the MEP using TES is usually slightly shorter than the one

evoked by TMS. Because of the different activation of the corticospinal cells, the descending corticospinal volleys are also different between TMS and TES. In general, it is agreed that the MEP evoked by TES consists of an early D-wave (Figure 8.3), which, as mentioned earlier, reflects the direct activation of cortical descending axons (Di Lazzaro et al., 2004; Ziemann & Rothwell, 2000). Di Lazzaro et al. (1998a) have reported a latency that is 1–1.4 ms longer in TMS than the volley recruited by TES in the hand area of the cortex. They also showed increases in the size of this volley, as well as later volleys, with increasing stimulus intensity. Thus, they concluded that the volleys recruited by TMS are likely to be I-waves (Figure 8.3). I-waves are numbered according to their latency and are referred as I₁, I₂, I₃, and I₄ waves. Although it has been shown that TMS mainly elicits I-waves, there is a possibility that, especially in the hand area, very high intensity stimulation (180–200% of active motor threshold, aMT) can lead to detectable D-waves (Di Lazzaro et al., 1998a, 2004; Ziemann & Rothwell, 2000; Hallett, 2007).

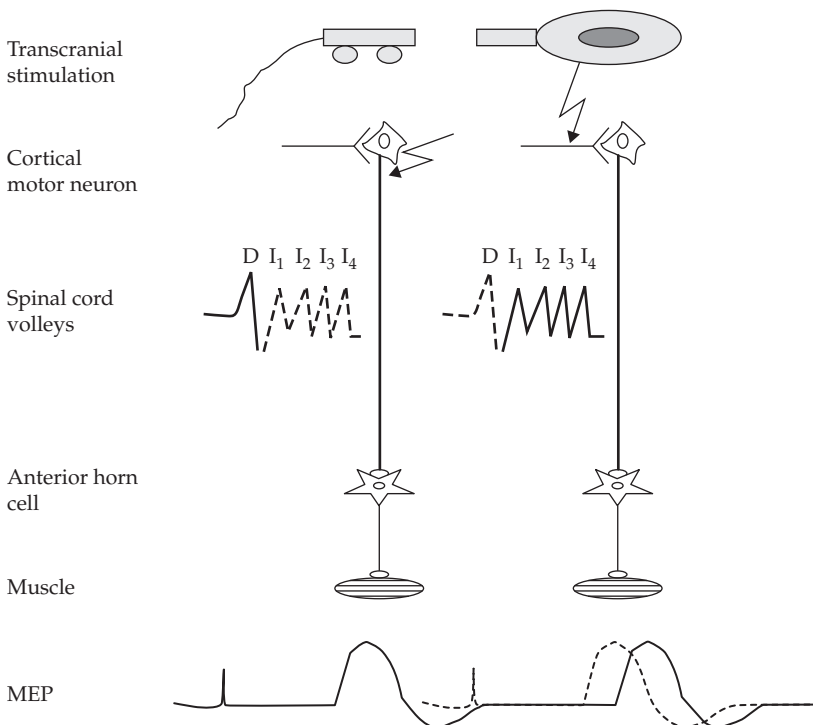


Figure 8.3 Different excitation of the pyramidal cells induced by TES and TMS. MEP, motor-evoked potential. (Modified from Weber & Eisen, 2002).

As noted earlier, it is rather well accepted that I-waves are produced by indirect activation of the pyramidal cells. The actual activation sites consist of axon collaterals of pyramidal cells, afferent fibers from the motor thalamus, intracortical interneurons and other associated fibers from other cortical areas, particularly the primary somatosensory area and premotor cortex. Several studies suggest that intracortical interneurons and corticocortical neurons must be responsible for the production of I-waves. Both of these systems have a powerful modulatory effect on the output of the motor cortex (Ziemann & Rothwell, 2000; Sakai et al., 1997).

The effect of TMS on the MEP depends critically on the direction of the induced current in the brain. This is because the facilitatory interneurons are preferentially aligned in one particular direction, while the inhibitory interneurons are oriented in random directions (Arai et al., 2005). It is important to place the coil so that the current runs approximately perpendicular to the central sulcus (Figure 8.2). Moreover, I-waves are best recruited when the induced current is directed posterior-to-anterior in monophasic stimulation and anterior-to-posterior to posterior-to-anterior in biphasic stimulation. However, there are small differences between subjects in the optimal direction of induced current. Presumably, this is because of variations in the precise anatomy of the central sulcus and motor cortex between individuals (Ziemann & Rothwell, 2000; Sakai et al., 1997).

It has been suggested that TMS in the leg area of the motor cortex recruits neurons in a different way than in the hand area. This could be due to different recruitment thresholds of the different structures of the motor cortex. On the other hand, lower limb neurons lay in the interhemispheric fissure, and the corticospinal cells are parallel to the brain surface. Therefore, in theory, D-waves could be more easily elicited in the leg area than in the hand area with TMS. This is in line with the findings of Priori et al. (1993) that the latency of MEPs in the TA muscle was the same for TMS and TES. Thus, they concluded that TMS of the leg area of the motor cortex is likely to produce D-waves. Terao et al. (2000) only reported this possibility with very high stimulating intensities, as is the case with the hand site

stimulation. This was particularly evident when stimulating with the double-cone coil. Regardless of the coil type, the near-threshold intensities of TMS always represented the I1-wave latencies. This is in line with the study of Di Lazzaro et al. (2001a), who found that TMS evoked latencies that were comparable to D-waves induced by TES with an anode positioned 2 cm lateral to the vertex, only at higher TMS output intensities. Thus, in contrast to some suggestions, it could be concluded that the behavior of the evoked potentials induced by TMS seems to be similar between the hand and leg areas of the motor cortex. In both cases, D-waves can only be elicited at very high TMS intensities. This information is relevant, since any shift in the MEP latency during the measurements would indicate changes in the site of stimulation, which makes interpretation of the data more complex.

Input–Output Relationship

In order to standardize the output intensity during TMS measurements, it is crucial to define the rMT in the relaxed target muscle. rMT reflects the global excitability of the corticospinal pathway, including large pyramidal cells, cortical excitatory and inhibitory interneurons, and spinal motor neurons. rMT has usually been defined as the minimum TMS intensity that elicits reproducible MEP responses of at least 50 μ V in about 50% of 5–10 consecutive trials (Borojerdi et al., 2001; Cacchio et al., 2009) or 3 out of 5 trials (Rossini et al., 1994). A slight voluntary contraction of a target muscle reduces motor threshold (MT), thus the aMT is lower compared to rMT.

In experiments that have investigated the properties of the corticospinal pathway within a single experimental session, TMS output intensity has usually been standardized in relation to rMT. In passive muscle conditions, higher intensities than those used to elicit rMT have usually been used. This is called “suprathreshold stimulation.” In active muscle conditions, stimulus intensities that are lower than rMT (subthreshold stimulation) can also be used.

As is the case with H-reflex measurements, in TMS it is important to understand the relationship between the intensity of the stimulation (input)

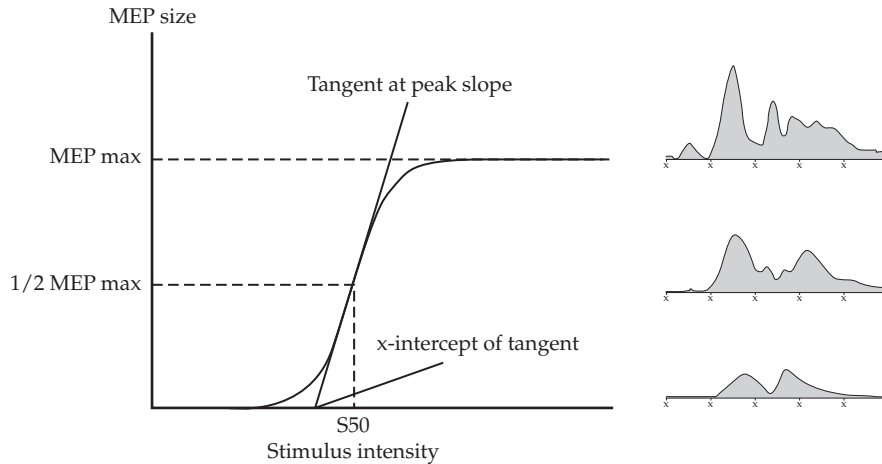


Figure 8.4 An example of a sigmoid fit to a MEP size versus stimulus intensity plot. MEP, motor-evoked potential. (Modified from Carroll et al., 2001.)

and the magnitude of the corresponding MEP (output). Systematic analysis of the input–output relationship represents the recruitment of motor units with increasing stimulation intensities (Figure 8.4). This relationship has usually been measured from the target muscle with certain incremental steps of the stimulation intensity, starting from rMT or slightly below it. In the process, increases in stimulation intensity can be related either to the stimulator output or to rMT. To reach acceptable measurement repeatability, 10 simulations have been used at each stimulation intensity (Devanne et al., 1997; Ridding & Rothwell, 1997; Boroojerdi et al., 2001; Carroll et al., 2001).

It has been shown in several experiments and a variety of muscles that the TMS recruitment curve represents a sigmoid increase in MEP amplitude with increasing stimulus intensity until it reaches a plateau (Devanne et al., 1997; Ridding & Rothwell, 1997; Cacchio et al., 2009). Both the plateau value and the slope of the curve have been taken as a general measure of the excitability of the corticospinal pathway (Carroll et al., 2001). However, in some cases, especially in the lower leg muscles, where rMT is higher than in hand muscles, the plateau phase may not be reached. This emphasizes the importance of the parameters related to the slope of the curve. In order to analyze the slope of the curve, the magnitude of the MEP response (peak-to-peak

amplitude and/or area) should be plotted against the stimulus intensity. The data can then be fitted to some mathematical function to quantify the shape and/or steepness of the curve (Figure 8.4). For this purpose, several functions have been suggested (Capaday, 1997; Devanne et al., 1997; Carroll et al., 2001).

The measure of the MEP response using a single sub- or suprathreshold intensity or several intensities for the recruitment curve has usually been used in studies including a single testing session as an indication of the global excitability of the corticospinal pathway. There are only a few experiments that have attempted to use TMS to detect adaptations in the corticospinal pathway that occur over time. This is due to the fact that MEP responses are very sensitive to several factors that can be difficult to control across different testing sessions. The most crucial part is to precisely replicate the position of the magnetic coil on different days. Failure to do this may lead to stimulation of different corticospinal cells across testing sessions, and thus the recording of responses from different motor units. Carroll et al. (2001) studied the reliability of the different parameters of the TMS recruitment curve in a longitudinal study design, both in passive and active muscle conditions. Data were recorded from the first dorsal interosseous muscle of eight subjects on three separate days. They found moderate (0.47)

to high (0.81) intraclass correlation coefficients for the parameters of the sigmoid function of the curve. Although there is an obvious lack of studies showing the reliability and repeatability of the different TMS measurements over time, based on the results of Carroll et al. (2001), the measurement of input–output parameters of the corticospinal pathway seems to be a reliable method during longitudinal investigation of corticospinal plasticity.

The Effect of Muscle Activity on the MEP

For the interpretation of corticospinal excitability, it is important to understand how muscle activity can affect MEP responses. As already mentioned in the previous section, voluntary activity decreases MT of the target muscle. In turn, it increases the size of the MEP. However, this increase is not similar in all muscles. It has been shown that MEP size can increase with increasing force of contraction up to around 50–75% of maximal voluntary contraction (MVC) in the brachioradialis and biceps brachii (BB) muscles, but then decrease again (Martin et al., 2006). On the other hand, in some other muscles, such as abductor digiti minimi, only a slight increase in MEP size has been observed with increasing muscle activity (Hess et al., 1987). Although the reason for different behavior of the different muscles is not clear, the increased MEP size has been attributed to increased excitability of motor cortical output cells and motor neurons during voluntary contraction (Di Lazzaro et al., 1998a; Taylor et al., 2002). However, the reduction in MEP size with further increases in muscle activity seems to be mediated by changes below the motor cortex, most likely at the motor neuron pool. Martin et al. (2006) found some evidence that while the motor neurons become less responsive during very strong contractions, output from the motor cortex is not limited in the same way. This is especially true if the force levels correspond to those where motor neuron pools operate by rate coding.

If MEPs are measured during a movement with varying background EMG activity (BGA), it is important to normalize the MEP to BGA (Schubert et al., 1997). In longitudinal studies, it is possible to express the MEP size as a proportion of BGA

or M_{\max} , as well as force or torque, for example (Carroll et al., 2002). However, it has to be kept in mind that the relationship between MEP size and BGA is complex and has to be controlled carefully (Martin et al., 2006; Taube et al., 2008).

Silent Period

The silent period (SP) is an interesting physiological feature that occurs after TMS during voluntary muscle activation. The SP usually refers to the pause in the ongoing EMG activity after the MEP. It has been shown in several studies that the duration of the SP increases with increasing TMS output intensities (Homgren et al., 1990; Wilson et al., 1993). In a study by Wilson et al. (1993), an increase in TMS stimulus intensity from 10% to 50% resulted in an increase in the duration of the SP from a mean of 50 to 80 ms. Surprisingly, the level of prestimulus BGA does not seem to have an effect on SP duration (Haug et al., 1992; Inghilleri et al., 1993).

It is generally accepted that the SP originates from both spinal and cortical mechanisms. Studies with H-reflex stimulation have shown that a deep depression of the reflex exists at the early phase of the SP (the first 30–50 ms), suggesting the involvement of spinal mechanisms. However, since the H-reflex recovers well before the end of the SP, the latter part of the SP is considered to be of cortical origin (Chen et al., 1999; Ziemann et al., 1993).

Although spinal and cortical origins for the SP have been suggested, the exact mechanisms have not been thoroughly defined. As for spinal inhibitory modulation, Renshaw inhibition has probably received the most attention (Inghilleri et al., 1993). Possible explanations for the latter part of the SP include intracortical mechanisms, such as recurrent collaterals from pyramidal tract neurons, and activation of groups of inhibitory cortical neurons within the cortex (Terao & Ugawa, 2002; Werhahn et al., 1999).

Overall, these findings seem to imply that TMS is capable of stimulating some inhibitory cortical neurons within the motor cortex, and thus SP duration can be regarded as an indicator of cortical excitability. However, to the authors' knowledge, this parameter has not been used in experiments that

aimed to study muscle function. This is probably due to the fact that the exact mechanisms of the SP are still unknown.

TMS as a Tool to Evaluate the Role of the Motor Cortex in Muscle Function

As mentioned earlier, MEPs evoked by TMS originate from I-waves and thus the MEP represents the responsiveness of neurons located in the motor cortex as well as of spinal motor neurons. When aiming to distinguish between mechanisms or adaptations at the spinal and cortical levels, different methodological approaches can be beneficial. The following paradigms have been used in recent studies related to the field of exercise science to elucidate the role of the motor cortex during natural and sports-specific movements, and will be discussed with respect to methodological aspects and practical applications:

1. Comparison of MEPs evoked by TMS with other evoked potentials like evoked potentials by TES, cervicomedullary stimulation (CMS), and Hoffmann reflex stimulation (H-reflex).
2. Conditioning of MEPs evoked by TMS with subthreshold TMS stimulation prior to the suprathreshold stimulus, which results in short intracortical inhibition (SICI) or intracortical facilitation (ICF), respectively.
3. TMS with a subthreshold intensity to suppress the EMG of a contracting muscle.
4. Conditioning of MEPs evoked by TMS with ipsilateral TMS stimulation.
5. Conditioning of the H-reflex by subthreshold TMS.

Comparison of MEPs Evoked by TMS with Other Evoked Potentials

Transcranial Electrical Stimulation

It was mentioned earlier in this chapter (see Figure 8.3) that TES and TMS differentially excite the pyramidal cells. Whereas TES is supposed to activate the corticospinal neurons directly, probably at an axonal site (D-wave), the MEP evoked by a TMS pulse is more likely brought about by trans-synaptic excitation of corticospinal neurons (I-waves). In

consequence, MEPs evoked by TMS are influenced by responsiveness of neurons located in the motor cortex, whereas this is much less the case for MEPs in response to TES. By comparing the evoked responses to TES and TMS, it should be possible to gain insight into whether changes occur primarily at the cortical or subcortical levels in the CNS.

With this method, it has been shown that index finger abduction training with approximately 70% of one repetition maximum performed over 4 weeks (three sessions per week) that resulted in significant increased MVC also changed the input-output properties of the corticospinal pathway (Carroll et al., 2002). The authors observed a reduction in the magnitude of the evoked responses to both TMS and TES for a given level of torque or EMG activity following resistance training. They concluded that the repetitive execution of a simple movement (finger abduction) against a large resistance does not substantially affect the functional properties of the motor cortex. Interestingly, Perez et al. (2004) reported increased MEPs after motor skill training for TMS but not for TES, indicating neural adaptations at the cortical level after the training of a coordinative demanding task. Similar results were reported during supported versus freestanding in man (Tokuno et al., 2009). MEPs evoked by TMS were significantly facilitated in freestanding compared to supported standing whereas MEPs evoked by TES remained unchanged. This result indicates enhanced cortical excitability during a postural task.

Cervicomedullary Stimulation

Cervicomedullary stimulation is applied between the mastoids to evoke SLRs in the EMG of skeletal muscles. Such stimulations are able to activate axons in the corticospinal pathway at the level of the cervicomedullary junction. As a result of this activation, a short-latency response in the muscle, termed a "cervicomedullary motor-evoked potential (CMEP)," can

be observed (for review see Taylor & Gandevia, 2004). Based on the assumption that the corticospinal pathway is free from presynaptic control (Nielsen & Petersen, 1994; Jackson et al., 2006) and that the CMEP contains a dominant monosynaptic component (Petersen et al., 2002), changes in CMEPs reflect alterations at the spinal motor neuron pool itself, whereas changes in the ratio of CMEP to MEP area reflect effects at the motor cortex. The fact that CMEPs work well during maximal contractions (in contrast to motor potentials evoked by TES) makes the comparison of MEPs with CMEPs the most direct possibility to identify modulatory actions at the cortical versus spinal level during strong muscle contractions.

It should be acknowledged that MEPs and CMEPs may activate motor neurons differently because the MEP involves multiple volleys and the CMEP only a single volley. Therefore, motor unit potentials after TMS are more dispersed and some motor neurons may discharge more than once, which can affect the size of potentials averaged from surface EMG (Keenan et al., 2006). However, Taylor et al. (2002) provided some evidence that these differences may not affect the susceptibility of CMEPs or MEPs to changes in spinal excitability specifically. The authors reported an occlusive interaction between responses to CMS and descending action potentials evoked by TMS at short interstimulus intervals (ISIs) in BB. This observation is consistent with the two stimuli activating some of the same corticospinal axons for this muscle.

Martin et al. (2008) used experimental muscle pain to investigate the effects of group II and IV muscle afferents at the cortical and spinal levels. During pain, the size of CMEPs in relaxed biceps and triceps muscles increased significantly. MEPs did not change, but relative to CMEPs, they decreased in both muscles significantly. Since the results for active muscles were rather similar, they concluded that activity of these small muscle afferents facilitates motor neurons innervating

elbow flexors and extensors but depresses motor cortical cells projecting to these muscles. Gruber et al. (2009) observed lower CMEPs and increased MEP to CMEP ratios in maximal lengthening compared to maximal isometric contractions and concluded that properties of motor neurons, as well as neurons located at the motor cortex, were modulated differently during lengthening versus isometric contractions. The observed reduction in CMEPs indicates that spinal excitability was considerably lower in lengthening than isometric contractions, whereas a moderate increase in MEP/CMEP ratio indicates that cortical excitability was slightly higher. It has been suggested that increased cortical excitability results in extra excitatory descending drive during muscle lengthening in order to compensate for spinal inhibition.

H-reflex

For some muscles like M. SOL and M. abductor pollicis brevis, MEPs evoked by TMS were compared to potentials evoked with H-reflexes (for review see Nielsen et al., 1999). This was done mainly because H-reflexes can be easily recorded in these muscles and peripheral nerve stimulation to elicit H-reflexes is much less painful compared to TES and CMS. However, there is an important difference that limits the interpretation of results regarding the comparison of MEPs and H-reflexes. As mentioned earlier, the corticospinal pathway can be regarded as being free from presynaptic control (Nielsen & Petersen, 1994; Jackson et al., 2006), whereas in the Ia afferent pathway, presynaptic control is modulated permanently and extensively according to postural and kinematic constraints (for review see Zehr, 2002). Therefore, a direct comparison in order to infer cortical excitability is improper. Nevertheless, some studies have demonstrated benefits of applying H-reflexes and TMS together, mainly when a diverging effect can be expected.

Solopova et al. (2003) compared standing on a rigid floor and on a rocking platform. The authors reported increased MEPs but a tendency toward decreased H-reflexes in the SOL muscle while balancing. Taube et al. (2008) observed a time-dependent modulation in both MEPs and H-reflexes over the course of ground contact in DJs (Figure 8.5). The first peak in EMG after touchdown seems to rely predominantly on Ia afferent feedback, thereafter spinal contributions progressively decline and the MEP is not facilitated until approximately 120 ms. Therefore, cortical influence via the corticospinal pathway was suggested to be minor during the first 100 ms of ground contact in DJs. Lundbye-Jensen and Nielsen (2008a) showed increased H-reflex amplitudes in abductor pollicis brevis without changes in MEPs after hand immobilization. The authors speculated that changes in presynaptic inhibition or postactivation depression could potentially be underlying mechanisms. This was confirmed in a later study (Lundbye-Jensen & Nielsen, 2008b). However, it has to be acknowledged once again that by comparing H-reflexes and MEPs, it is not possible to draw explicit conclusions about the site of adaptation.

SICI and ICF

Within the motor cortex there are intrahemispheric interactions, and the net output to the spinal cord depends on facilitating and inhibiting mechanisms. By applying a TMS pulse over the motor cortex, we influence the state of many cortical neurons located in this region of the brain, and it is therefore not surprising that such a pulse can systematically alter the MEP of a second pulse elicited directly after the first one. Interestingly, suppression or facilitation of this MEP depends mainly on the time delay between the first and second pulses. It was shown that a subthreshold pulse (conditioning stimulus) is able to suppress the subsequent suprathreshold

pulse (test stimulus) when delivered 1–6 ms prior to the test stimulus (SICI) but can facilitate this test stimulus when delivered 10–15 ms prior to it (ICF) (Kujirai et al., 1993). Valls-Sole et al. (1992) elicited two suprathreshold TMS pulses at ISI of 50–200 ms and demonstrated suppression of the second MEP (long intracortical inhibition, LICI). SICI, LICI, and ICF are referred to as paired-pulse paradigms.

Many studies were done to elucidate their underlying mechanisms. For LICI these mechanisms still remain largely unknown, whereas more information is available on the mechanisms underlying SICI and ICF (for review see Chen, 2004; Reis et al., 2008). Nakamura et al. (1997) and Di Lazzaro et al. (1998b) were able to demonstrate suppression of the initial I-wave after the subthreshold conditioning TMS stimulus by directly recording the spinal cord volleys. These studies provided evidence for intracortical mechanisms to explain the suppression of the test stimulus. It has already been mentioned that GABA acts as the inhibitory neurotransmitter within the motor cortex. Interestingly, it has been shown that after administration of GABA agonists, SICI was only enhanced at ISIs from 2.5 ms onward, but not with an ISI of 1 ms, indicating different underlying mechanisms for these ISIs (Fisher et al., 2002). With an ISI of 1 ms, SICI is probably restricted to changes in synaptic transmission between cortical neurons, whereas with an ISI of 2.5 ms, mainly GABAergic inhibition influences the measure of SICI. Consequently, in most of the studies concerning the role of intracortical inhibition in motor control, an ISI of 2.5 ms has been used.

Intracortical inhibitory mechanisms are supposed to play a functional role in motor control, especially during the initiation of voluntary contractions (Reynolds & Ashby, 1999). In this study, they found reductions in intracortical inhibition starting approximately 95 ms before muscle activity. Floeter and Rothwell (1999) interpreted this result as “Releasing the brakes before pressing the gas pedal.” Based on this assumption, alterations in intracortical inhibitory or facilitatory mechanisms should be expected after motor skill training or strength training.

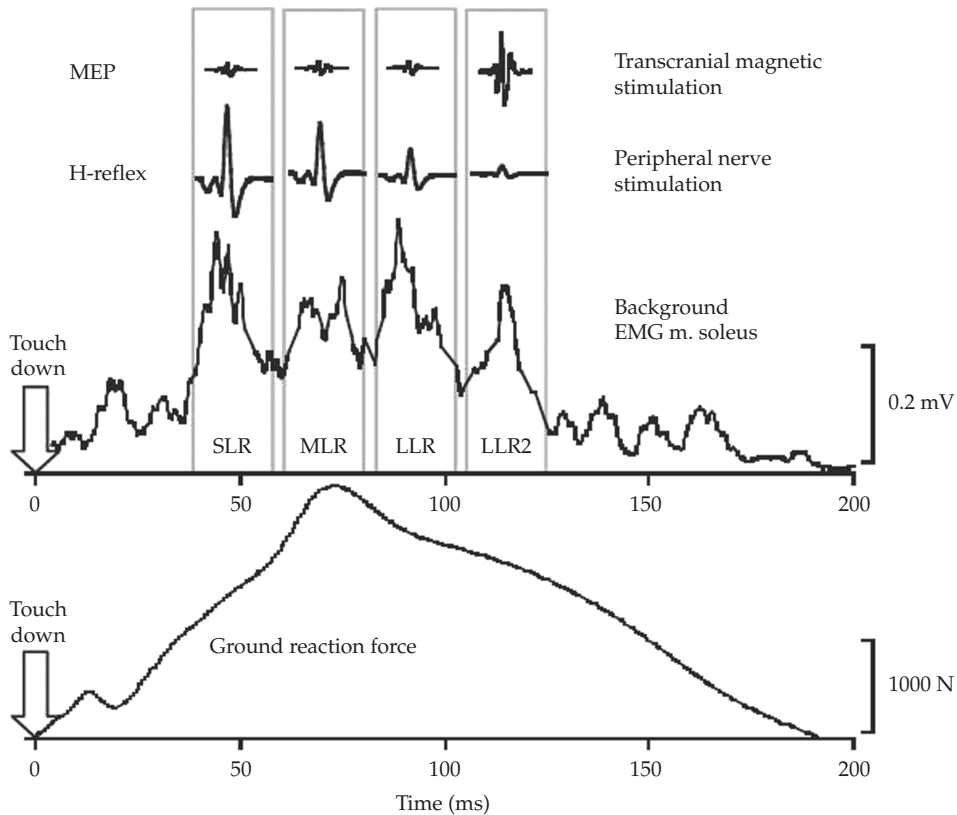


Figure 8.5 Stimulations for H-reflexes and MEPs were triggered on distinct EMG responses of the BGA during DJs (short- [SLR], medium- [MLR], and long-latency response [LLR1 and LLR2]). A clear increase of the MEP at LLR2 occurred whereas the H-reflex decreased steadily throughout the ground contact phase. MEP, motor-evoked potential; EMG, electromyography. (Modified from Taube et al., 2008, p. 1244).

Perez et al. (2004) studied the effect of motor skill training on SICI with an ISI of 2.5ms. They found that SICI was significantly reduced up to 15 min after visuomotor skill training. At the same time, ICF was not affected. They suggested that the existing cortical projections to the muscle are probably suppressed under normal conditions due to intracortical inhibition. However, after training this inhibition maybe removed and more functional connections maybe activated by TMS. On the contrary, ICF increased after a simple task involving active repetitive wrist

movements (Lotze et al., 2003), whereas after training of a more complex sensorimotor task, ICF remained unchanged (McDonnell & Ridding, 2006). These results indicate a functional role of SICI during complex tasks, while ICF seems to be more sensitive to simple repetitive movements.

Subthreshold TMS to Suppress the Output from the Motor Cortex

The above-mentioned paradigms (1 and 2) provide insight into changes in cortical excitability relative

to a defined task (e.g., rest or tonic contraction at a certain contraction level) or allow a comparison before and after an intervention. However, it has to be mentioned that these paradigms do not give direct evidence that cortical neurons contribute more or less to a given muscle activation. To demonstrate the direct contribution of cortical neurons during a motor task, very weak TMS can be used. It has been shown that with TMS far below MT, it is possible to selectively activate local inhibitory neurons in the motor cortex that can produce suppression in the EMG of a voluntarily contracting muscle (Davey et al., 1994). A prerequisite of this method is that the subthreshold TMS does not evoke any visible MEP in the EMG neither any facilitation that could induce an SP. Several experiments have suggested that this TMS-evoked EMG suppression is indeed a consequence of the activation of intracortical inhibitory interneurons that suppress the output from the motor cortex (Davey et al., 1994; Petersen et al., 2001).

Petersen et al. (2001) used subthreshold TMS during human walking to investigate the involvement of the motor cortex. They were able to show that subthreshold TMS produced a clear suppression of ongoing EMG activity during walking (Figure 8.6). Thus, they demonstrated that motor neuronal activity can be suppressed by activation of intracortical inhibitory circuits. They concluded that this shows for the first time that activity in the motor cortex is directly involved in the control of the muscles during human walking. Van Doornik et al. (2004) used the same method to investigate the transcortical nature of the long-latency stretch reflex (M3) in the human TA muscle. Stretch reflexes were elicited using a special ankle dynamometer. Subthreshold TMS was applied with several intervals prior to M3. The authors quantified reflex components using 20ms EMG windows and found that subthreshold TMS evoked significantly larger depression of M3 than of the background

EMG in the same time frame when applied clearly prior to M3. In addition, the effect on M3 was clearly larger than on the other reflex components. Therefore, they concluded that the long-latency reflex in the TA muscle is at least partly transcortical in nature.

Ipsilateral TMS

It is well known that motor cortices (M1) in both hemispheres are interconnected via transcallosal projections and that a TMS pulse applied over M1 leads to changes in the responsiveness of neurons located in the contralateral M1 (Ferbert et al., 1992). The physiological background is complex, and at least two inhibitory and three facilitatory interactions

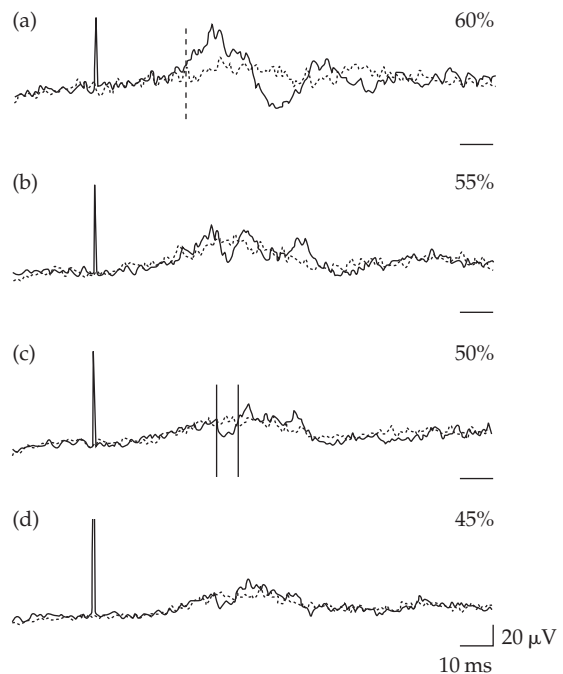


Figure 8.6 The effect of different subthreshold TMS intensities on m. TA EMG in human walking. Two hundred recordings were averaged for controls (gray traces) and for TMS (black traces). At 50% of the maximum stimulator output, no excitatory effect (MEP) appears, but rather a significant reduction in the muscle activity. (Modified from Petersen et al., 2001.)

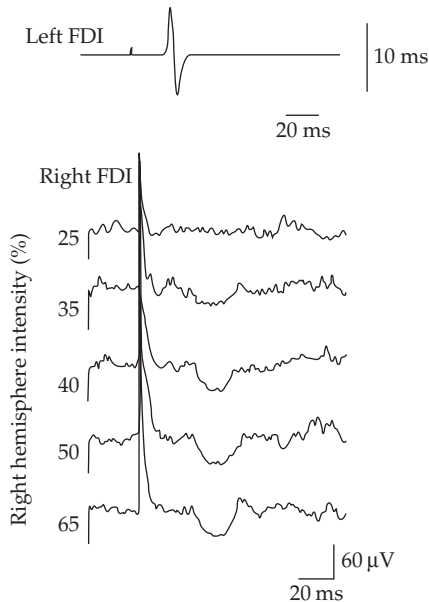


Figure 8.7 TMS over the right hemisphere evokes an MEP in the left FDI muscle and a suppression of the voluntary EMG activity in the right FDI muscle. This suppression increases with increasing stimulus intensity while the time course remains constant. FDI, first dorsal interosseus. (Modified from Ferbert et al., 1992, p. 538.)

have been described, depending on the ISIs between the conditioning and test stimuli (for review Reis et al., 2008). In contrast to SICI and ICF, and this is one big advantage of this method, ipsilateral TMS affects the BGA directly. Thus, during a tonic contraction, a TMS pulse over the ipsilateral hemisphere can evoke an SP in the ongoing EMG activity of a voluntarily contracting muscle (Ferbort et al., 1992) (Figure 8.7).

It is most likely that the inhibition is mediated via the transcallosal connection and is cortical in nature. If we assume that the suppression of BGA is mainly due to cortical mechanisms, then reductions in the EMG indicate the involvement of cortical output.

With this method, Irlbacher et al. (2006) demonstrated that output from the motor cortex modulates all three phases (first agonist burst, antagonist burst, and second agonist burst) of a ballistic movement.

Conditioning of the H-reflex by Subthreshold TMS

The MEP comprises the overall effect of transmissions in excitatory and inhibitory pathways. If the investigation aims to detect adaptations of specific corticospinal pathways, special paradigms like conditioning of the H-reflex with subthreshold TMS can be used (for review see Petersen et al., 2003). The idea behind this technique is that a TMS pulse that does not evoke an MEP in the target muscle can still produce EPSPs in the spinal motor neuron pool. However, these EPSPs are not able to raise the excitation of spinal motor neurons above their threshold level. With the H-reflex timed to increase the spinal excitability at distinct times during the time course of subthreshold transmissions from the motor cortex, it is possible to separate the monosynaptic and polysynaptic effects and determine their inhibitory or facilitatory nature (Nielsen et al., 1993). As a result, the effect of subthreshold TMS on the H-reflex (suppression or facilitation of the H-reflex) can be displayed for the systematically varied ISIs (see Figure 8.8).

During a voluntary contraction, facilitation and suppression of H-reflexes at distinct ISIs have been observed without any effects on H-reflexes of similar amplitudes at rest. Moreover, it has been shown that this is only the case for MEPs evoked by TMS but not for MEPs evoked by TES, which indicates changes in excitability at the cortical level (Nielsen et al., 1993). This offers a fascinating possibility for studies that aim to investigate specific corticospinal projections during functional tasks. If TMS is adjusted to an intensity that has no effect on H-reflexes during rest, effects during a functional task should be related to changes in cortical excitability during the preparation or execution of the task. Consequently, changes in effects after training most likely reflect adaptations at the cortical level (Schubert et al., 2008).

This paradigm implies some important features for longitudinal training studies. Firstly, a comparison between rest and active conditions is included. Secondly, functional tasks can be investigated. Thirdly, changes in suppression or facilitation of the H-reflex indicate cortical adaptation (if the intensity of TMS is adjusted such that no effects occur at rest). Fourthly, the role of specific corticospinal projections can be investigated.

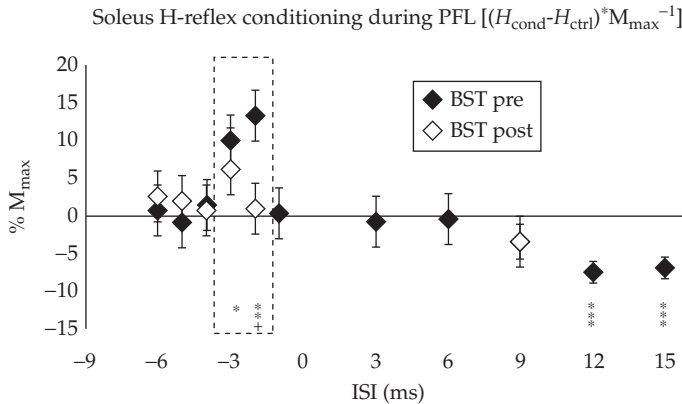


Figure 8.8 Time course of H-reflex conditioning with subthreshold TMS during a ballistic plantar flexion task before and after ballistic strength training. In this subject, short-latency facilitation occurred with ISIs at -3 and -2 ms (dotted rectangle). After training, short-latency facilitation significantly increased. ISI, interstimulus interval; PFL, plantar flexion; BST, ballistic strength training. (Modified from Schubert et al., 2008 p. 2012.)

Taube et al. (2006) used this paradigm to investigate cortical involvement in a postural task. The authors demonstrated a facilitatory effect of subthreshold TMS on the H-reflex timed in such a way that it coincided with the long-latency reflex component but not with short- or medium-latency reflex components. These results indicate a transcortical reflex loop that might play a functional role in the compensation of perturbed stance. Interestingly, in a later study, it was shown that the facilitation of the H-reflex was reduced after 4 weeks of balance training. These results indicate a reduced cortical excitability for the fastest corticospinal projections after balance training in a postural task. Even more interestingly, these reductions were correlated to improvements in balance control, whereas no correlation was found for alterations in H-reflexes with training. This suggests that predominantly supraspinal adaptations contribute to improved balance performance after training. In a comprehensive study by Schubert et al. (2008), two different training regimens were compared with this paradigm during rest and two motor tasks, each of which was similar to one type of training. The authors reported diminished short-latency facilitation of the H-reflex in the trained task (see Figure 8.8) and enhanced short-latency facilitation

of the H-reflex in the untrained task but no changes at all during rest. These results rather conclusively demonstrate that training can affect fast corticospinal projections by altering cortical influence. These adaptations were highly context specific as indicated by a significant interaction of training and task.

TMS During Movement

It has been demonstrated rather conclusively that there is a dependency between the training intervention and the motor task that is performed during testing for cortical plasticity (Liepert et al., 1998; Muellbacher et al., 2001; Jensen et al., 2005; Beck et al., 2007; Schubert et al., 2008). This is not really surprising and is well in line with observation using H-reflexes in order to test for spinal plasticity (Aagaard et al., 2002; Voigt et al., 1998). As a consequence, adaptations maybe masked if testing is not specific to the practiced movement or type and level of muscle contraction. Therefore, it seems to be crucial to match the motor condition during testing with the presumed training effects. As with other electrophysiological methods (e.g., peripheral nerve stimulation), testing under natural conditions (movement, muscle contraction) places large demands on the experiment itself, and often affords modifications to the “classical” experimental setup.

When attempting to measure corticospinal excitability during movement, the crucial point is to

ensure that the movement of the coil relative to the head is only minimal. As mentioned earlier (see Technical Background for TMS), the electrical field is complex and a small movement or tilting of the coil relative to the head may lead to a different stimulation intensity in the motor cortex, and thus to changes in the MEP. Schubert et al. (1997) introduced a harness system, the “halo vest,” to fixate the upper body and head and to provide the possibility to fixating the coil to that system. With this system, they were able to measure MEPs during walking on a treadmill and during ballistic muscle contractions while sitting on a chair (Figure 8.9a). In sport-specific movements with higher velocities and accelerations, a fixation of the upper body and head can lead to changes in the kinematics of the movement, which then influence motor control. Therefore, Taube et al. (2008) used a helmet system to stabilize the coil during DJs (Figure 8.9b). Additionally, elastic straps from the ceiling to the coil can be used in order to unload the participant’s head from the weight of the coil and allow a natural movement pattern during the experiment (Figure 8.9b). A similar unloading device was developed to stabilize the coil to measure MEPs during landing experiments in a sledge system (Avela et al., 2009; Figure 8.9c). It has to be stressed that the stability of the coil on the head is a prerequisite of any TMS measurement. In contrast to other methods like the H-reflex method, where the actual M-wave can be considered as a measure of the stimulation intensity to the nerve, with TMS it is impossible to verify a potential change in the stimulation intensity perceived by the neurons located in the motor cortex by analyzing the EMG itself. Therefore, it seems necessary to measure the movement and the tilt of the coil relative to the head throughout the measurements to be sure that it is negligible ($<2\text{ mm}$).

Conclusion

Based on the technical and physiological background of TMS, it is clear that the MEP reflects the overall excitability of the corticospinal system. However, there are several paradigms that can be used to differentiate between adaptations located at the cortical and/or at the spinal level. These

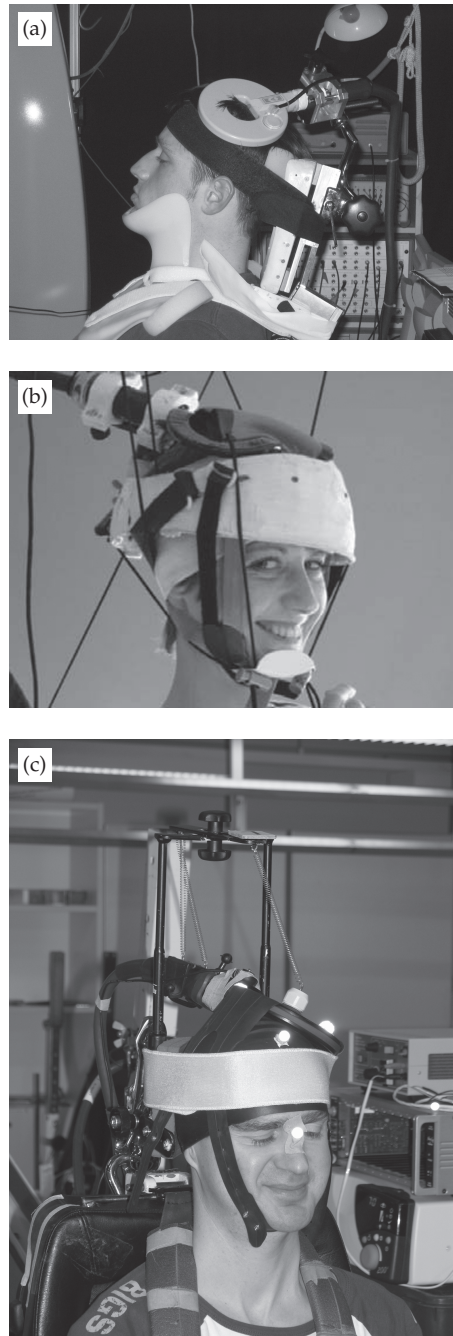


Figure 8.9 Different methods to stabilize the coil. (a) A harness system introduced by Schubert et al. (1997). (b and c) Two unloading systems to minimize the inertia of the coil during movement (Taube et al., 2008 and Avela et al., 2009, respectively).

paradigms have to be chosen carefully according to the aim of the investigation, including the experimental setup and the motor task. Thus, it is necessary to understand not only the physiology of the TMS itself, but even more importantly, the physiology of the specific paradigms.

In recent years, different studies have reported that neural adaptations maybe training and task specific. Consequently, neural adaptations should only be observed and measured in a matching task. This leads to the fact that TMS has to be used in demanding active conditions, even during dynamic movements, which further increases the technical demands of TMS measurements. Proper fixation of

the coil throughout the task is naturally an important prerequisite for the interpretation of the data. This can be done with an off-line analysis of 3D kinematic data recorded from the coil and the head. In the future, navigational systems that currently work well in static conditions could provide online analysis of this data during the measurements, and help to enhance the benefit of TMS as a tool to study the role of the motor cortex in human muscle function.

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Chapter 9

Contribution of In-Vivo Human Tendon Force Measurements for Understanding Tendon-muscular Loading During SSC

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Introduction

Measurements with isolated muscle preparations and also laboratory tests in humans can give the basis for understanding the mechanical behavior of skeletal muscle. Information given in Chapter 2 is primarily based on these types of measurements and experimental setups. Unfortunately, however, the human movement is often very complex and cannot be correctly characterized through information received from isolated muscle preparations. At the end of Chapter 2, it was concluded that while human locomotion involves application of several types of muscular contractions—*isometric, eccentric, and concentric*—the knowledge and understanding of the interaction between nervous input (and feedback in the form of reflexes) and mechanical output from the MTU is a major challenge for researchers in this field. In particular, there is a need to be able to capture exactly and continuously the loading of the various musculoskeletal structures in the human body. Especially relevant in this regard has been a need to record continuously the loading of an individual tissue (e.g., tendon or ligament) in its natural environment,

that is, when it is functioning together with other similar or different (e.g., muscle) tissues. It is very likely that the loading (and function) of a tissue maybe very different, when one compares the “natural” setting with that of an isolated tissue loading. The natural setting involves not only different structural elements, but also interference from the nervous system (e.g., sensory input with various connections, variable central command to the muscles, and so on). Tendons are of particular interest, because besides being an important part of the MTC (or MTU), its chief functions during locomotion are (1) to transfer force produced by the CC to the joint and/or bone connected in series, and (2) to save mechanical energy coming from the human body as strain energy. In addition, tendon mechanics may influence running economy, sprint performance, and dynamic stability control after sudden postural perturbations (Arampatzis et al., 2009). Muscles are usually interacting with each other as a group (e.g., synergists) or antagonists and their individual contribution is often difficult to assess or measure. Thus, it is no wonder that forces produced by individual muscular-tendon structures have attracted interest among researchers in motor control, muscle and neurophysiology as well as in musculoskeletal biomechanics.

In order to obtain values for these forces, both indirect and direct measurements have been applied, especially for characterizing the force

contribution of the muscles in a specific movement. If we consider the loading of the triceps surae MTC as an example, two possibilities are available and have been applied in human locomotion: (1) force levels of the specific muscle can be estimated mathematically with the help of force platforms and video (film) analysis, (2) forces can be recorded directly by applying suitable transducers around the tendon. The former approach is indirect but useful, especially in static loading situations, but its use for fast, dynamic-type movements may sometimes prove problematic. An example of this indirect but successful approach can be taken from the study of Scott and Winter (1990) who described an AT model and its use to estimate AT loading during the stance phase of human gait. EMG is another method that has been used to estimate loading of individual human skeletal muscles. The method may sound promising, especially because a linear or slightly curvilinear relationship can be established between EMG activity and muscular force (Bouisset, 1973; Komi & Buskirk, 1972). Unfortunately, however, EMG is a very poor predictor of continuous force record, because it is very sensitive to the types of muscle action (isometric, eccentric, concentric) and to the velocity of action in dynamic situations (Komi, 1983). The list of problems increases, when one considers the fact that EMG is also dependent on muscle length and very adaptable to training and detraining (Häkkinen & Komi, 1983) as well as to fatigue (Komi, 1983). In certain dynamic situations such as hopping, EMG maybe momentarily silent while considerable force can be recorded from AT at the same time period.

The direct in vivo registration has been applied quite frequently in animal experiments and it has produced considerable information of the mechanical behavior of certain tendons, e.g., AT during cat locomotion (Walmsley et al., 1978). In our Biomechanics laboratory in Jyväskylä University, we were fortunate to have had possibilities to develop these direct in vivo transducer techniques for human subjects and to obtain records from very natural locomotion performed at different intensities, often up to very maximum effort.

Methodological Development

As is often the case in technological developments of measuring devices, the tendon transducers were first developed and used in animal experiments. Salmons (1969) was the first to introduce the design of the buckle-type transducer for recording directly in vivo tendon forces in animals. Since these pioneering efforts, it took, however, a considerable time before the first in vivo tendon forces were measured in humans. Further technical developments for direct tendon force measurements resulted later in introducing a new approach, namely, the OF technique (Komi et al., 1996). This chapter deals with studies performed with these two methods, buckle transducer and OF transducer. The series of studies with these methods have produced information that has helped to understand the mechanics of individual muscles and tendons (and ligaments) in vivo. This chapter is an updated version of our earlier publications of similar nature (Komi et al., 1992; Komi & Ishikawa, 2007).

The Buckle Transducer

The application of an in vivo measurement technique required several stages of development and trials with animals (for details, see Komi et al., 1987). These stages included such technical questions as transducer designs, details of surgical operation, and duration of implantation. These features were then followed by an experiment with a human subject in whom the E-form transducer was implanted around AT and kept in situ for 7 days before the measurements were performed during slow walking (Komi et al., 1984). This type of transducer did not, however, satisfy the requirements of the pain-free, natural locomotion, and the transducer design was changed to Salmons's original buckle type (Salmons, 1969).

The design of the transducer has previously been presented in detail (Komi, 1990; Komi et al., 1987, 1992). The transducer consists of a main buckle frame, two strain gauges, and a center bar placed across the frame (Figure 9.1). The frame and the center bar are molded from stainless steel. Three

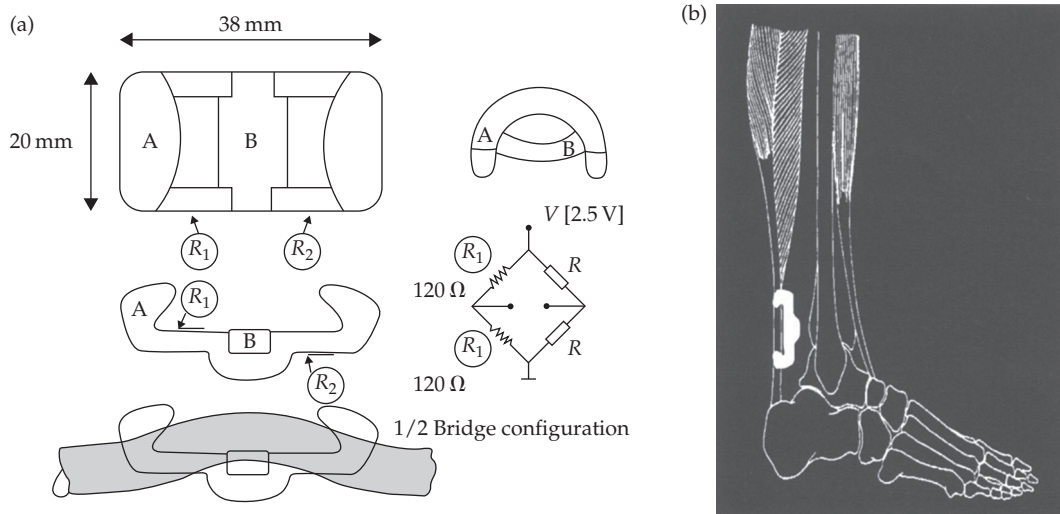


Figure 9.1 (a) Schematic presentation of the “buckle”-type transducer designed for experiments in which human subjects could perform even maximal activities, e.g., in running and jumping. (A) Main buckle frame; (B) crossbar. R_1 and R_2 = resistors of the $\frac{1}{2}$ Wheatstone bridge configuration. The lower part demonstrates schematically (and with slight exaggeration) the bending of the AT when the transducer is in situ. (b) Schematic presentation of the buckle transducer implanted around the AT. (Komi et al., 1987.)

different kinds of frame are available, and each frame has three different kinds of crossbar. The differences in frame size and crossbar bending ensure that a suitable transducer is available for almost any size of adult human AT. To assist in the selection of the best possible transducer size, a roentgenogram of ankle of the subject (lateral view) is obtained before surgery. The final selection is performed during the operation, when the tendon is visible and can be easily palpated and the thickness measured again. The transducer shown in Figure 9.1 has a frame of middle size (38mm in length, 20mm in width, and 13.5mm in height).

The transducer is implanted under local anesthesia. During the surgery, which lasts 15–20 min, the subject is in a prone position on the operating table. To provide normal proprioception during movements, local anesthesia is not injected into the tendon or muscle tissue. Evidence has been presented that when it is injected directly into the muscle, the myoelectric response of the muscle could be affected (Sabbahi et al., 1979). An incision of

approximately 50mm in length is made on the lateral side just anterior to the tendon to avoid damage to small saphenous veins and the sural nerve. The size of the buckle is matched with that of the tendon. The correct-sized crossbar is then placed under the tendon into the slots of the frame. This causes a small bend in the tendon, as demonstrated schematically in Figure 9.1. The cable containing the wires from the strain gauges is threaded under the skin and brought outside approximately 10cm above the transducer. After the cut is sutured and carefully covered with sterile tapes, the cable of the transducer is connected to an amplifying unit for immediate checkup. Figure 9.1 demonstrates a lateral radiographic view of the transducer in situ.

Calibration of the transducers is performed immediately prior to the experiments. In contrast to animal studies, an indirect method must be applied to calibrate the AT transducer placed on the human subjects. It is performed on a special calibration table where both static and dynamic loads can be applied. The details of the calibration

have been explained (Komi et al., 1987), and some critical aspects of the procedure were subsequently discussed (Komi, 1990).

Application of the Buckle Transducer in Different Locomotor Activities

Use of the buckle transducer in the study of AT force measurements produces important parameters such as peak-to-peak force and RFD which can then be used to describe the loading characteristics of the tendon under normal locomotion. When these parameters are combined with other external measurements, such as cinematography for calculation of MTC length changes, the important

concepts of muscle mechanics, such as instantaneous length–tension and F–V relationships, can be examined in natural situations such as SSC activities (Fukashiro & Komi, 1987; Komi, 1990; see also Table 9.1). Simultaneous recording of EMG activities can add to the understanding of the force potentiation mechanism during SSC-type movement.

Walking and Running

The first measurements with the E-form transducers demonstrated that during slow walking, the AT force builds up before the heel contact on the ground, but it is then suddenly released for 10–20 ms during early impact. Thereafter, the force

Table 9.1 In vivo tensile measurements during natural human locomotion

References	Year	Tendon	Transducer type	Movement(s)
Komi et al.	1984	Achilles	E-form (invasive)	Slow walking
Gregor et al.	1984	Achilles	Buckle (invasive)	Cycling
Komi et al.	1985	Achilles	Buckle (invasive)	Walking (1.2–1.8 m/s) and running (3–9 m/s)
Komi	1990	Achilles	Buckle (invasive)	Walking (1.2–1.8 m/s), running (3–9 m/s), and jumping
Gregor et al.	1991	Achilles	Buckle (invasive)	Cycling
Komi et al.	1992	Achilles	Buckle (invasive)	SJ, CMJ, hopping
Fukashiro et al.	1993	Achilles	Buckle (invasive)	SJ, CMJ
Fukashiro et al.	1995	Achilles	Buckle (invasive)	SJ, CMJ, hopping
Nicol et al.	1995	Achilles	Optic (invasive)	Ankle dorsiflexion reflex
Komi et al.	1995	Achilles, Patella, Biceps brachii	Optic (invasive)	Isometric plantar flexion, Isometric knee flexion, Isometric elbow flexion
Finni et al.	1998	Achilles	Optic (invasive)	Walking (1.1–1.8 m/s)
Arndt et al.	1998	Achilles	Optic (invasive)	Isometric plantar flexions
Nicol and Komi	1998	Achilles	Optic (invasive)	Passive dorsiflexion stretches
Finni et al.	2000	Achilles, patella	Optic (invasive)	Submaximal SJ and CMJ
Finni et al.	2001a	Achilles, patella	Optic (invasive)	Hopping
Finni et al.	2001b	Achilles	Optic (invasive)	Submaximal SJ, CMJ, and DJ
Finni et al.	2001c	Achilles	Optic (invasive)	Maximal knee extension (SSC), CMJ, and rebound jump
Finni and Komi	2002	Achilles	Optic (invasive)	Submaximal SJ and DJ
Finni et al.	2003a	Achilles	Optic (invasive)	Knee extension (eccentric and concentric)
Kyröläinen et al.	2003	Patella	Buckle (invasive)	Running (3–5 m/s) and long jump
Ishikawa et al.	2003	Achilles	Optic (invasive)	DJ, SJ
Ishikawa et al.	2005	Patella	Optic (invasive)	Walking (1.4 m/s)
Froberg et al.	2009	Achilles	Optic (invasive)	Walking

CMJ, countermovement jump; DJ, drop jump; SJ, squat jump; SSC, stretch-shortening cycle.

builds up at a relatively fast rate and reaches the peak at the end of the push-off phase. Figure 9.2 gives an example from the measurements with the buckle transducer while the subject walked on the long force platform at various constant speeds ranging from 1.2 to 1.8 m/s.

Each individual curve represents an averaged curve of a minimum of four ipsilateral contacts.

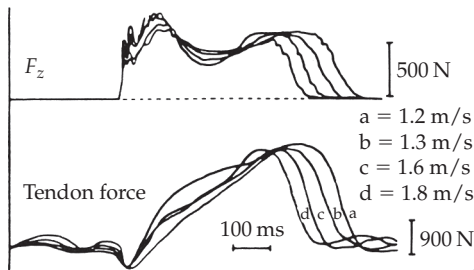


Figure 9.2 ATF curves during walking at different speeds. The beginning of the upward reflection of the ground reaction F_z force curve shows the heel contact. (From Komi et al., 1992, with permission.)

Figures 9.2 and 9.3 also demonstrate the occurrence of the slack in ATF upon the heel contact. The heel contact occurs at the instant of increase in the vertical (F_z) and horizontal (F_y) curves of the force platform reaction forces. The peak-to-peak amplitude of ATF reaches the value of 2.6 kN corresponding to 5.9 kN/cm² when the cross-sectional area (CSA) of the AT was 0.44 cm² in this particular subject. This peak value seems to be similar across all speeds, whereas the rate of ATF development is sensitive to speed so that it increases at faster walking.

Running with the heel contact demonstrates similar quick release of ATF upon early impact as in walking. This coincides with the release of EMG activation of the TA muscle, resulting in a reduction in stretch of the AT. Consequently, the velocity of plantar flexion is momentarily greater than the shortening velocity of the active SOL and GAST muscles. On the other hand, running with the ball contact (Figure 9.3) gives slightly different ATF response. The quick release of the force is almost absent in these curves.

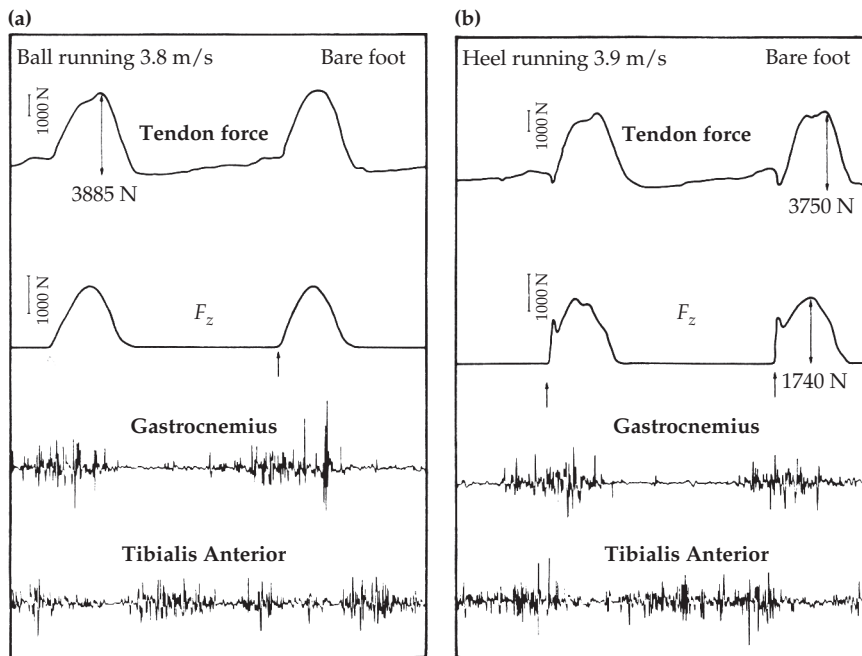


Figure 9.3 Example of the barefoot running with ball (a) and heel (b) contacts. Note the sudden release of tendon force upon heel contact on the force plate. (From Komi, 1990, with permission.)

When the ATF measurements are performed simultaneously with recordings of EMG, cinematography, and GRFs, the resulting information gives quite a strong evidence to demonstrate the SSC muscle function for the entire MTU. Figure 9.4 is a typical example of such a recording. The figure shows several important features of the AT loading in moderate speed running. First, the changes in the muscle–tendon length (segment length) are very small (6–7%) during the braking phase of the ground contact. This suggests that the condition favors the utilization of short-range elastic stiffness (SRES) (Rack & Westbury, 1974) in the muscle. Various length changes are reported in the literature demonstrating that the effective range of SRES in vitro preparation is 1–4% (Wickiewicz et al., 1983; Perttunen et al., 2000). In the intact muscle length, in vivo, this value is increased because series elasticity and fiber geometry must be taken into account. This could then increase the muscle–tendon lengthening to 6–8%. When measurements are made at the muscle fiber level, the values could be naturally smaller, as shown by Roberts et al. (1997) in turkey's running on ground level.

The loading of AT has been usually characterized by the magnitude of the peak ATF. Figure 9.4 (top) gives an example of one subject, who ran at different constant speeds. The highest maximum ATF has been attained already at a speed of 6 m/s, in which case the value was 9 kN corresponding to 12.5 body weight. When the CSA of the tendon was 0.81 cm^2 , the peak force for this subject was $11.000 \text{ kN} \times \text{tensile strength}^2$ and also higher than those observed by Scott and Winter (1990) from their AT model calculations.

It must be noted, however, that at the same speed of running the different subjects seem to obtain different ATFs. Thus, no representative average ATF values can be presented. The Scott and Winter (1990) model provided similar observations as those in Figure 9.4 that the greatest tendon force was not obtained at the highest speeds. Thus, the plateauing of ATF after 6 m/s in Figure 9.5 suggests reconsidering the importance of the force magnitude to characterize the loads imposed on

the AT at various conditions. Figure 9.5b demonstrates that the highest rate of ATF development increased in both running conditions linearly with the increase in running speeds. Consequently, instead of emphasizing the magnitude of forces only, it maybe more relevant for the discussion of tissue loading in living organisms to look at the rates at which the forces are developing at particular loading phases.

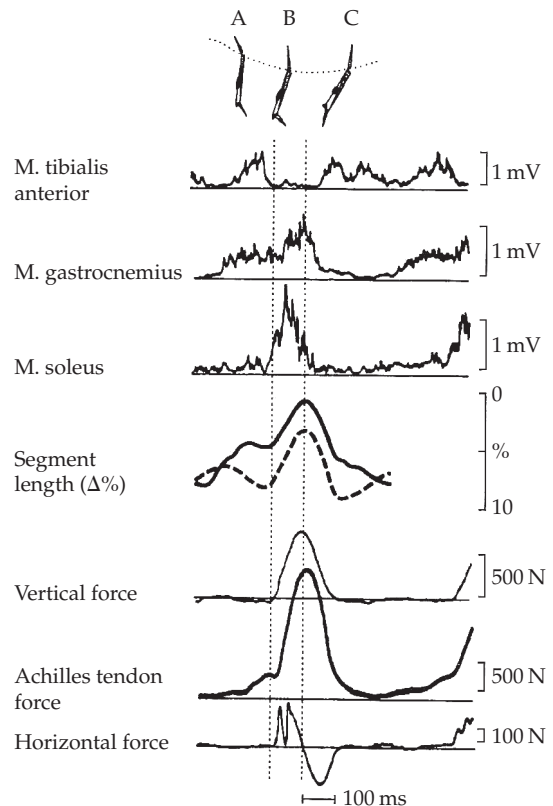


Figure 9.4 Demonstration of SSC for the triceps surae muscle during the (functional) ground contact phase of human running. Top: Schematic position representing the three phases of SSC (see Figure 3.2). The rest of the curves represent parameters in the following order (from top to bottom): Rectified surface EMG records of the TA, GAST, and SOL muscles, segmental length changes of the two plantar flexor muscles, vertical GRF, directly recorded ATF, and the horizontal GRF. The vertical line signifies the beginning of the foot (ball) contact on the force plate. The subject was running at moderate speed. (From Komi, 1992, with permission).

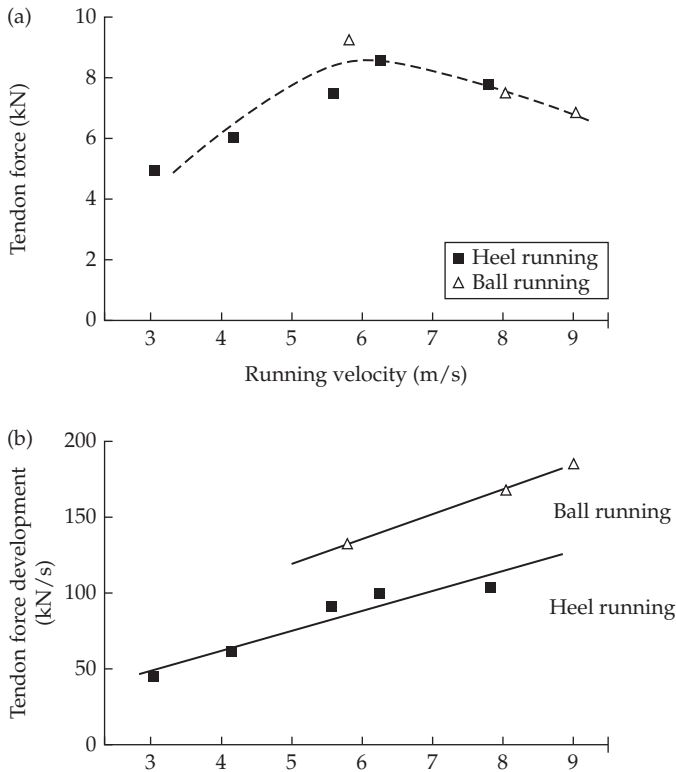


Figure 9.5 Peak tendon forces (a) and peak rate of tendon force development (b) for one subject running at different velocities. (From Komi, 1990, with permission.)

Hopping and Jumping

Loading of individual MTC is naturally dependent on how specifically the movement influences the respective joints. For example, vertical jump on a force platform can be performed in different ways, and one could expect the ATF to become also correspondingly differently loaded. There are three examples of jumps and hops on the force platform that have been used in connection with the ATF measurements: (1) maximal vertical jump from a squat position without countermovement. This condition is performed as a pure concentric action and is called SJ (Komi & Bosco, 1978); (2) maximal vertical jump from an erect standing position with a preliminary countermovement. This jump is called a CMJ (Komi & Bosco, 1978); and (3) repetitive submaximal hopping in place with preferred frequency.

Figure 9.6 presents the results from a representative subject who performed these jumps while keeping his hands on his hips. It is noteworthy from this figure that although the peak ATFs in maximal SJ and maximal CMJ were 2.2 and 1.9 kN, the respective value in the submaximal hopping was much higher, 4.0 kN. Hopping is characterized by the large mechanical work of the ankle and quite small work of the hip joint (Fukashiro & Komi, 1987). It can be further suggested that the submaximal hopping with preferred frequency demonstrates great use of the elastic potential of the plantar flexor muscles.

Long Jump

The long jump is expected to be an activity where the joints, muscles, and tendons are highly loaded. Much to our surprise, the peak-to-peak ATF

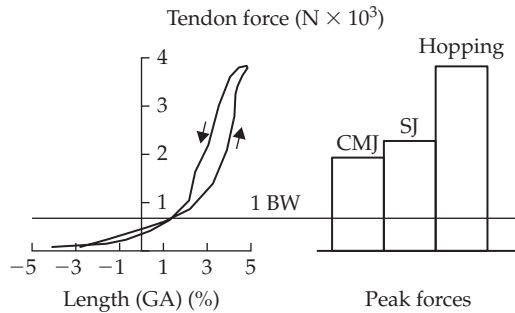


Figure 9.6 Peak ATFs of a subject performing maximal counter movement (CMJ) and squatting (SJ) jumps as well as submaximal hopping. The left side of the figure gives the F–L relationship during hopping for the contact phase on the ground. The length axis refers to the gastrocnemius (GA) muscle. BW, body weight. (From Komi et al., 1992, with permission).

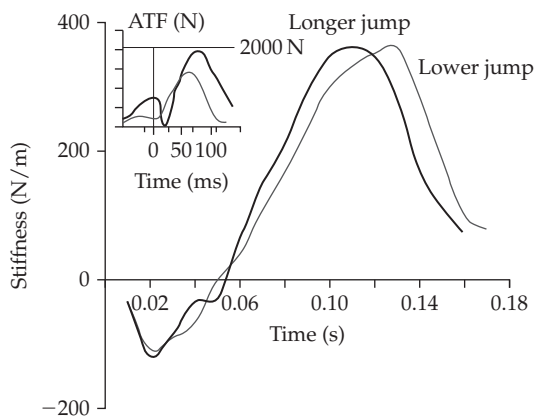


Figure 9.7 The tendomuscular stiffness–time curves of the AT–triceps surae muscle complex during the take-off contact phase in the long jump measured in the successful (longer jump) and less successful (lower jump) attempts. The insert shows the ATF–time curves for the successful jump (thick line) and as a comparison for a submaximal running (gray line) (5 m/s). The vertical line denotes the beginning of the ground contact. Note the dramatic ATF reduction immediately after the heel contact of the long jump. (From Kyröläinen et al., 2003.)

values were relatively low (~ 2000 N) at the end of the braking of the long-jump take-off motion (Figure 9.7). The subject was an experienced long jumper. More importantly, however, the sudden release of ATF upon heel contact was very dramatic, especially in the near-maximal effort condition.

Cycling

Most of the previous examples of activities for which ATF had been recorded represented situations in which the impact and braking phases were of considerable magnitudes. Except for the SJ, the AT and its muscular component acted in an SSC (Komi, 1984) in which the stretching of an active muscle (eccentric action) is followed by a shortening phase (concentric action). Cycling, on the other hand, is regarded as an activity in which musculoskeletal injuries in lower extremity are rare and the loads in AT are also relatively small.

In our example, a middle-aged male subject pedaled on a bicycle (Figure 9.8) at three different workloads: (1) 1 kp at 90 rpm (88 W); (2) 3 kp at 60 rpm (176 W); and (3) 3 kp at 90 rpm (264 W). The results indicated that the maximum ATF increased 38% from 480 N at 1 kp (90 rpm) to 661 N at 3 kp (90 rpm). However, no increase in peak force was observed as a result of increased pedaling cadence. In all cases, peak ATF was recorded at 115° in the pedaling cycle of this subject. Figure 9.8 presents average records of five pedaling cycles at 265 W, and is representative of the patterns observed at the other two work loads. These observations demonstrate relatively low loading of AT in these submaximal cycling exercises. In fact, ATF buildup occurred quite smoothly, but the EMG pattern as well as the length changes in Figure 9.8 suggests that even in cycling it is possible for muscles to act in SSC. Its consequences for the elastic potentiation and related other features for muscle mechanics have been discussed in detail elsewhere (Gregor et al., 1991).

Advantages and Disadvantages of the Buckle Transducer

The major advantage of the direct in vivo measurement is its possibility to have continuous recording of AT force (ATF), which is also immediately available for inspection. The second important feature in this measurement approach is the fact that several experiments can be performed in one session and the movements are truly natural. The buckle transducer method is naturally quite invasive, and may receive objections for use by the ethical committee in question. Due to the relatively large size of the

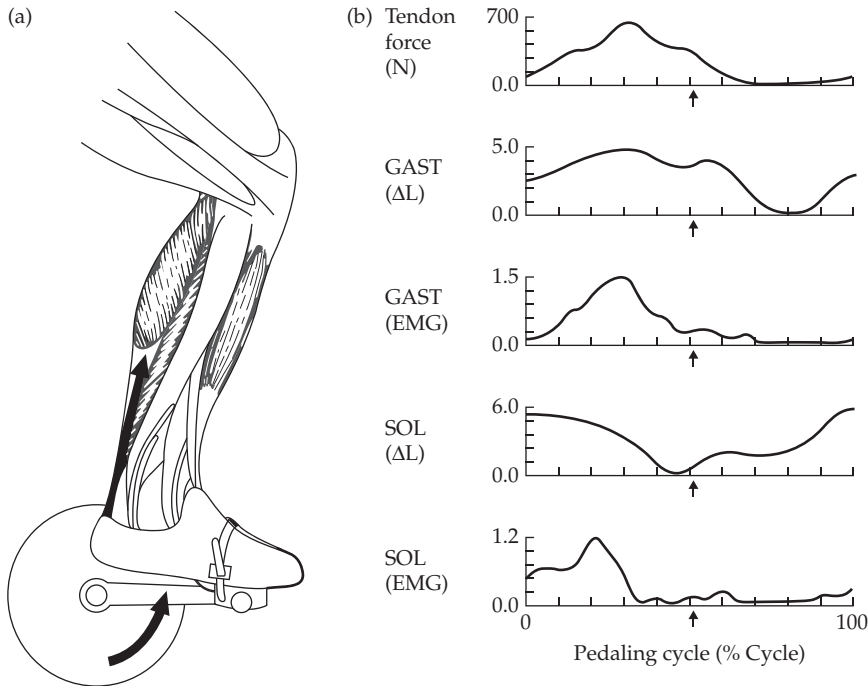


Figure 9.8 (a) Schematic illustration indicating the major skeletal muscles and measurement of ATF acting on the lower extremity during cycling. (b) ATF pattern, gastrocnemius (GAST) and soleus (SOL) muscle length change patterns, and SOL and GAST, electromyographic (EMG) patterns for one subject (average of five pedaling cycles) at 265 W (90 rpm at 3 kp). Arrow indicates BDC. (From Gregor et al., 1987, with permission.)

buckle there are not many tendons, which can be selected for measurements. AT is, however, an ideal one due to large space between the tendon and bony structures within the Karger triangle. Other restrictions in the use of this method are difficulties in calibration procedure and problems in the application of the technique when long-term and repeated implantation maybe of interest. As is the case in animal experiments, the buckle transducer method cannot isolate the forces of the contractile tissue from the TTs. The method can therefore be used to demonstrate the loading characteristics of the entire MTC only.

OF Technique

In order to overcome some of the disadvantages of the buckle transducer technique, an alternative method was then developed. As was the case for the buckle method, this new OF technique was also first applied to animal tendon (Komi et al., 1996). However, it had already earlier been applied with success as a pressure transducer in sensitive skin application (Bocquet & Noel, 1987) and for

measurement of foot pressure in different phases of cross-country skiing (Candau et al., 1993). The measurement is based on light intensity modulation by mechanical modification of the geometric properties of the plastic fiber. The structures of optical fibers used in animal and human experiments (Komi et al., 1996; Arndt et al., 1998; Finni et al., 1998, 2000) consist of two layered cylinders of polymers with small diameters.

The principle of this method is as follows: when a thin OF is bent or compressed, the light can be reduced linearly with pressure and the sensitivity depends on fiber index, fiber stiffness, and/or bending radius characteristics. Figure 9.9 characterizes the principle of the light modulation in the two-layer (cladding and core) fiber when the fiber diameter is compressed by external force. The core and cladding will be deformed and a certain amount of light is transferred through the core-cladding interface. In order to avoid the pure effect of bending of the fiber, the fiber when inserted through the tendon (Figure 9.10) must have a loop large enough to exceed the so-called "critical bending radius."

Both ends of the fiber are then attached to the transmitter–receiver unit and the system is ready for measurement (Figure 9.10). Calibration procedure usually gives good linear relationship between external force and OF signal. Figure 9.11a gives a representative example of such a relationship for the patella tendon measurements. The OF technique has recently been applied to study mechanical behavior of Achilles and patella tendons in several experiments. Table 9.1 includes the most important references.

Examples of Forces When Measured Simultaneously from the Achilles and Patella Tendons with the OF Technique

Fukashiro et al. (1987) demonstrated with indirect estimation that the mechanical work of knee extension and plantar flexion maybe considerably different depending on the type of activity. For example,

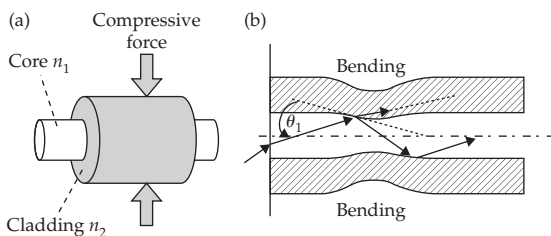


Figure 9.9 Basic principle to demonstrate how the compression on the OF (a) causes microbending (b) and less of light through the core–cladding interface.

in the CMJ, the knee extension produces greater work in CMJ, whereas the plantar flexors do the same in hopping. On tendon level, this would imply that patella tendon is more loaded in CMJ and AT more in hopping. Our measurements with the OF technique confirmed this suggestion. Figure 9.12 is from the work of Finni et al. (2000) and it shows that in CMJ, the patella tendon force (PTF) is greater than AT force and that in hopping the situation is the opposite. Secondly, when increasing the intensity of activity, PTF increased more in CMJ and AT force more in hopping.

As mentioned earlier in Chapters 1 and 2, the classical F–V relationship (Hill, 1938) describes the fundamental mechanical properties of skeletal muscle. Its direct application to natural locomotion, such as SSC, may, however, be difficult due to necessity of in situ preparations to utilize constant maximal activation. When measured in vivo during SSC, the F–V curve (Figure 9.13) is a dramatic demonstration that the curves are very dissimilar to the classical curve obtained for the pure concentric action with isolated muscle preparations (Hill, 1938) or with human forearm flexors (Wilkie, 1950; Komi, 1973).

The dashed line in Figure 9.13 shows the classical F–V curve, measured with the quick release method. The shaded area of the ATF in the submaximal hopping shows that the instantaneous F–V curve shows considerable potentiation of performance over that observed with the Hill curve.

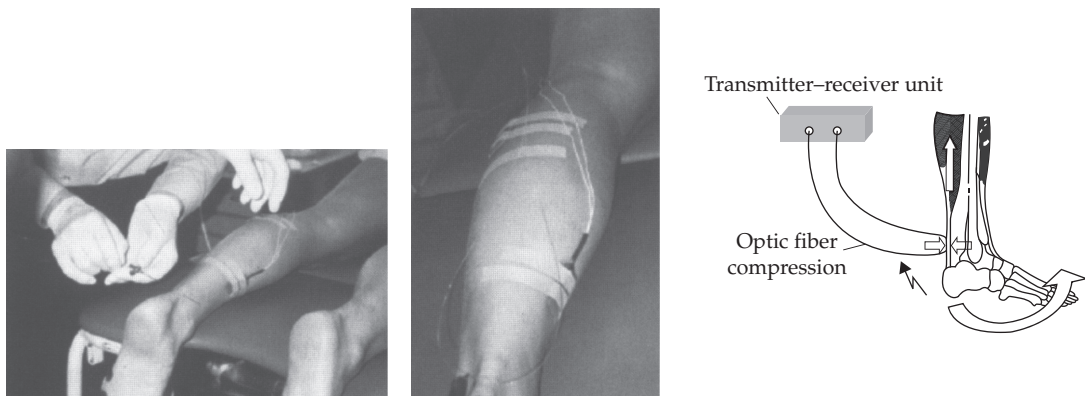


Figure 9.10 Demonstration of the insertion of the OF through the tendon. A hollow 19-gauge needle is first passed through the tendon. The sterile OF is then passed through the needle; the needle is removed and the fiber remains in situ.

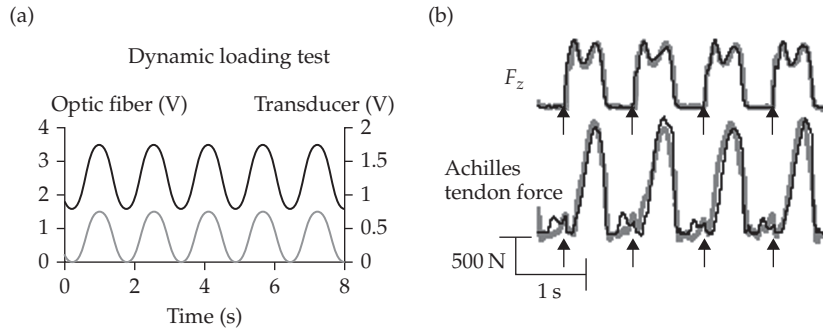


Figure 9.11 (a) Records of OF as an external force transducer during quick dynamic loading and unloading of the knee extension movement. Note how the two records follow each other. (b) AT (OF) and F_z GRFs measured for one subject while walking at the same speed at 4-month interval. Black line (first measurement); gray line (second measurement). The arrows represent the first point of heel contact with the ground.

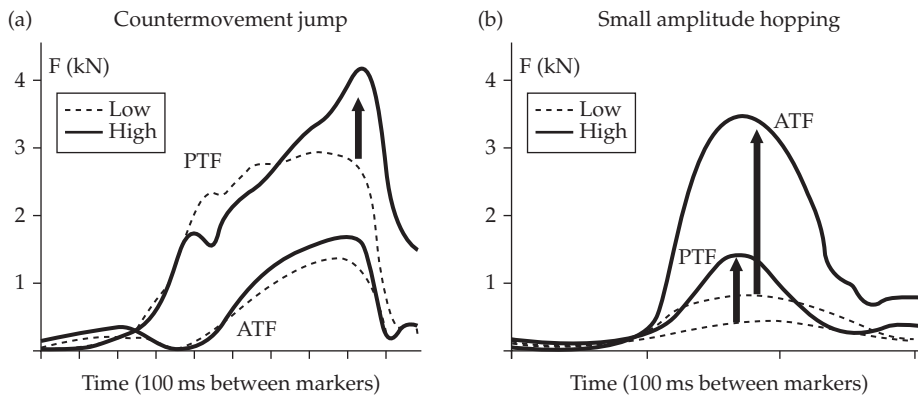


Figure 9.12 Effects of intensity of effort on tendon loading. During the large amplitude movements such as CMJ (a) or hopping with 56° knee joint displacement, the patellar tendon was primarily loaded. In small amplitude hopping with knee flexion of 23° during the ground contact phase, the AT produced greatest forces (With kind permission from Springer Science+Business Media: Finni, T., Komi, P.V. & Lepola, V. (2000) In vivo triceps surae and quadriceps femoris muscle function in a squat jump and counter movement jump. *European Journal of Applied Physiology* 83, 416–426.) (b). Increase in jumping intensity (upward arrows) did not alter the loading patterns, but the peak forces were increased. ATF, Achilles tendon force; PTF, patella tendon force. (With kind permission from Springer Science+Business Media: Finni, T., Komi, P.V. & Lepola, V. (2001a) In vivo muscle mechanics during normal locomotion is dependent on movement amplitude and contraction intensity. *European Journal of Applied Physiology* 85, 170–176.)

As is usually the case, the in vivo measurement technique for humans has been developed following reports on animal experiments (Sherif et al., 1983). Many of these animal studies have included similar parameters to those used in our human studies, such as muscle length, force, and EMG. The most relevant report for comparison with our human experiments is that by Gregor et al. (1988); they measured mechanical outputs of the cat SOL muscle during (buckle transducer technique) treadmill locomotion. In that study, the results indicated that the force generated at a given shortening

velocity during the late stance phase was greater, especially at higher speeds of locomotion, than the output generated at the same shortening velocity in situ. Thus, both animal and human in vivo experiments seem to give similar results with regard to the F–V relationships during SSC.

Important additional features can be seen in Figure 9.13. The patterns between ATF and PTF records differ considerably when the movement is changed from a CMJ to hopping. On CMJs—characterized by smaller eccentric phase—the patella tendon is much more loaded as compared to AT which

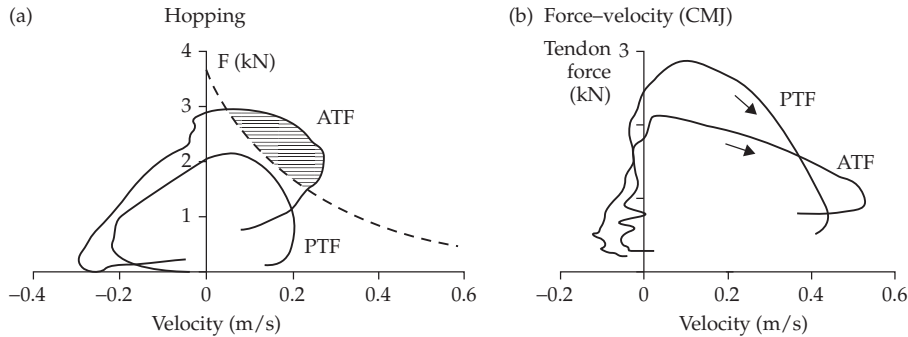


Figure 9.13 Examples of instantaneous F-V curves measured in human hopping and countermovement jumps (CMJs). (a) The records (submaximal hopping) presents greater loading of the ATF as compared to the PTF. The situation is reversed in case of CMJs (b). The records signify the functional phases of the ground contact. The left sides of both figures represent eccentric action and the right side concentric action. The dashed line signifies the F-V curve for plantar flexors measured in the classical way (From Finni, 2001).

in its turn is more strongly loaded in hopping. Thus, the muscle mechanics is not similar in all SSC activities, and generalization should not be made from one condition and from one specific muscle only. In contrast to hopping, for example, the elastic recoil of the triceps surae muscle plays a much smaller role in CMJ (Fukashiro et al., 1993, 1995; Finni et al., 1998). This is expected because in CMJ the stretch phase is slow and the reflex contribution to SSC potentiation is likely to be much less than in hopping.

One important notion of caution must be given when interpreting the muscle mechanics based on the methods shown earlier. Both in animal and human experiments—when the buckles and OFs have been applied to the tendons—the measured forces cannot be used to isolate the forces or the movements of the contractile tissue from those of the TT. The methods can therefore be used to determine the loading characteristics of the entire MTC only. It must be mentioned, however, that the fascicle F-V curves in isolated forms of maximal eccentric and concentric action resemble quite well the classical F-V relationships, and that the instantaneous F-V curve in SSC resembles that of the total MTC, but with more irregular form.

As compared to AT, patella tendon has not been frequently explored for in vivo direct measurements during human movement. There are, however, some efforts which are worth introducing here. Ishikawa et al. (2003) instructed his subject

to make maximal SJs and CMJs and submaximal DJs (the rebound jump height was equal to SJ) on a sledge apparatus. In these conditions, PTF, as measured with the OF technique, reached highest values (≈ 7 kN) in DJ. The respective values in maximal SJ and CMJ conditions were 3 kN and 6.5 kN. These movements also clearly demonstrate that the length change in MTC does not necessarily coincide with the length changes experienced by the fascicles and TTs during SSC (Fukunaga et al., 2001; Kurokawa et al., 2001; Finni et al., 2001b; Ishikawa et al., 2003). They also suggest that TT is lengthened and subsequently shortened during the contact phase in all conditions, namely, energy storage and its release from TT could occur even in SJ performance (Kurokawa et al., 2001).

Another important observation from the work of Ishikawa et al. (2003) is that during high-intensity DJs, the entire MTC showed dramatic shortening during the last movements of the push-off. This was entirely due to the quick recoil (shortening) of TT, as the fascicles changed its length only slightly during this phase. Clinical and performance-related significance of this phenomenon needs naturally further explorations.

Pros and Cons of the OF Technique

Although the OF method may not be more accurate than the buckle transducer method, it has several

unique advantages. First of all, it is much less invasive and can be reapplied to the same tendon after a few days of rest. In addition, almost any tendon can be studied provided that critical bending radius is not exceeded. This method is also quite reliable. First, as shown in Figure 9.11a, the dynamic testing shows good match between the OF and external force records. When the fiber is inserted at the several month intervals, the obtained records of the AT forces are similar as shown for walking in Figure 9.11b.

The OF technique can also be applied to measure the loading of the various ligaments. In the hands of experienced surgeon, the OF can be inserted even through deeper ligaments such as anterior talofibular ligament (ATFL) (Lohrer et al., Chapter 16). In such a case, however, special care must be taken to ensure that the OF is in contact with the ligament only and that it is preserved from interaction with other soft tissue structures by catheters.

Clinical Relevance and Future Challenges

Information available on loading characteristics on human tensile tissue in vivo conditions have revealed that in dynamic activities such as walking, hopping, and running, the tensile loading modes maybe considerably different from those measured under isometric conditions. A wide range of tensile forces (1.4–9 kN) have been measured under these conditions. The tendon force usually increases with movement intensity, but only to a certain submaximal level of effort, after which it may remain the same. However, the rate of tendon force development maybe more relevant to show the tendon loading, as this parameter increases linearly with, for example, the speed of running. Individual differences are large, and no representative values can be given for various age groups. This is partly due to the difficulties in using invasive tendon force measurements in large population studies. The obtained data is, however, important for describing the tendon loading patterns within an individual, who can perform many different activities in one measurement session lasting 2–3 h. These measurements have shown that each activity

gives a specific loading pattern, which can be modified by intensity of effort within the specific joint as well as in the neighboring joints or muscles.

These individual responses make it more challenging to characterize the loading of the tensile tissue with respect to the injury mechanisms. It is of considerable clinical relevance that rate of force increase during the braking phase of the loading is probably much more related to tissue rupture and overuse than the simple loading amplitudes. This fact also has to be taken into consideration when artificial structures are being developed to replace the tendon or ligament.

In general, however, the tendon loading during normal locomotion is very dynamic. Consequently, tendon should be seen as an “organ” which has considerable performance potential, but whose in vivo function is under influence of other factors, such as the contractile tissue of the same MTC and related sensory and motor control. It must be emphasized that the tendon does not have much functional meaning, if it were not connected anatomically with the true “motor,” the skeletal muscle. In this regard, the muscle (or its fascicles) makes it possible that TT can store considerable amount of elastic strain energy and that this energy can be utilized to potentiate the performance in the natural SSC action. It is not only the external force (e.g., gravity), however, that can regulate the TT loading. The fascicles play an important role in this task of elastic storage and utilization. As this interaction between fascicle and TT depends on several factors, such as intensity of effort (stretching and shortening velocity of the SSC action) as well as task specificity, no general rules can be written to cover the loading characteristics of TT. This is another challenge, not only for clinical work, but also for modeling of musculoskeletal function in specific activities.

Finally, there is no reason to study the tendon loading alone without considering other important parameters. Figure 9.14 is an example of our efforts to combine the different methods simultaneously in human movement studies. Combination of the fiber technique, ultrasound technique, as well as the kinematic (video) and kinetic (force platform) are very useful for objective and more comprehensive information of the factors involved.

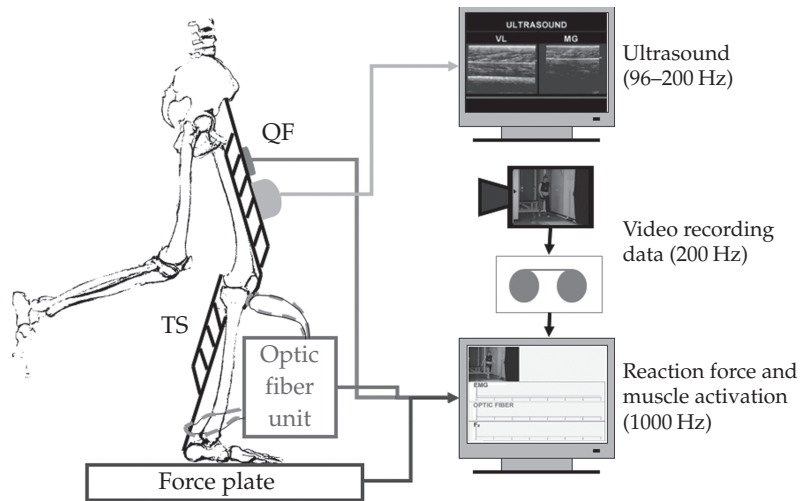


Figure 9.14 Schematic presentation of how the OF and ultrasound measurements can be combined with each other and together with other instruments. QF, Quadriceps Femoris muscle; TS, Triceps Surae muscle.

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Chapter 10

Ultrasound as a Tool to Record Muscle Function During Normal Locomotion

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Introduction

It has been frequently mentioned in the previous chapters and also demonstrated that the ability of the muscle to generate power depends on its structure. This does not refer only to the quality of the contractile elements (e.g., fast versus slow-twitch fibers), but also the size (and volume) of the muscle. The history of sport tells frequently about big, muscular wrestlers, power lifters, body builders, etc. The question of the validity of the apparently, subjective observation of muscle size and performance needed to wait for the developments of imaging techniques to make the respective assessments more reliable for living tissues.

In this regard, we refer specifically to the musculoskeletal US, which has been developed from its initial clinical interest during the early 1950s to the reliable means to record human muscle architecture noninvasively. Technical advances continue to improve the utility of ultrasound as a research technique in the muscle–tendon behavior.

Historical Development of US

Ultrasound is a unique imaging method in which the sound waves are transmitted from the transducer to the soft tissues and returned back as

echoes, which are further converted to the ultrasound images. In the musculoskeletal US, Howry and Bliss (1952) developed the brightness-mode (B-mode) displaying human skeletal structures in a 2D and sectional image. They were able to demonstrate an ultrasonic echo interface between tissues, such as that between fat and muscle, so that the individual structures could be outlined. For the purpose of understanding more of the skeletal muscle, this noninvasive ultrasound scanning has been used primarily to characterize the transverse skeletal muscle architecture in vivo (Ikai & Fukunaga, 1968; Yeh & Wolf, 1978). The study of Ikai and Fukunaga can be considered as a classical reference to all interested in learning how the ultrasound methodology started for applications to reflect the performance of the skeletal muscle. The space available for this chapter does not allow to explain all the details of their study published in 1968. Figure 10.1 gives a general view of the ultrasound scanning of the arm flexor muscle. The basic finding in their study was that there was a positive relationship between the CSA of the forearm flexors and the isometric force at certain elbow angles. However, their strength values per unit CSA (specific tension) for the flexors of the upper arm were 6.7 kg/cm² and 6.3 kg/cm² for men and women, respectively (Figure 10.2). There is not significantly any gender difference in the specific tension. When the measurements were taken with the arm in an extended position, the average value was reduced

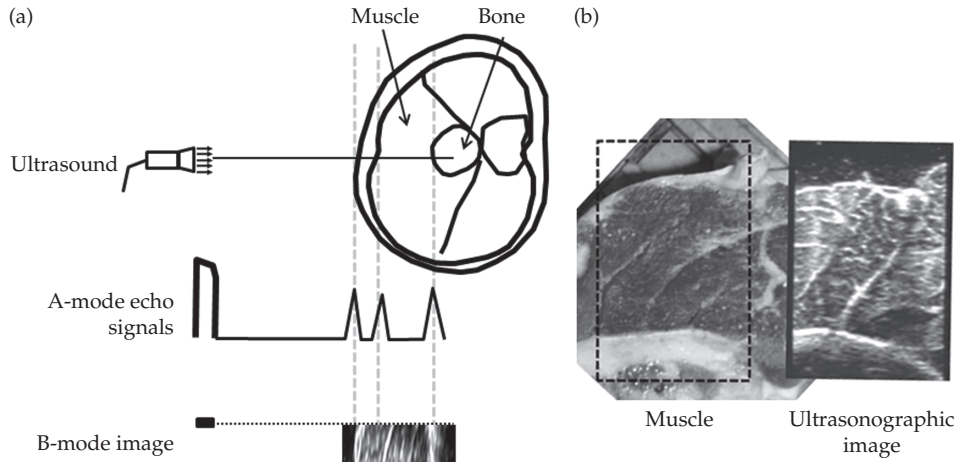


Figure 10.1 (a) Schemes of musculoskeletal ultrasonography and (b) cross-sectional view of skeletal muscle and ultrasonographic image.

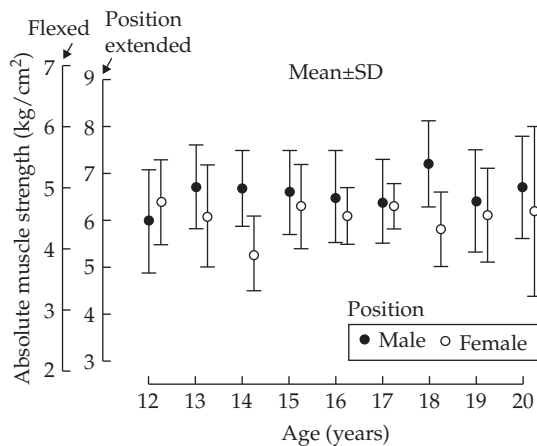


Figure 10.2 Relationship between the absolute muscle strength (specific force) of the upper arm flexors, and age and gender (Fukunaga, 1978. Used with permission).

to approximately 4.7 kg/cm². These observations have, in a broad sense, stood the test of time until today, although the current knowledge has given certain limits and boundaries for the muscle size to indicate the performance. For example, the original suggestion of Ikai and Fukunaga (1968) that the strength per CSA is independent of the training status has been questioned not only by them, but also

by many other researchers as well. It is now well established that the absolute strength can be influenced by ageing (Morse et al., 2005) and training (Reeves et al., 2004).

Since early 1990s, progress continued in the technical development. Linear array probe systems are currently capable of lateral resolution in the order of less than 1 mm, depending on the frequency of the probe (3.5–13 MHz). When the probe is correctly attached over the muscle in its longitudinal direction, the lengthening and shortening of skeletal muscles during in vivo human movements can be monitored by the B-mode sonogram gray-scale image (Fukashiro et al., 1995; Fukunaga et al., 1996). In an ultrasound image, the solid areas are generally depicted in “white” and the fluid areas in “black” (Figure 10.1). In this ultrasound scanning, muscle fascicles, which consist of the juxtaposed bundles of parallel muscle fibers with ensheathing connective tissues, can appear as dark (hypoechoic) lines lying between light (echogenic) striations of connective tissues (Figure 10.3) and run between the superficial and the deep aponeuroses. The precision, reliability, and reproducibility of this method have been confirmed in many studies (Henriksson-Larsen et al., 1992; Kawakami et al., 1993, 2000; Narici et al., 1996; Rutherford & Jones, 1992).

It must be emphasized, however, that great skill and experience is needed to place the ultrasound scanning probe correctly on the place of interest and so that it is not disturbed by skin movement during actual test locomotion. The major objective is to get clear ultrasonic echoes from interfascicle connective tissues and aponeurosis, even during explosive human dynamic movements.

Fascicle–Tendinous Behavior During Isolated Muscle Actions

Traditionally, the knowledge on human muscle architecture was based on measurements performed on cadavers. These geometric approximations follow the principle of Hill's classical model

(Voigt et al., 1995) in which muscle fibers and tendons are treated as an array of parallelograms. This method may sound convenient but is still based on many assumptions. With the ultrasound imaging techniques, it has become possible to examine directly the internal behavior of skeletal muscle during movements. For example, during isometric plantar flexion contraction, the traveling distance on the cross-point of one certain MG muscle fascicle on the deeper aponeurosis (the arrow point in Figure 10.3a) and the end-point echo of the MG tendon in the myotendinous junction (the arrow point in Figure 10.3b) during the contraction can be measured from the series of ultrasonographic images. These traveling displacements of the reference point (the arrow point in Figure 10.3) during

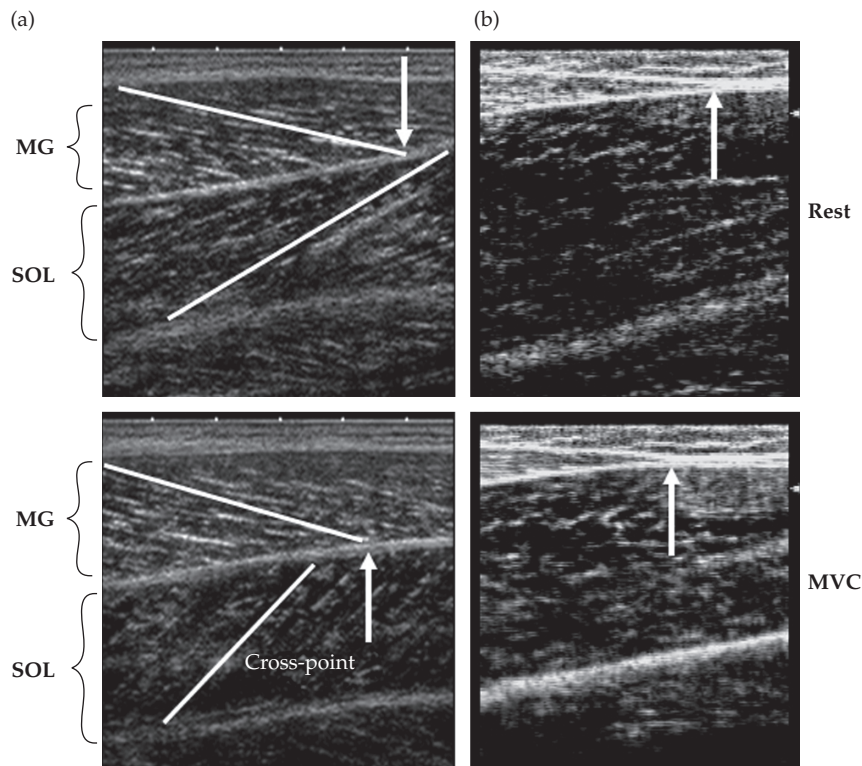


Figure 10.3 Ultrasonographic images of the medial gastrocnemius (MG) and soleus (SOL) muscles during rest (upper) and maximal voluntary isometric contraction (bottom). (a) The lines represent the muscle fascicles of the two muscles. Please note the different changes in the length of fascicles and in the pennation angles between both muscles from rest to maximal voluntary contraction. (b) The arrows show the MG myotendinous junction during rest and maximal voluntary contraction.

contraction are considered to represent the MG tendon elongation upon the loading. With the change in tensile force, which can be obtained, for example, from the direct in vivo tendon force measurements (see Chapter 9), the property of tendon parts is expressed by stress–strain or F–L relations. These controlled isometric actions are very often used as a test situation to examine the tendon hysteresis (Maganaris & Paul, 2002) (Figure 10.4) and to compare F–L and/or stress–strain curves of the

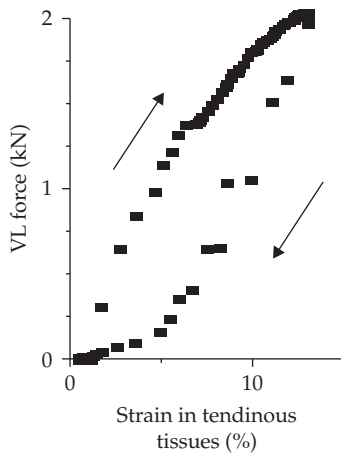


Figure 10.4 The force-strain relationship of vastus lateralis muscle (VL) during the maximal isometric knee extension. Arrows indicate loading and unloading directions.

different muscles and conditions (Kubo et al., 1999, 2001).

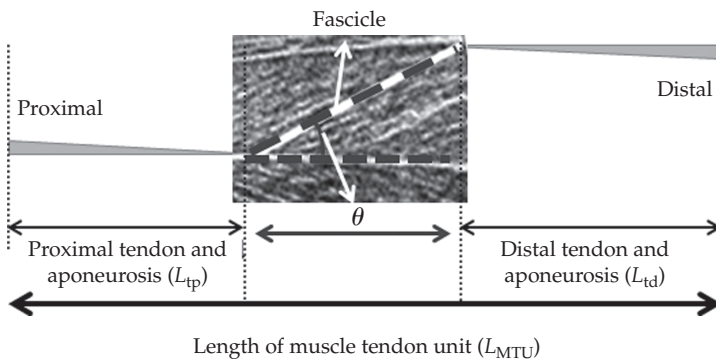
In dynamic forms of isolated muscle actions, the geometric MTU models (Zajac, 1989) are often applied during human movement conditions. In this model, the tendon length is defined as the sum of the proximal and distal tendinous structures, and aponeuroses (Fukunaga et al., 2001; Kubo et al., 2000; Kurokawa et al., 2001, 2003; Muraoka et al., 2001) (Figure 10.5). The length changes in tendon are calculated by subtracting from the MTU length the horizontal part of fascicle in the direction to the aponeurosis:

$$L_{\text{Tendon}} = L_{\text{MTU}} - L_{\text{fa}} \cdot \cos \alpha$$

where L_{Tendon} is the length of tendon parts, L_{MTU} the MTU length, L_{fa} the muscle fascicle length, and α the pennation angle in each muscle (see Figure 10.5).

The fascicle lengthening and/or shortening usually follow the basic definition of the action in question. In the eccentric and concentric actions of the knee extensors (e.g., VL), the fascicles shorten in the pure concentric action and lengthen in the pure eccentric action. On the tendon parts, however, they do not necessarily follow shortening and lengthening during these contractions, respectively (Finni et al., 2003).

Muscle fascicle–tendon model



Total tendon length = $L_{\text{tp}} + L_{\text{td}}$
(Tendinous tissues) = $L_{\text{MTU}} - \text{horizontal part of fascicle}$

Figure 10.5 Schematic model of the muscle fascicle–tendon unit. The method requires that the total MTU length is recorded and calculated continuously during locomotion (e.g. kinematics). The rest of the measurement is based on the continuous ultrasound records, such as shown in Figures 10.3 and 10.6 (see also Fukunaga et al., 2001; Kubo et al., 2000; Zajac, 1989). L_{tp} , proximal tendon and aponeurosis; L_{td} , distal tendon and aponeurosis.

Dynamic Movements Including SSC Actions

Our own work with ultrasound scanning has been more in the dynamic movement situations and especially in the manners how the fascicles and tendons behave during actual locomotor tasks, including especially SSC. By definition, SSC involves the entire MTU, although the fascicles and tendons can experience different changes in length. To catch the behavior of fascicles and tendons during rapid SSC movements, an increase in the scanning frequency of US (the usual scanning frequency is 25–60 Hz) was an important step forward in the study of how fascicles and tendons truly interact during SSC. The high-speed ultrasonographic scanning (Aloka SSD $\alpha 10$, 96–200 Hz) of muscle fascicles can help better understanding of not only the role of elastic mechanisms but also the function of muscle spindles during human SSC exercises. The preparatory muscle activation and external stretch can affect the distribution of a stretch between muscle fascicles and tendons. This fundamental question also includes the timing of the stretch to the muscle spindles that triggers the stretch reflex. In this regard, Figure 10.6 presents a situation where high-speed

ultrasound scanning of two muscles is performed while the subject is running on a 10m long force plate at various speeds. The setup is demanding because it requires two separate ultrasound machines placed on a carriage. The carriage is then pushed by the experimenter along a rail constructed at the side of the force plate system. The measurements can be combined with the recording of EMG activities of the relevant muscles and/or direct in vivo measurements of the ATFs (and PTFs). The results to be reported are very consistent and show typical intermuscular differences as well as reliable behavior of the single muscle. We present the hypotheses that, in addition to the existence of the stretch reflex potentiation during human SSC, changes in length of the fascicles and tendon, when acting together, depend on the movement task, movement intensity, and muscles involved as well as adaptation to aging and training.

Movement Specificity

As already defined in Chapter 2, SSC refers to the entire MTU so that the shortening and stretching

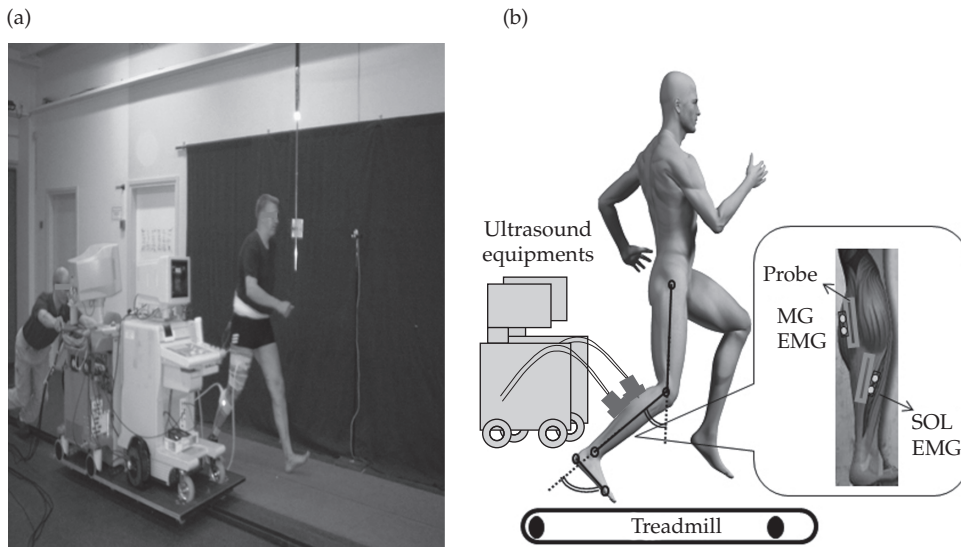


Figure 10.6 Setup for the walking and running experiments on the ground (a) and treadmill (b). MG, medial gastrocnemius; SOL, soleus; EMG, electromyogram. (From Ishikawa et al. 2007. Copyright 2006 by Elsevier B.V. Reprinted with permission; Ishikawa & Komi, 2008. Copyright 2008 by American College of Sports Medicine. Used with permission.).

do not imply events in the muscle fascicle and tendon parts of MTU. The question regarding the specific length changes in these two components is very relevant, as it may explain better the difference in behavior of the various muscles under the same movement conditions. More importantly, however, it helps to understand how fascicles and tendons interact as a function of movement specificity and intensity, for example. It has been suggested that the human skeletal muscle can adapt to the functional requirements of specific sport activities (Herzog et al., 1991), so that the working range of the sarcomere F-L relationship could be also movement task dependent. Ultrasound studies of human fascicles have indeed confirmed this hypothesis. For example, the MG fascicles most likely exhibit different patterns of behavior during walking as compared to running when examined in the same subject group (Figure 10.7).

In walking, the fascicles remain the same (Fukunaga et al., 2001), or even lengthen (Ishikawa et al., 2005a; Mian et al., 2007) during the single leg stance phase (25–75% contact period in Figure 10.7b) similarly to the MTU length changes. In running, however, the MG fascicles can shorten throughout the entire contact phase after a clear short-lasting stretch of the fascicles (see a following paragraph “Can High-Speed Ultrasonography Reveal Stretch Reflex Activity During Normal Dynamic Actions?”) even though the stretch of the MTU can be greater in terms of amplitude and velocity during running (Figure 10.7b). In addition, shortening of the MG fascicles in running is timed with increased preactivation just before the contact. The length at which the fascicles operate may thus be different in walking than in running. In fact, after the fascicle length curves begin to differ between running and walking during the

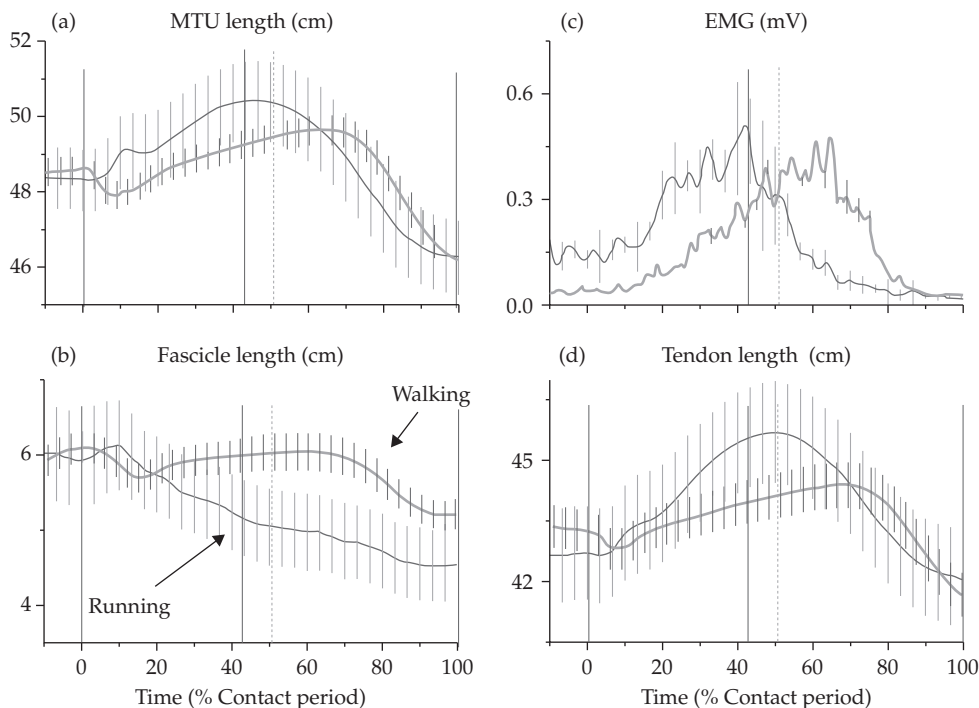


Figure 10.7 Records of MG muscle behavior during running and walking: length of the muscle–tendon unit (MTU) (a), fascicle length (b), electromyogram (EMG) (c), and tendon length (d). The vertical lines denote the contact, the transition point from the braking to push-off phases, and the take-off from the left side. (From Ishikawa & Komi, 2008. Copyright 2008 by American College of Sports Medicine. Reprinted with permission.)

MTU stretching phase (Figure 10.7b), the difference remains statistically significant toward the end of contact phase. In walking, this fascicle length could correspond to the plateau part of the sarcomere F–L relationship (Ishikawa et al., 2007). In running, it could correspond to the ascending limb of this F–L relationship (figure 3 in Ishikawa et al., 2007).

One question that has yet to be answered is why the working length of the fascicles is not the same during running and walking. The shorter fascicle length during contact phase of running implies a reduced relative force output of the fascicles. However, the shorter fascicles maybe beneficial as they result in a larger stretch of the tendon in running than in walking. The peak tendon length in Figure 10.7d was significantly greater in running than in walking. It is therefore likely that in running, where the MG fascicles are initially (at contact) very short and continue shortening during the short braking phase, the tendon stretch rate can be increased. The corresponding tendon recoil can then occur more rapidly due to the tendon's viscoelastic properties. Thus, no fixed "position" of the working length can be identified in the MG sarcomere F–L relationship for all SSC activities. It has also been observed that the MG fascicle length is dependent on running speed so that it becomes shorter with higher speeds of running (Ishikawa & Komi, 2007). This may also imply that effective utilization of tendon elasticity is regulated by the fascicle length, which in turn is under the influence of intensity of effort of a specific SSC task.

How Is the Fascicle–Tendon Interaction Related to the Intensity of SSC Task?

The finding of a change in fascicle length with different movement tasks prompts further questions concerning whether the fascicle and tendon behavior is modulated by movement efforts and intensities. When the force enhancement in SSC was introduced in Chapter 2, it was then discussed also in connection with the relationship between drop height and rebound performance (see Figure 10.8). As is clear from this figure, the performance increases as a function of the drop height until a

certain breaking point, after which the added drop height (prestetch intensity) causes the maximum jump height decrease. Reasons for this particular form of the drop height versus rebound performance curve can be at least partly sought by examining how the fascicles and tendons interact in the different DJ conditions. This may imply that fascicle and tendon interaction is modified when both impact (braking phase) loads and rebound efforts are involved, and when these parameters are varied. Several experiments were designed to examine these phenomena. Let us first look at the problem related to the drop height versus rebound performance curve.

The sledge apparatus introduced in detail in Chapter 2 was used to drop the subjects from different heights and the instruction was to perform the rebound phase with maximum effort. As expected, the drop height versus jump height curve showed the well-established relationship with the performance decreasing after a certain breaking point. Three conditions were then chosen for the comparison of the fascicle–tendon interaction: low drop, medium (optimal) drop, and extremely high drop heights. From these, the extremely high drop condition represents the reduced performance situation. Figure 10.8 gives representative examples of the fascicle–tendon behavior under these three DJ conditions. When the dropping height exceeded the optimal impact load (medium drop condition) before the rebound performance started to decrease, the MG fascicles were suddenly stretched during the braking phase (* in Figure 10.8a), indicating a loss of tolerance of the MG fascicles to the high impact loads (Ishikawa et al., 2005b; Sousa et al., 2007). In the initial braking phase of these extreme drop height conditions, tendons can still be stretched rapidly and can reach high forces (ATF: 10–12 times body weight) in the early braking phase (Ishikawa et al., 2005b). If the MG fascicles were able to tolerate this extremely high impact braking phase, the elastic energy stored in the tendon could be increased and utilized during the push-off phase, or tendons could experience structural changes due to extreme strain. However, as a result of this sudden stretch of the fascicles during the braking phase (* in Figure 10.8a), the

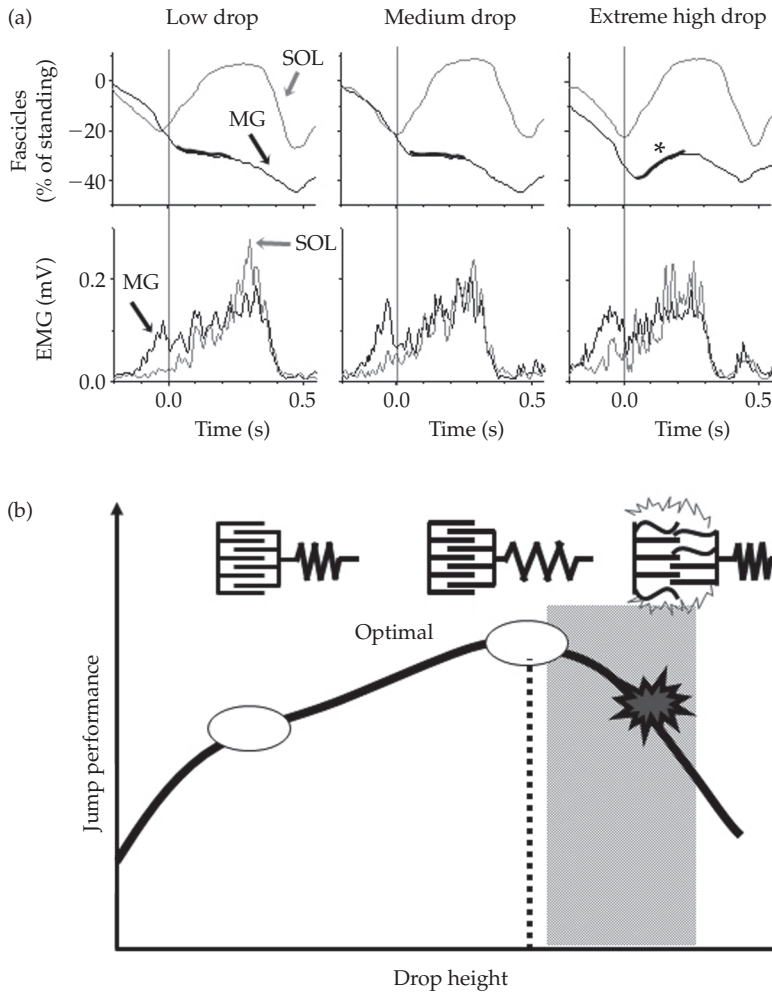


Figure 10.8 (a) Time course data of fascicles and EMGs in both MG and SOL muscles during the contact of the short contact DJs with three different drop intensities. The vertical lines indicate instants of the ground contact. (b) Relationship between drop height and rebound performance together with muscle-tendon behavior during drop jumps. MG, medial gastrocnemius; SOL, soleus; EMG, electromyogram.

elastic energy stored in the tendon can be partially lost before the start of the push-off phase. Thus, the extremely high drop conditions do not favor effective utilization of elastic energy in tendons or fascicles. When plotting the rate of change in the MG fascicle length against the ATF slope measured during the braking phase of DJs, we obtain the relationship shown in Figure 10.9. The filled circle point of this quadratic relationship maybe indicative of the critical stretch load for the MG fascicles to utilize tendon elasticity effectively (Ishikawa et al., 2005b; Sousa et al., 2007). Figure 10.9 makes additional efforts to characterize this important finding.

The precise cause of this sudden MG fascicle stretch during the high impact braking phase remains to be determined. Possible reasons for this behavior maybe as follows: (1) the stretch load is so mechanically high and rapid that actin-myosin cross-bridge interactions are simply detached or slip apart, (2) inhibitory responses (e.g., GTO) aimed at injury protection may become more dominant, (3) increased central input may cause presynaptic Ia afferent inhibition as a protective strategy to prevent tendomuscular injury due to the high stretch load. All of these mechanisms maybe operative either together or independently during extremely high impact SSC exercises.

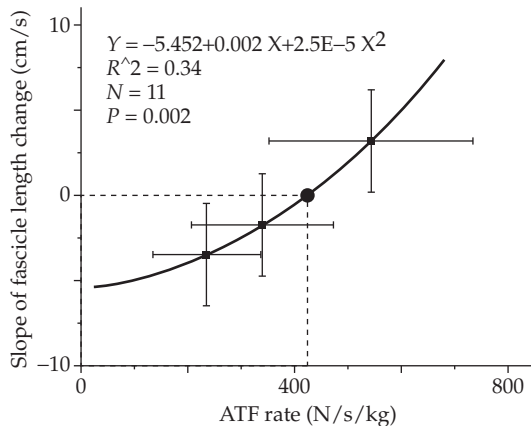


Figure 10.9 Relationship between the slope of MG fascicle stretching and ATF rate according to body mass. The filled circle corresponds to the point where the rate of the MG fascicle stretching is zero during the braking phase, and it can be considered as the point of the critical stretch load for the MG fascicles to use tendon elasticity effectively ATF, Achilles tendon force. (From Ishikawa et al., 2005. Copyright 2005 by The American Physiological Society. Reprinted with permission.)

Effects of Variation of Both the Impact and Push-Off Intensities

The fascicle–tendon interaction in the controlled sledge DJs can also be examined from the point of view of varied muscle activation. When the dropping height was varied and the subsequent jumping height was kept constant (Figure 10.10a) (Ishikawa & Komi, 2004), the increased impact force resulted in shorter fascicle lengths of the examined VL muscle at the constant MTU stretching point, with greater EMG activity during the braking phase. It also resulted in an increase in the amplitude and rate of tendon stretch, even when the stretch of the MTU was similar. The subsequent constant rebound performance was then associated with an increase in tendon recoil and reductions of EMG activity and fascicle shortening work during the push-off phase. Thus, in this experimental setup, tendons demonstrated shortening as a function of the preceding intensity of the impact loads. At the fascicle level, the push-off phase was characterized by the decreased EMG activities with the corresponding lengthening of fascicles in the higher drop height condition (Figure 10.10a). This pattern of the fascicle–tendon

behavior during the push-off phase is in line with the concept of “timing of the muscle lengthening for effective release of elastic energy” proposed by Ettema (1996). This could be interpreted to mean reduction of fascicle work. Reduction of fascicle work with a concomitant increase of the storage and release of elastic energy in the tendons can explain how an increased impact load affects the efficiency of the push-off phase of SSC exercises.

Effects of Push-Off Intensity on Fascicle–Tendon Interaction

When DJs were performed from a constant drop height with variations in the subsequent push-off (rebound) heights (Ishikawa et al., 2003), the pre-activation and initial impact forces were similar due to the constant drop height (Figure 10.10b). However, as the rebound height was increased up to maximal efforts, the EMG activation increased from the late braking phase, corresponding to the smaller fascicle stretch. This resulted in additional tendon stretch and recoil (Figure 10.11). This emphasizes the fact that the external stretch load in the braking phase is not the only factor that determines fascicle and tendon behavior during human SSC exercises. These results clearly indicate the existence of the push-off intensity-specific interactions between fascicles and tendons.

Muscle Specificity

The preceding discussions of fascicle and tendon behavior during SSC exercises may seem relatively uncomplicated. Unfortunately, the observations presented earlier cannot be generalized to the function of all skeletal muscles. In the same motor task, the muscles of the same and/or different joints may not behave similarly in terms of fascicle and tendon behavior. As already mentioned, the lengthening or shortening of the MG fascicles during SSC exercises can be varied in order to utilize tendon elasticity effectively, and to facilitate protection from injury. In contrast, during the same SSC exercises, the fascicles of the synergistic SOL muscle exhibited a stretch-shortening behavior during the contact phase (Figure 10.8) (Sousa et al., 2007). When the DJ

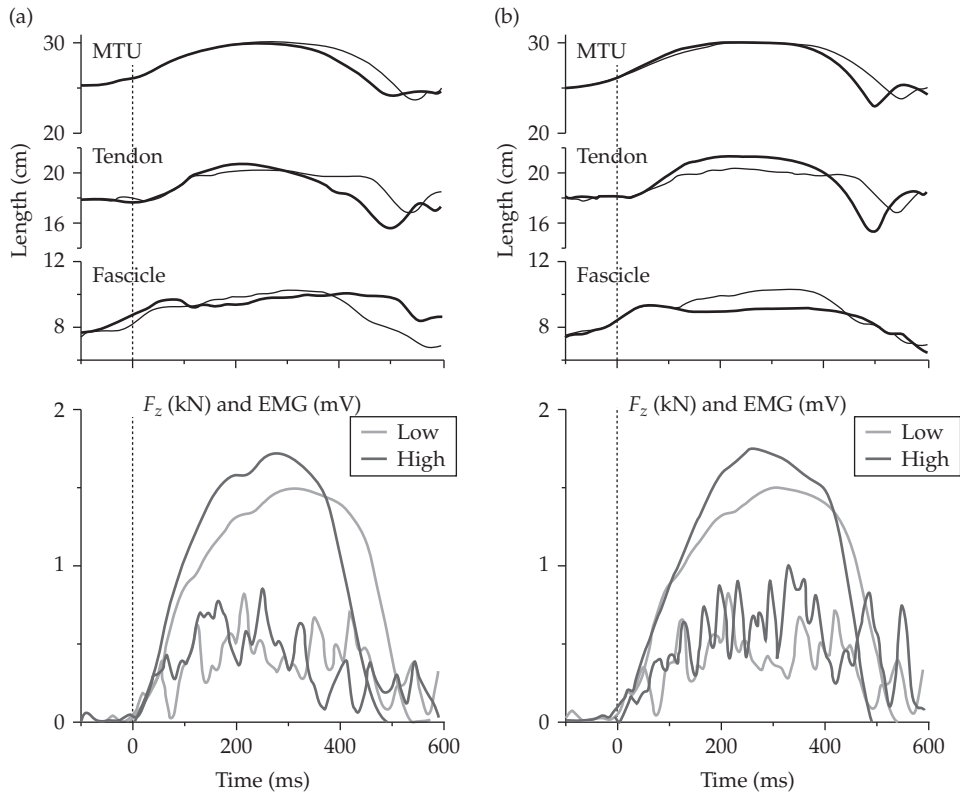


Figure 10.10 Drop and rebound intensity-specific muscle behavior during the contact phase of DJs. The curves represent the length (muscle–tendon unit [MTU], tendon and fascicle length of vastus lateralis muscle [VL]), electromyogram (EMG), and ground reaction force (F_z) data during Djs. The comparison (a) is that the rebound height was kept constant but the drop height was varied (low vs. high). The comparison (b) is that the drop height was kept constant but the rebound height was varied (low vs. high). The dotted vertical lines denote the contact on the force plate. (From Ishikawa & Komi, 2008. Copyright 2008 American College of Sports Medicine. Reprinted with permission.)

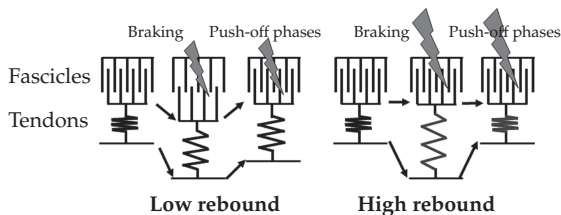


Figure 10.11 Schematic diagrams illustrating the effects of efforts of the push-off intensity on the muscle fascicle–tendon interaction during stretch-shortening cycle exercises. When the rebound efforts are increased, the fascicles are less stretched together with higher EMG activities during braking phase for utilizing the tendon stretch and recoil efficiently.

was performed with an extremely high drop height condition and maximal rebound effort, the SOL fascicle behavior was similar to that exhibited in the lower drop height conditions. They were thus still able to function “normally” without any additional rapid fascicle yielding. Interestingly, with increasing drop height, the peak tendon strain estimated during the contact phase remained the same in MG but increased in SOL. The observed differences in fascicle and tendon behavior between muscles (Ishikawa et al., 2005a,b) may simply be related to differences in functional requirements between mono- and biarticular muscles (Elftman, 1939;

van Ingen Schenau et al., 1987). The monoarticular SOL muscle may act mainly as a force generator or load bearer during SSC exercises, whereas the biarticular GA muscle is likely to function as a fine tendon strain regulator. These different functions may not be surprising as the synergistic MG and SOL muscle activities respond selectively to a SSC task such as hopping (Moritani et al., 1991).

Can High-Speed US Reveal Stretch Reflex Activity During Normal Dynamic Actions?

Again we refer to Chapter 2, where the role of stretch reflexes during SSC exercise was discussed. As the true existence and/or contribution of this simple reflex loop had been questioned and challenged (van Ingen Shenau et al., 1997), we decided to undertake a closer examination of this problem. We were especially wondering why the use of US has given the impression that the MG fascicles are not rapidly stretched during the contact phase of SSC exercises (Fukunaga et al., 2001; Ishikawa et al., 2003; Ishikawa et al., 2005b; Kawakami et al., 2002), implying that the stretch load and preactivation are

not high enough to induce such a stretch. However, when we started to work with higher frequencies of ultrasound scanning, a clear short-lived stretch of the MG fascicles occurred during the very early stance phase of running (Figure 10.12a) (Ishikawa et al., 2007; Ishikawa & Komi, 2007), resulting in the occurrence of short-latency stretch reflexes (SLR) (Figure 10.12b). We believe that this was simply due to the fact that the new scanning frequency was 96–200 Hz, which is much higher than what we and many others had routinely used before.

In the synergist SOL muscle, the muscle fascicles were stretched continuously during the braking (MTU stretching) phase, and the timing of the resulting stretch reflex was the same in different conditions. However, the timing of the short-lasting MG fascicle stretch differed between different conditions (Ishikawa & Komi, 2007). The stiffness regulation of the MG fascicles due to the preactivation may affect this timing. For example, the MG fascicles shortened more clearly at a faster running speed (6.5 m/s) compared to slower speed running (5.0 m/s). While a sudden fascicle stretch was evident in both conditions, its timing was slightly delayed at the faster running speed, where the

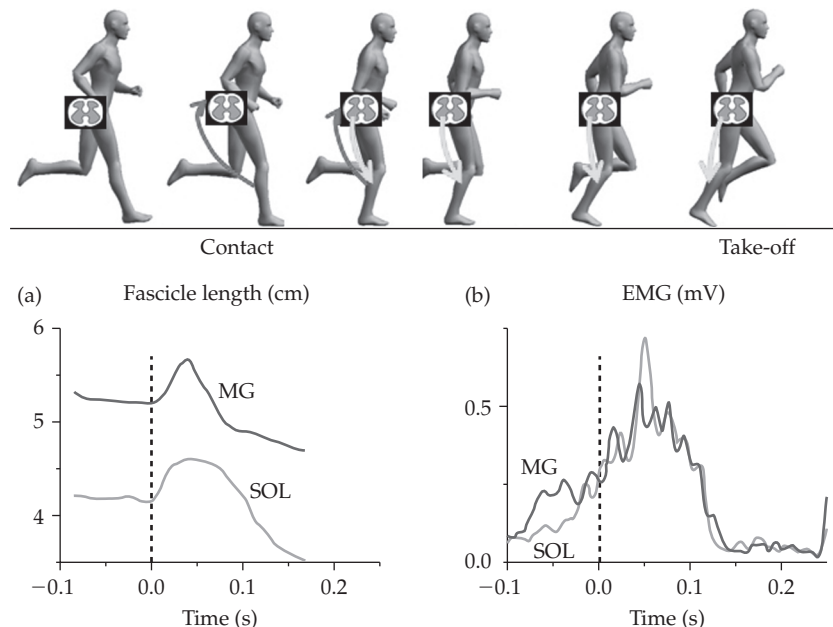


Figure 10.12 Time course data of (a) fascicle length and (b) electromyogram (EMG) activity of the medial gastrocnemius (MG) and soleus (SOL) muscles during human running (5.0 m/s). The first dotted vertical line represents the moment of ground contact. Please note that the both muscle fascicles are clearly stretched after ground contact of running and the corresponding stretch reflex responses are occurred during the contact phase of running.

MG fascicle stretch occurred approximately 26 ms after ground contact (18 ms in the 5.0 m/s condition), and the corresponding peak SLR responses occurred approximately 69 ms (56 ms in the 5.0 m/s condition) after ground contact. The end of the braking phase was approximately 68 ms and 87 ms after ground contact in the 6.5 and 5.0 m/s conditions, respectively (Figure 10.12). When we consider the electromechanical delay (10–15 ms) between the onset of SLR responses and the mechanical response (Nicol & Komi, 1998), it is clear that SLR activities can still contribute to force enhancement during the push-off phase in the 6.5 m/s condition. Consequently, the results imply that the MG SLR during the stance phase of running either influences the fascicle stiffness in the braking phase of slower speed running (5.0 m/s) or the stretch-induced force potentiation during the push-off phase of faster running (6.5 m/s) and that the contribution of the stretch reflex can be specific depending on running speed. The occurrence of a sudden MG fascicle stretch during the braking phase of running is a unique but expected finding, and is in accordance with the logical nature of the stretch reflex contribution.

Adaptation of Fascicle–Tendon Interaction with Aging and Training

Earlier observations and notions then lead us to the question, how the process of aging and training may modify the fascicle–tendon interaction

for specific movements and muscles. It is beyond the purpose of this chapter to discuss in detail the problems of aging and training at large. These questions will be dealt more thoroughly in chapters “Training Adaptation of the Neuromuscular System” and “Control and Training of Postural Balance.” However, certain aspects related to aging and training possibilities among elderly can be shortly mentioned. With aging humans, the fascicle length and pennation angle reportedly decrease (Kubo et al., 2003a; Narici et al., 2003), and the tendon compliance increases (Karamanidis & Arampatzis, 2005; Kubo et al., 2003b; Morse et al., 2005). These changes may influence the force and power production not only in static but also in dynamic movements.

Although it is not known in detail whether the muscle activation pattern in agonist muscles shows age-specific modifications, the measurements of antagonist coactivation have shown increased level of coactivation in elderly individuals (Burnett et al., 2000; Häkkinen et al., 1998; Seidler et al., 2002). This increased coactivation may influence the joint stiffness, for example, in downward-stepping condition (Hortobagyi & DeVita, 2000). On the other hand, high joint and/or leg stiffness in the braking phase have been suggested to be prerequisite for efficient SSC performance (Asmussen & Bonde-Petersen, 1974; Cavagna, 1977; Gollhofer et al., 1992). Thus, there is a considerable need to explore whether there is age specificity in regulation of the fascicle–tendon interaction during SSC exercises.

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Chapter 11

Neuromechanical Loading of Biological Tissues

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Introduction

This chapter is designed to summarize the recent knowledge on methods quantifying neuromechanical loading of biological tissues and on results describing and quantifying the tissue loading in normal locomotion and exercise. The importance and relevance of the material presented and discussed is twofold: (1) Neuromechanical loading of biological tissue in the context of the neuromuscular aspects of sport performance, is strongly related to force transmission to the skeleton and/or to the connective tissue in general and therefore directly linked to motor performance and performance enhancement. (2) Neuromechanical loading of biological tissues while normal locomotion and sport performance may overload the biological structures and lead to injury and partial tissue damage or complete tissue breakdown. Partial or entire tissue damage may interrupt training in sports and exercise which therefore hinders and counteracts performance enhancement. Both aspects will be covered in the following chapters. Additionally, a third aspect of tissue mechanical loading will be discussed. The mechanical load to biological structures and thus biological tissue is a prerequisite for its formation, maintenance, and remodeling and consequently its neuromechanical potential and performance capacity. From this perspective

of knowledge on neuromuscular loading, it is predominant or of major importance to control and steer physical training in the elite as well as in recreational sports, in sport of adolescents, of adults as well as of the elderly. The chapter contributes to establish the structural foundation necessary to appreciate both the normal function of the human musculoskeletal system and how overuse or injury may affect this function. In addition, some details of particular connective tissue (tendon, ligament) and muscle tissue will be highlighted because these tissues are most often involved in overload and in injuries of the musculoskeletal system.

The chapter begins with the presentation of variables which are used and are significantly critical to study neuromechanical tissue loading. These variables, such as force, stress and strain, stiffness and elasticity, are examined in the context of connective tissue damage, maintenance, or adaptation. A brief overview on methods and measuring techniques applied to tissue load quantification in vivo, in vitro, in human, and animal models will be presented in a separate section.

The focus of the chapter is on connective tissue and will cover neuromechanical loading of ligaments, tendons, and muscles. These tissues play the major roles in force generation and load transmission through the joints to the bones and to the physical abutment. For that reason, they are of extreme importance to sport performance and performance enhancement. These tissues are prone to injury and damage and are in some aspects the limiting tissues to training and performance enhancement in sport.

Variables and Methods

Variables of Neuromechanical Loading

Loading of biological structures is predominantly related to the force applied to the tissue and the resultant strain within the tissue. When the force applied to a tissue is related to the load-bearing tissue area, mechanical stress as the force per tissue area describes the tissue structural loading. Stress and force can appear as tensile or shear stress and force. Absolute forces are given in Newton (N) and normalized forces are in relation to a subject's body mass (N/kg) or in times of bodyweight ($\times BW$). Mechanical stress within a tissue is given in Pascal (Pa), kilopascal (kPa), megapascal (MPa), or even gigapascal (GPa). The force applied to connective or muscle tissues leads to tissue deformation as elongation. Elongation of a biological structure is indicated by the change in length of the structure in millimeters (mm) or micrometers (μm). Mechanical stress applied to a biological structure results in a relative change in tissue length. This strain (relative length change due to mechanical stress) is given in percent of the tissue resting length or in microstrain ($\mu\epsilon$).

Direct variables describing the neuromechanical loading are (1) the forces applied to the biological structure, (2) the mechanical stress within the tissue under study, (3) the tissue's deformation or elongation, and (4) the tissue strain and the strain distribution within the tissue. For indirect variables estimating the mechanical loading of the muscular-skeletal system, the resultant joint moments and bone-to-bone forces at a given joint are discussed. Even the GRFs and the pressure distribution at the contact area of the human body to the environment are sometimes used as indicators of mechanical loading of the biological structures. These variables dramatically mislead the approach to describe tissue loading due to the fact that, for example, the GRFs represent the acceleration of the CoM of the body and the forces acting at the distal end of a kinematic chain. They are not directly related to joint and especially tissue loading. The frequently reported resultant joint moments and resultant joint force, often calculated through inverse dynamic approaches, represent the net sum of all moments

and forces applied to a joint. In some model approaches, they are distributed to the load-bearing structures of the joint and thus to ligaments, MTUs, and bones. But this distribution is strongly related to the model used and especially the optimization method and the cost function used. Resultant joint moments and joint forces are related to the mechanical loading of the ligaments and the MTUs but they normally underestimate the real tissue loading. They are indicators of tissue loading but cannot be used as absolute quantifiers of neuromechanical loading of biological structures.

Forces acting on or within a biological structure, stress and stress distribution within the tissue, structural elongation of a biological structure, strain and strain distribution within the biological structure are variables quantifying neuromechanical loading of biological tissues. GRFs, plantar pressure distribution, net joint moments, and net joint force can be used as indicators of tissue loading but they do not have the potential to deliver quantitative data of the mechanical loading.

The mechanical properties of the biological tissues are extremely diverse and different tissues show a large range in mechanical stiffness, ultimate tissue stress, and tensile strain. Therefore, the mechanical properties of the tissues are summarized before the chapter will focus on neuromuscular loading in locomotion and exercise.

Methods Estimating Neuromechanical Tissue Loading

Two principal approaches are applied to study mechanical tissue load in vivo or in vitro: (1) direct measurements and (2) indirect methods. While the direct techniques use sensors and transducers based on different technical principles and provide data describing variables connected to neuromechanical loading, the so-called "indirect methods" compromise mathematical models allowing calculation or estimation of loading-related variable on or within a biological tissue. The latter can be differentiated in multibody models and finite element models, or combinations of both. EMG-driven models have also been used to quantify mechanical loading. Ultimately, then due to the fact that the

majority of mathematical models used for loading calculations have more unknowns than equations, these approaches are underdetermined. Additional assumptions (e.g., tissue material properties, joint surface areas) must be taken into account and static optimization often has been used to minimize the solution space. It is obvious that the numerical solution is strongly related to the cost function used to solve the problem. Therefore, model outcomes have to be interpreted carefully and results of different models cannot be (quantitatively) directly compared. A good example of an application of a mathematical multibody dynamics model applied to tissue load quantification in sports is given by Morlock (1990), who calculated the elongation of the medial and lateral ligaments of the ankle joint during lateral cutting maneuvers and estimated the tensile strain to these ligaments. The model compared two experimental footwear conditions and presented data on differences in ligament loading due to different court shoes. The maximum elongation of more than 6 mm is calculated for the lateral ligaments in the cutting maneuvers which is related to a ligament strain of approximately 10%. The results indicate higher elongation values of the calcaneofibular ligament and lower value of the ATFL for the stiff shoe condition and provide information on the footwear-related ligament mechanical loading.

The direct methods measure the elongation and the strain of a biological material or the force applied to a structure using different transducer technologies. From the strain and the strain distribution, the mechanical stress and the stress distribution within the biological structure or the tissue can be derived. In addition, measurements of bone position relative to each other using optical methods and bone pin techniques have been applied to quantify joint translation and to calculate ligament elongation.

Several sensors have been developed to quantify forces in tendons and ligaments *in vivo* and *in vitro*: foil strain gauge transducer, liquid metal strain gauge transducer, buckle transducer, implantable force transducer, and OF transducer. To measure tissue strain, Hall effect strain transducers (HESTs) and differential variable reluctance transducers

(DVRTs) have been applied. Strain distribution has been estimated through optic methods and in limited applications ultrasound technology has also been used.

Force measuring transducers are commonly classified as either direct meaning that force is measured directly, or indirect meaning that force is measured indirectly, for example, via deformation or strain. Buckle transducers and implantable force transducers are generally considered direct force transducers. Strain gauge transducers, optical dimension analysis, and Hall sensors are generally considered indirect force transducers. This categorization is somewhat artificial since every force measuring device must quantify force indirectly. A buckle transducer uses deformation and thus strain to determine force.

A foil strain gauge transducer consists of a foil gauge and a stainless steel shim that is sutured to the ligament or the tendon (White & Raphael, 1972). Theoretically, these transducers are highly accurate but sometimes problems occur, because the sutures are unable to act as rigid displacement attachments between the tissue and the shim (Shive et al., 1992).

Liquid metal gauge transducers consist of a gauge body of silastic or silicone tubing, a liquid metal (mercury), two isolated wires plugging the ends of the gauge tubing, and two end seals of silicone heat shrink tubing attaching and sealing the gauge tube to the wires (Brown et al., 1986). Axial strain in a tendon or ligament causes the gauge to deform by increasing the column length of the mercury and decreasing the CSA which effects a net increase in electrical resistance. The liquid metal strain transducer is small enough to fit into individual fiber bundles. The liquid metal strain transducer interferes minimally with the mechanics of the tissue, because mechanically it is not very stiff.

Buckle transducers consist of a rectangular, stainless steel frame element for supporting a portion of the tendon or ligament, a stainless steel beam mounted transversely between the tendon and the rectangular frame element, and two foil strain gauges mounted on the buckle for measuring deformation (Salmons, 1969). Under axial loading, the tendon or ligament lengthened, producing

a transverse force and a resulting deformation to the beam element, can be quantified. The buckle transducer is a relatively stable measuring device for chronic *in vivo* experiments (Komi et al., 1987; Komi, 1990; Herzog et al., 1993). Buckle transducers require a finite tendon or ligament length and distance between bony structure and the sensor. For other details, see Komi, Chapter 9.

Implantable force transducers are either based on the deflection of a metallic element (Xu et al., 1992) or on a modification of a miniature, circular pressure transducer (Holden et al., 1991). The former consists of a curved steel spring with foil strain gauges mounted on the upper and lower side. This transducer is directly inserted into a longitudinal slit in the tendon or ligament mid-substance and is normally held in place with sutures through the adjacent tissue fibers and at the slit edges. Implantable transducers allow an absolute force and an absolute zero line to be determined. Another application of implantable transducers has been reported by Dürselen et al. (1995). The authors attached in an *in vitro* setting a small Ω -shaped copper sheet equipped with strain gauges by sutures to the antero-medial bundle of the anterior cruciate ligament (ACL) and to the postero-medial bundle of the posterior cruciate ligament (PCL) and measured the ligament strain under different muscle forces applied to the knee joint.

The measurement of the OF transducer is based on modulation of light intensity by mechanical modification of the geometric properties of the plastic fiber which is inserted into the tendon or ligament. The structures of OFs used in animal and human experiments (Komi et al., 1996; Arndt et al., 1998) consist of two-layered cylinders of polymers with small diameters. When the fiber is bent or compressed, the light can be reduced linearly with pressure. The fiber can be inserted into the tendon or ligament with help of a hollow needle which is first passed through the tendon or ligament. The sterile OF is then passed through the needle. The needle is removed and the fiber remains *in situ*. Both ends of the fiber are then attached to the transmitter–receiver unit. The calibration usually gives a good linear relationship between external force and OF signal. See also Komi, Chapter 9.

The OF can be applied also for measurements of loading of various ligaments. An experienced surgeon can even insert the fiber through deeper ligaments such as the ATFL (Komi, 2003). However, special care must be taken to ensure that the OF is in contact with the ligament only and that it is preserved from interaction with other soft tissue structures by catheters.

Hall effect strain transducers consist of a magnetized cylindrical rod and a tube mounted with a Hall generator (Arms et al., 1983). The device is fixed to the tendon or ligament by barbs attached to the magnet and the tube. As the tendon or ligament is strained, the rod displaces with respect to the tube, and the Hall generator produces a proportional voltage. HESTs may be firmly anchored to the tissue and does not load the tissue during strain monitoring. The sensor is small and minimal surgical intervention is required to affix it to the tissue. HESTs measure local strain and therefore local stress and forces. Beynnon and Fleming (1998) used in *in vivo* studies of the ACL the HEST (MicroStrain Burlington, VT) to measure the displacement behavior of the ACL to calculate its strain response. In more recent studies, this group has used the DVRT (MicroStrain Burlington, VT). Both devices are similar in many respects: they are small (typically 4–5 mm in length for application to the human ACL), highly compliant, can be sterilized, designed for implantation *in vivo* using an arthroscopic approach, use a similar barbed attachment technique enabling characterization of tissue structure strains, and they measure specific ligament regions enabling characterization of the strain distribution of the ACL. This attribute is important because ligaments such as the anterior and posterior cruciate have complex morphometry with collagen bundles spiraling about their length and fanning out at the insertions (Figure 11.1).

Butler et al. (1990) and Woo et al. (1983) have developed optical techniques for the measurement of surface strains in soft tissues. These techniques are ideal for monitoring surface strains and strain distribution, particularly during high rate tests. They are not useful for out-of-plane movements, or for ligaments such as the cruciate ligaments that cannot be directly viewed. The major advantage of

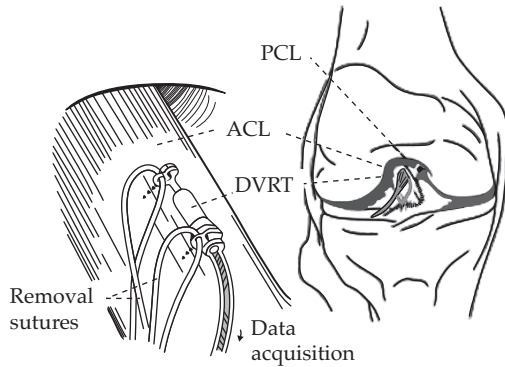


Figure 11.1 DVRT attached to the antero-medial aspect of the ACL. PCL, posterior cruciate ligament; ACL, anterior cruciate ligament; DVRT, differential variable reluctance transducer. (Taken from Fleming et al., 1998.)

these optical methods is that they allow to determine the distribution of tissue strain.

Currently, the most widely used technique to investigate human tendon displacement, *in vivo*, during muscle contractions is B-mode US which was developed in the mid-1990s for measurements in the whole tendon–aponeurosis complex (Fukashiro et al., 1995a) and in the late 1990s for measurements in isolated tendon (Maganaris & Paul, 1999) (see also Chapter 10). The method accounts for only two dimensions of structural deformation (i.e., in the sagittal plane). The technique was originally based on the displacement of intramuscular fascicular structures that can be observed on the ultrasound image and so the resulting deformation does not represent that of the free tendon, but rather the total deformation of the combined tendon and aponeurosis distal to the measurement site.

This can, in some MTCs but not all, be circumvented by identifying the very junction between the aponeurosis and free tendon or alternatively by introducing a visible landmark such as a needle. For the patellar tendon, the problem can be overcome by having two bony landmarks (tibia and patella) (Hansen et al., 2006). The technique is most commonly applied during “isometric” conditions in which small amounts of joint rotation or body movement may take place that can affect the displacement measurements. Another limitation is that

the tendon elongations obtained *in vivo* depend not only on the tensile force applied but also on the length of the in-series contractile machinery: for a given tendon length and sarcomere shortening, the greater the number of serial sarcomeres, the greater the absolute shortening in the entire muscle and the lengthening of the tendon on isometric contraction. To account for this effect and to avoid misinterpretation of results from studies across different tendons, differences in the ratio of tendon length/muscle fascicle length should be considered (Zajac, 1989; Trestik & Lieber, 1993).

Lichtwark and Wilson (2005b) combined ultrasound imaging and inverse dynamic tendon net force calculation to determine the *in vivo* AT stress and strain in one-legged hopping.

Using cine phase-contrast magnetic resonance imaging (PC-MRI), it was recently demonstrated that the AT strain during voluntary contractions (40% of MVC) was 4.7% while that of the mid-region aponeurosis was 2.2% (Finni et al., 2003). PC-MRI measurements can be made even in areas with no special anatomic features that have high visibility with ultrasound. PC-MRI has been used to study the AT and SOL muscle aponeurosis behavior during voluntary contractions in humans under *in vivo* conditions.

To determine different fascicle lengths (proximal, central, and distal) and other architectural muscle variables, piezoelectric crystals have been implanted on the MG muscle of adult cats. Measurements of the distance and distance changes between the six piezoelectric crystals were made with a sonomicrometer (TRX8, Sonometric Corporation, London, ON, Canada) using the ultrasound transmit time technique (Griffiths, 1991). This technique offers the access to deformation and strain measures within the fascicle of the skeletal muscle in an animal model.

In summary mathematical modeling and direct measuring techniques offer possibilities to estimate or even quantify tissue loading in terms of force, stress, and strain measurements. A number of techniques can be applied *in vivo* and some are restricted to *in vitro* applications. The direct methods are invasive or are often restricted to static positioning of the subject and have therefore

limited applications in dynamic movements and especially high impact physical activities in sports and exercise.

Mechanical Properties and Neuromechanical Loading

Morphology and Mechanical Properties

Muscle and Connective Tissues

Muscle tissue can be divided into three categories: skeletal, smooth, and cardiac. Muscle tissue is derived from the mesoderm and all three types of muscle cells perform the specialized task for conductivity and contractibility. At present the focus is on skeletal muscle tissue. Skeletal muscle is called striated muscle because its fibers have multinucleated striations and they are under voluntary control. They are enveloped in a sheath of connective tissue that blends with its tendon. Because of the contractibility of skeletal muscle cells, they have the prime function of generating force to maintain posture and produce body movements by the moments or torques throughout the joints.

Connective tissue is also derived from the mesoderm. It differs primarily from the other three types of tissue (epithelial, nervous, muscle) in the amount of extracellular substance. Cells are soft, easily deformable structures, and by themselves would be unable to transmit substantial loads. The extracellular matrix that holds the connective tissue together gives it form and allows the tissue to transmit load. The ratio of cells to extracellular matrix and the composition of the matrix gives the physical characteristics of the connective tissue. The composition of the matrix can range from a relatively soft, gel-like substance as in a ligament to the rigid matrix found in the bone. A primary role of the cells in the tissue is to produce and maintain the extracellular matrix. Tendons and ligaments are categorized as dense connective tissue and specialized load-bearing tissues.

In organized tissue, the collagen fibers run in parallel bundles. These regularly arranged tissue types include tendons, ligaments, and aponeuroses. In all these, the tissue is primarily composed

of fibers and extracellular matrix components. Fibroblasts are the principle cells in these tissues. These tissues have great tensile strength but are able to resist stretching primarily in one direction, that is, along a tensile force generated parallel to the fiber line.

Ligaments

Morphology and Function

Ligaments are dense, regular connective tissue structures that join bone to bone. The major functions of ligaments are (a) to attach articulating bones to one another across a joint, (b) to guide the joint motion, (c) to maintain the joint congruence, and (d) to possibly act as a positional bend or strain sensor. In terms of pure mechanics, a ligament has to resist tensile forces along the line of the collagen fibers. In general, joint ligaments have a structure that is similar to that of a tendon, but while collagen fibril bundles in tendons are typically aligned parallel to each other (in line with the pull of the muscle). The collagen fibril bundles in ligaments may be orientated in parallel, obliquely, or even in a spiral arrangement. The geometry of the collagen fibril bundles in ligaments is specific to a ligament's function. The color of collagenous ligaments is a duller white than tendon because of the slightly greater percentage of elastic and reticular fibers found between the collagen fibers bundles.

The ligament insertion to bone is either direct or indirect. Insertions anchor the ligament into the rigid, no-compliant bone. The direct attachment or insertion occurs where the ligament inserts directly to the bone. Direct insertions contain four different cellular zones, all of which occur approximately 1 mm of each other. The first zone is a normal ligament mid-substance with organized parallel collagen bundles, some elastin, and elongated fibroblasts. The second zone consists of nonmineralized fibrocartilage. The collagen fibrils continue to extend into this region. The third zone is characterized through mineralized cartilage and is distinguishable from the previous zone by the "tidemark" (dark line). The fourth zone is where ligament collagen blends directly with bone collagen. Indirect insertions occur where ligament, temporarily during growth

and development, inserts into the periosteum which is connected to the bone. Insertions are mechanically stiffer near the bone than near the ligament mid-substance. Such stiffening could lessen stress concentration and reduce the risk of tearing due to shear forces within the tissue at the interface.

Fibroblasts are the principle cells in the ligaments, while the main fibrous component of the extracellular matrix is type I collagen (36%) (Miller & Gay, 1992). Several other proteoglycans are also present although fewer than in articular cartilage. Because almost two-thirds of a ligament is composed of water, the hydrophilic proteoglycans may play a role in the mechanical behavior of a ligament.

Anatomically, joint ligaments such as those in the knee joint contain several types of sensory receptors that are capable of providing the nervous system with information about movement, position, strain, and pain. Nevertheless, the exact neurosensory role of ligaments and mechanoreceptors in joint proprioception is still controversial.

Mechanical Properties

The mechanical properties of soft connective tissue can be classified into structural and material properties. The structural properties of a ligament are derived from the behavior of a bone–ligament–bone complex and hence involve the mid-substance of the ligament, the insertions, and the local bone to the insertions. The material properties describe the material irrespective from geometry. Material properties in this perspective are usually measured in the mid-substance of a ligament. The structural (mechanical) properties of ligaments are somewhat similar to those of tendons. A typical force deformation curve describing the nonlinear structural properties of ligaments is shown in Figure 11.2.

The stiffness of ligaments varies nonlinearly with force applied. It permits initial joint deformation with minimal resistance. At higher forces, the ligament becomes stiffer, providing more resistance to increasing deformation.

Collagen, the main tensile-resisting substance in the ligament, is crimped in an unloaded situation. This crimp is thought to allow some extensibility of the ligament along its length under low forces.

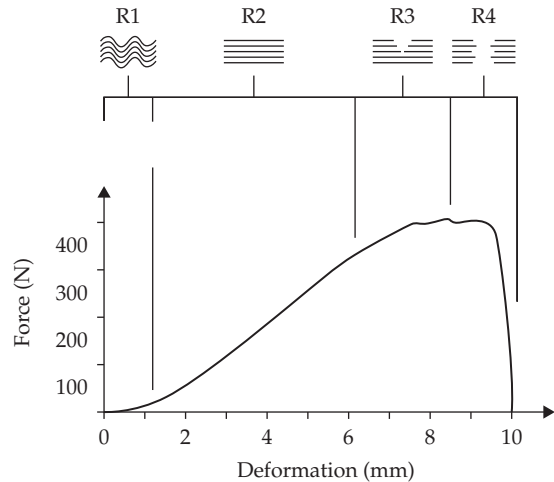


Figure 11.2 Force deformation curve of a ligament.

As tension is added the crimp flattens out. Once no more crimp can be removed, stiffness increases and an increasing force is required for further displacement. The toe region of the force deformation curve corresponds to the stretching out of the crimp. With the crimp gone and the whole matrix under tension, a region with more constant linear stiffness begins.

Neither fibers nor crimp are homogeneously distributed along the length of ligaments. When ligaments are distracted, different fibers are recruited into force bearing at different displacements. As soon as all the fibers have been recruited, the stiffness behavior becomes more linear until some of the fibers fail. At this point, the net stiffness of the structure begins to drop (region R3 in Figure 11.2). Since some fibers fail, the force is redistributed onto the remaining fibers increasing the force on them and the likelihood of their failure. Small additional deformation produces large structural failure of the ligament through all remaining fibers (R4 in Figure 11.2).

Microscopically, some fibers cross between parallel fibers in the ligament substance, some run perpendicular to the long axis, and some at every angle in between. While there are not many non-axial fibers (Liu et al., 1991) and they tend to be smaller than their longitudinal oriented partners,

they should nonetheless contribute to nonlinear stiffness behavior as the ligament is forcefully stretched. Depending on how and where the non-axial fibers are connected, they could serve as tethers (stabilizers) for longitudinal fibers. The crossing fibers could alternatively be connected in a separate network from end-to-end in some oblique fashion. The microarchitecture of even the relatively simple ligaments is not known completely. However, it is known that gross mid-substance strains from around 8% are sufficient to cause failure of that area, e.g., of the medial collateral ligament (MCL) of the knee (of a rabbit) (Lam, 1988).

Like other connective tissue, ligaments exhibit force relaxation and creep as a result of the viscous component of the ligament's response to force.

The material behavior of ligaments, e.g., stress-strain behavior, is also nonlinear (Woo et al., 1983). With increasing strain, the stress in the ligament increases as well. In the toe region, there is little stress relative to the strain applied. This area corresponds to the straightening out of the collagen fibers. This is followed by a linear region, presumably as the collagen fibers take up force. Finally, the curve flattens out eventually dropping dramatically toward the strain axis. The flattening is presumably related to increasingly rapid microfailure followed by catastrophic failure. The reasons for the nonlinear behavior at the stress-strain level are not as easy to explain as that of the structural behavior. Less is known about the actual molecular nature of the ligament components or their interactions. Of course there should be some overlap with structural explanation. Collagen fibers may display nonlinear characteristics as they are in tension due to a molecular rearrangement, or some internal reordering of their relation with elastin or fibronectin.

The definition of ultimate tensile strain or even stress of a ligament is not easy because in normal joint motion together with other ligaments and MTUs, the ligament under study cope with varied angles of stress. The ACLs, for example, are stronger in direction similar to their predominant fiber orientation (ultimate force applied to failure: around 2600N (Woo & Adams, 1990) than in the direction aligned with the tibial axis (around 1900N).

Some of the most important factors that affect the normal integrity of ligaments include exercise, immobilization, aging and maturing, and previous injuries. Therefore, the identification of a numerical value of ultimate ligament stress and ultimate ligament strength, or strain, is vague and is not subject specific and uncertain.

Tendons

Morphology and Function

Tendons are pearly white, collagenous flexible bands that connect muscle to bone. Tendons vary considerably in size and shape as well as depending on the morphological, physiological, and mechanical characteristics of both the muscle and bone to which it is attached. The tendon consists of an external tendon which is typically referred to as tendon, and an internal tendon which is typically referred to as aponeurosis. The external tendon connects the muscle proper to the bone; the aponeurosis provides the attachment area for the muscle fibers. Despite the variable nature of tendon size and shape, every tendon has three distinct regions of organization: the muscle-tendon junction (myotendinous junction), the tendon proper (often referred to as tendon), and the bone-tendon junction (osteotendinous junction).

The basic building blocks are the tropocollagen molecules. Tropocollagen molecules generally are aligned in parallel rows to form a microfibril. Subsequently, the microfibrils aggregate into parallel bundles to form subfibrils and then fibrils. Fibrils are gathered into fascicles bound together by a loose connective tissue, which permits a relative motion of the collagen fascicles and supports blood vessels, nerves, and lymphatics. Tendon fascicles are grouped into the tendon proper. In tendons of extreme load-bearing functions like the AT, fascicles are arranged in a twisted mode in order to increase the ultimate tensile stress and to increase the stiffness.

The major component of tendon is type I collagen, which accounts for about 86% of the dry weight of a tendon (Woo et al., 1994). The insertion of tendon into the bone involves a gradual transition from tendon to fibrocartilage, to mineralized

fibrocartilage, and finally to bone. Some of the collagen fibers of the tendon pass through the mineralized fibrocartilage and into the subchondral bone. These penetrating fibers are sometimes called "Sharpey's fibers." An additional anchor is provided by fibers from the tendon, which blend with the bone's periosteum.

At the opposite end of the tendon, the myotendinous junction is a specialized region of longitudinal membranous infolding that increases the surface area and reduces the stress on the junction during the contractile force transmission. The strength of an adhesive junction such as the myotendinous junction depends both on the properties of the adjoining structures and on the orientation (direction) of forces across the junction. Junctions that are loaded in shear, with the force being parallel to the membrane surface, are stronger than junctions with a large tensile component perpendicular to the membrane (Tidball, 1983). Aponeuroses are fibrous, ribbon-like membranes similar in composition to tendons. These structures are sometimes called flattened tendons or internal tendons (Zajac, 1989). Aponeuroses are whitish in appearance due to the presence of collagen. The fibers of aponeuroses run in a single direction and thus differ from unorganized connective tissue (fascia).

Mechanical Properties

The primary role of tendon is to transmit the force of its associated muscle to the bone. As such, tendon needs to be relatively stiff and strong. The material properties of tendons, describing the material irrespective from geometry, are more or less similar to that of ligaments. The structural properties of a tendon are derived from the behavior of a muscle-tendon-bone complex and thus involve the mid-substance of the tendon, the insertions, and the aponeurosis. When a tendon is relaxed (no tensile force), it takes a crimped or wavy appearance. As a load is applied, the wavy pattern is straightened. In the stress-strain-function, the crimped pattern is indicated by the so-called "foot region" of the curve. The toe region typically lies below 3% strain, a region in which specimen elongation is accompanied by very low stress. In the linear region of the stress-strain

curve, where the tendon is stretched less than 4%, collagen fibers lose their crimp pattern. The slope of this linear portion of the curve has been used to define Young's modulus of the tendon. If the tendon is stretched over 4%, microscopic tearing of tendon fibers occurs. Beyond 8–10% strain, macroscopic failure occurs. Further stretching causes tendon rupture. It should be noted that these values of tendon strain maybe underestimated. Using modern testing devices, a recent study has shown that avian flexor tendons can be elastically stretched up to 14% (Devkota & Weinhold, 2003). Lichtwark and Wilson (2005) reported peak AT strain with a range of 6.2–10.3% during one-legged hopping. The Young's modulus in tendons of different mammalian species ranges from 1.25 to 1.65 GPa with the mean across all species of 1.5 GPa (Bennett et al., 1986). Beyond the region of linear or reversible strain, permanent deformation occurs. Traditional numbers of the ultimate or failure strain of tendon are about 8–10% (Rigby et al., 1959); more recent data reported a strain at failure of the human patella tendon of up to $14 \pm 6\%$ in young donators (Johnson et al., 1994). There is a considerable yield region in which tendon deformation is accompanied by very little increase in stress. Tendon failure should result from the pulling apart the collagen fibers. The ultimate or failure stress is often defined as tendon strength. The strength of the tendon material, like any solid material, depends on the presence of flaws. Since the number and the severity of flaws likely vary between specimens, strength measurements for tendons are expected to be variable as well. Elliott (1965) reported tendon strengths (ultimate tensile stress) in the range of 20–140 MPa. More recently Bennet et al. (1986) measured ultimate strengths of various mammals and reported values ranged from 90 to 107 MPa, with a global mean across all species of about 100 MPa. Johnson's data (Johnson et al., 1994) of the human patella give an ultimate tensile strength for young donators of 64.7 ± 15 MPa (53.6 ± 10 MPa for older donators) and Young's modulus were found to be 66.0 ± 26.6 MPa (50.4 ± 22.2 MPa for the older donators). A study by Maganaris and Paul (1999) estimated the in vivo mechanical properties of the human TA tendon. Tendon stiffness and Young's

modulus were determined at 161 N/mm and 1200 MPa.

Besides being relatively stiff and strong in tension, tendon is highly resilient. Ker (1981) reported that the hysteresis area and thus the energy dissipation of sheep plantaris tendon was only 6% when loading and unloading the tissue. Bennett et al. (1986) showed that mean values of hysteresis of several vertebrate tendons range from 6% to 11%, and were independent of loading frequency. Thus 89–94% of energy associated with longitudinal deformation or stretch is recovered when the load on the tendon is removed. For a biological material, tendon shows marked elastic behavior within a range of relevant frequency of deformation.

Because tendons are highly resilient structures, they are capable of storing and releasing significant amounts of elastic energy. This property of tendon is thought to be of considerable importance for the functionality of the locomotor system and its evolution.

Muscles

Morphology and Function

Skeletal muscles are the prime executors of the nervous system. The most basic property of muscles is its ability to produce force. Contractile proteins and a network of connective tissue are the two basic elements of muscles. Fibrous connective tissues within the muscle belly that blend with the tendon provide important functional stiffness, which enhances the transmission of tension. There are significant cellular interactions that direct a muscle's physiological response, but muscle adaptation and injury are best described by considering the mechanics of a muscle's functional units. From the whole-muscle level, individual muscle fibers (muscle cells) are subdivided into myofibrils, sarcomeres, and finally to actin and myosin. The connective tissue surrounding the entire muscle is called the epimysium, and the bundles of muscle fibers (fascicles) are surrounded by the perimysium. Each individual muscle fiber is surrounded by endomysium.

Muscle fibers vary in length. They can shorten to approximately half of their resting length. Human

skeletal muscle comprises several different fiber types that have different functional characteristics. Most muscles in the body are a mixed variety containing a combination of muscle fiber types.

Muscle fibers with the same biochemical properties tend to have similar force-producing characteristics. A muscle fiber shortening to one half of its length will have the same force characteristics whether it is long or short because the sarcomeres are in series. Increasing the number of muscle fibers in parallel, however, increases the effective force of a muscle.

Mechanical Properties

One mechanically important aspect of muscle architecture is the angle of pennation of the muscle fibers. Longitudinal or fusiform muscles have muscle fibers lying parallel to the line of pull of the tendon. These fibers pull in a straight line, and the full magnitude of the force is directed along the tendon's line of action. The fibers of a pennate muscle (uni-, bi-, or multipennate) arise at an oblique angle to the line of pull (usually considered as a straight line along the tendon). Thus, only a portion of the force generated by the contracting fiber is transmitted along the tendon. Pennation of the fibers allows the number of fibers to increase without significantly increasing the muscle's diameter. The force-producing potential of the muscle is enhanced by the increased number of fibers lying adjacent to each other.

The muscle's force output is, in addition to the neural activation profiles, modulated by the length of the muscle when contraction is initiated and by the velocity of contraction. Force, velocity, and length are all interrelated variables that affect a muscle's mechanical response.

The maximum tension can be generated when a muscle is forcibly lengthened while it attempts to shorten (eccentric action), and the tension declines as an active muscle shortens (concentric action). Maximum strength in rapid eccentric muscle action exceeds the maximum isometric strength, and the strength is even less in concentric muscle action. (See also Chapter 1.)

Skeletal MTUs also have inherent passive properties that affect the force output. Some of the passive

structures function in series with the active muscle cells, while others function in parallel. The term series elastic component and parallel elastic component are derived from these functions. Together, the two components account for the passive tension properties of muscle.

Activated cross-bridges within the myofibrils exhibit a resistance to stretching, consequently generating an internal force that is often termed muscle stiffness. Measured as change in force per change in length, stiffness is a property of muscle believed to operate over length changes and to have functional significance during locomotion and other movements.

Since muscles are active force-producing structures, they should not have unique material or mechanical properties. If basic material properties such as force–elongation relations are measured passively, they should not be meaningful for the understanding of muscular function. Nevertheless, two properties of muscles are relevant and repeatedly used in biomechanical experiments involving muscles or the musculoskeletal system: (1) the F–L and (2) the F–V relation of muscles.

Force–length relations describe the relation between the maximal force a muscle (or fiber or sarcomere) can exert and its length. F–L relations are normally obtained under isometric conditions and for maximal activation of the muscle. For details of the relation of muscle strength and length under nonisometric condition, one can refer to Chapters 1 and 2. Under isometric condition at maximum activation, the optimal length of thin and thick myofilaments was reported between 1.1 and 1.3 μm and at 1.6 μm (Herzog et al., 1992; Walker & Schrodt, 1973).

Force–velocity relations are defined as the relation between the maximum force of a muscle (or fiber) and its instantaneous rate of change in length. F–V properties are determined for maximum activation conditions, and are typically obtained at optimal length of the sarcomeres. As described in more detail in Chapter 2, this classical F–V characteristics of human muscles and especially MTUs do not represent the so-called “instantaneous F–V curves,” that are specific to normal locomotion, such as the SSC (Komi, 2003).

Neuromechanical Tissue Loading in Normal Locomotion and Exercise

Ligaments

Only relative little information of direct measurements of ligament mechanical loading in complex joint motions, locomotion, or even sport activity is reported in the literature. One reason should be the nature of the invasive methods and the technically restricted fields of application in humans. Therefore, some results of direct measurements of in vitro studies and data gathered with indirect methods will be presented in this section. Due to records available, the focus will be on ligaments of the knee and the ankle joint.

Knee Joint

Knee ligament biomechanics have been studied with several different approaches. Due to the clinical relevance, the focus was mainly on the cruciate ligaments. Anatomical observations were reported by Girgis et al. (1975). Blankevoort and Huiskes (1996) or Komistek et al. (1998) used mathematical modeling to estimate the magnitude of knee ligaments' mechanical loading, and direct in vivo ACL force measurements have been presented by, for example, Markolf et al. (1990, 1995) and Woo et al. (1997). ACL displacement and strain measurements have been performed by Arms et al. (1984), Beynnon et al. (1992a), Beynnon et al. (1997a,b), and others. The last approach and measured ACL displacement have been used for the calculation of strain and to understand the effect of knee position and muscle activity on ligament biomechanics. The different strain measuring devices used by the different groups are similar in many respects: they are small in length for application to the human ACL, highly compliant, can be sterilized, designed for implantation in vivo using an arthroscopic approach, use a similar barbed attachment technique enabling characterization of tissue structure strains, and they measure specific ligament regions enabling characterization of the strain distribution of the ACL. This attribute is important because ligaments such as the anterior and posterior cruciate have complex morphometry with collagen bundles spiraling about their length and fanning out at the insertions (Girgis, 1975).

Consequently, the strain distribution of the ACL varies along its length and about its cross-section. Therefore, the strains in the mid-substance portion of the ligament are different in comparison to those at the insertion sites. This has been studied by Woo and colleagues *in vitro* who used a robot combined with a six degree-of-freedom force sensor to apply anterior-directed load to the tibia. They showed that the restraint forces in the antero-medial and postero-lateral portions of the ACL are different and change with respect to knee flexion angle (Woo et al., 1997).

Henning et al. (1985) measured the elongation of the antero-medial fibers of the ACL with an external tubercle gauge. To allow for a convenient reference, the authors scaled the data to a 358 N (80 pound) Lachman test which equals 100 units of elongation of the ACL antero-medial fibers. The antero-medial fiber elongation for an activity can be related to the approximate ACL load. In walking 36% of the 358 N Lachman test was measured which gives an equivalent of 129 N to the ACL. Jogging at 8 km/h produced an antero-medial fiber elongation ranging from 62% to 64% of the 358 N reference. A stationary bicycle, while riding, produced antero-medial fiber elongation only 7% as great as the 357 N reference (Lachman) test. Downhill running at 8 km/h was at 125% of the 357 N Lachman loading which gives an estimate of 446 N acting on the ACL. A one leg half squat (20°) was related to an elongation of 21% as the reference. Isometric contraction of the quadriceps muscles at 22° knee flexion was shown to load the ACL with an equivalent of 121% as great as the Lachman 357 N load. At 0° knee flexion, 107% of the reference load was measured. From the data, one can derive the first figures of ACL neuromechanical loading for isolated knee extension, normal locomotion, and recreational sport activities (Figure 11.3). Highest ACL loading was shown in downhill running and knee joint extension at approximately 20° . Cycling was shown to produce very low ACL elongation and thus ligament strain and stress.

Markolf et al. (1990) studied in an *in vitro* setting, the effect of a quadriceps pull and an external varus-valgus torque on ACL resultant force. As expected in minimum knee flexion, the quadriceps tendon pull results in 90% of the resultant ACL force. In 10° knee flexion, an application of 10 Nm

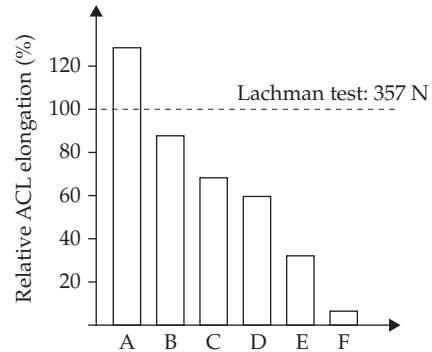


Figure 11.3 Relative ACL elongation in different activities (data of one female subject). (A) down hill jogging at 2.2 m/s, (B) jogging on floor, (C) jogging on treadmill at 2.2 m/s, (D) jogging on treadmill at 2.0 m/s, (E) walking on treadmill at 1.0 m/s, (F) stationary biking. The data are related to the elongation at a 357 N Lachman test. ACL, anterior cruciate ligament. (Data from Henning et al., 1985.)

valgus moment to the knee results in an increase of ACL force from 5 N to more than 30 N (without a quadriceps pull). A 10 Nm varus moment increases the ACL load up to >50 N. Therefore, the ACL neuromechanical loading is dominantly influenced by the quadriceps pull but also significantly related to varus or valgus moments to the knee joint.

Beynon and Fleming (1998) reported using the HEST *in vivo* the strain of the antero-medial band in active knee extension. A maximum strain value of 3% was registered at 20° knee flexion. In an isometric quadriceps contraction at a torque of 30 Nm, the ACL strain was measured at 4.4%. As a comparison, a Lachman test with 150 N shear force is related to a maximum strain of 3.7%. Squatting (20°) is related to 3.6% strain while stationary bicycling showed only 1.7% strain in the ACL (Figure 11.4).

One can conclude that peak loading of the ACL should be in an area of $15\text{--}20^\circ$ knee flexion in an active quadriceps pull. Minimum strain is related to 50° knee flexion. When an increasing anterior shear force is applied to the tibia at a knee flexion angle of 20° , the ACL strain increases from 0% at 20 N shear to >4% at 180–200 N anterior shear force. ACL strain along with ACL mechanical loading is strongly related to the shear force applied to the tibia.

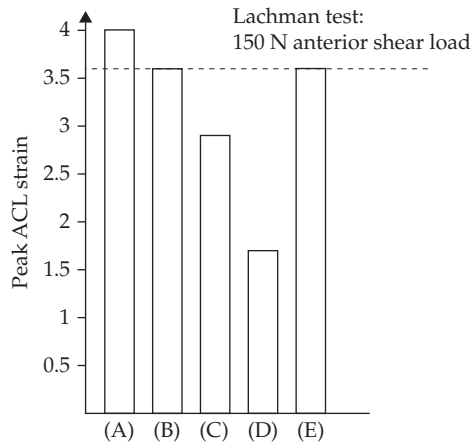


Figure 11.4 Peak ACL strain in different activities. (A) squatting with a sport cord (B) squatting (C) active flexion-extension of the knee (D) stationary bicycling (minimum resistance) (E) Lachman test (150 N anterior shear load). ACL, anterior cruciate ligament. (Data from Beynnon and Fleming, 1998).

Shear force to the tibia is a function of the quadriceps pulling and muscle force. The quadriceps muscle force can be estimated through the resultant joint moments to the knee in the sagittal plane. Therefore, ACL loading in normal locomotion, sports, or physical activity can be roughly estimated from the resultant joint moments to knee in the sagittal plane and—following the above-mentioned concept of varus and valgus moments as additional mechanical risk factor of ACL loading—in the frontal plane. An additional loading factor for the ACL is certainly the knee joint moment in the transverse plane. Quantitative data from direct ACL strain or elongation measurements in vivo in high impact and dynamic activities (e.g., landing, stopping, and jumping) are recently not available. A survey of knee joint moments in different activities is given in the section muscle–tendon neuromechanical loading. From this data, one can derive high risk activities for ACL loading due to tensile strain and stress.

Braces to the knee seem to have the capacity to apply an external posterior shear to the tibia and accordingly decrease the ACL tensile strain. Beynnon et al. (1992) reported a brace-related decrease of ACL strain of up to 30%. These results are valid for more or less static situations at a relative low level of shear forces applied to the knee.

However, Ramsey (2003) demonstrated in one-legged jumping that bracing the ACL deficient knee with noncustomized orthoses, resulted in only minor and nonsystematic changes in tibio-femoral joint motion. This study using 3D bone pin technique observed no consistent reductions in anterior tibial translation in dynamic movements as a function of the knee brace tested.

Foot and Ankle

The ankle joint ligaments' biomechanics have been studied with several different approaches. Due to the clinical relevance, the focus was mainly on the lateral ligaments. The most frequent injuries in sport, work, and leisure are ankle sprains which therefore overload the lateral ligament of the ankle joint. The cause or the mechanism of these injuries is the elongation and the strain applied to the ligament talofibulare anterius (LTFA) and posterius and the ligament calcaneofibulare. Colville et al. (1990) showed in vitro the elongation of the LTFA during ankle joint plantar flexion and Renström et al. (1988) described the impact of additional inversion to the strain of the LTFA with nearby 100%. An axial loading of the ankle joint increases the strain of the LTFA at about 2.5% (Cawley & France, 1991). The maximum strain in the LTFA were found by Nigg et al. (1990) in an in vitro study with the combination of 13° inversion and 30° plantar flexion.

Morlock (1990) calculated the elongation of the lateral ankle joint ligaments for a lateral side shuffle movement of 1.5 m length in two different footwear conditions, a stiff, and a flexible (torsional) court shoe. The maximum ligament length was reported to occur in the mid-stance when the foot is in maximum inversion and slight plantar flexion. Peak ligament elongation of the calcaneofibulare ligament showed its maximum at 4.7 cm for the stiff shoe condition and 4.5 cm for the flexible shoe. From Morlock's data, one can derive a resting length of the ligament of around 4.15 cm; therefore, the strain of the ligament can be estimated between 8.5% and 13%. When these numbers were compared with lateral ankle joint ligament loading in normal locomotion (Brüggemann et al., 2009), the strain of the lateral ligaments occur much lower in walking and running than in lateral shuffle movements. Using the shuffle lateral movement as a reference, normal

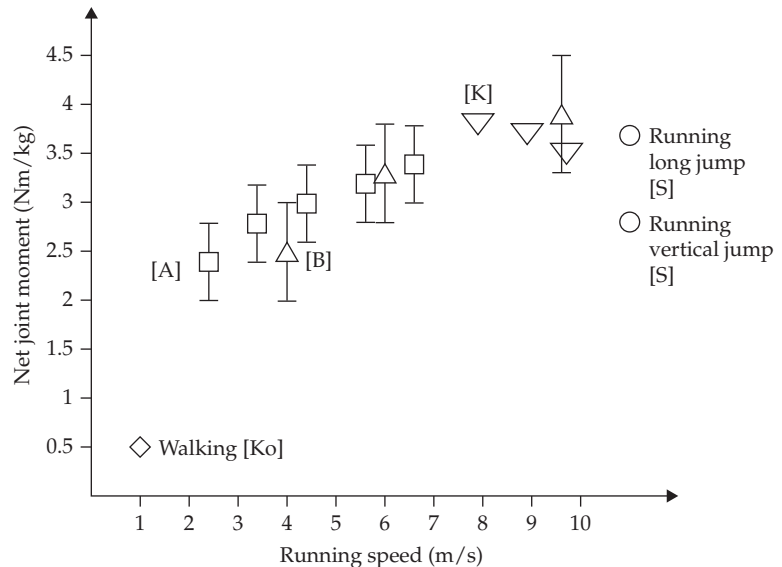


Figure 11.5 Peak net extension moment at the knee joint in walking, running at different velocities and jumping. Data from Komistek et al. (2005) [Ko], Arampatzis et al. (1999) [A], Belli et al. (2002) [B], Kuitunen et al. (2002) [K], and Stefanyshyn and Nigg (1998) [S].

walking is at 15% and running at 3.5 m/s at 30% of the reference strain.

During the stance of the lateral side shuffle, the force acting on the plantar aponeurosis reaches peak values of 2500 N (Morlock, 1990). The average force to the plantar aponeurosis during stance appears higher in the stiff shoe condition. Even if the absolute numbers derived from the model are strongly model dependent, footwear seems to have an impact on ligament loading at least in the reported lateral movements.

Recent data of lateral ankle ligaments loading in dynamic movements (Brüggemann et al., 2009) indicate that ankle bracing with dedicated orthoses can systematically decrease ligament elongation and strain in normal locomotion and in lateral movements.

Muscles and Tendons

Knee Joint

Direct measurements in the patella tendon using the OF have been performed by Komi et al. (1996), Finni et al. (1998, 2001), during walking, running, and hopping. Several authors used 2D or 3D modeling to calculate the resultant knee joint moment and to estimate the patella ligament and the quadriceps tendon force as well as the net force generated by the quadriceps muscle during locomotion (Komistek et al., 1998). In walking, the patella

ligament force as well as the quadriceps tendon is loaded at about 1.25–1.4 times body weight. During deep knee bends, the maximum force at the quadriceps tendon and in the quadriceps muscle increase up to 3.5 times body weight. In running the resultant external flexion moment to the knee is strongly dependent on running speed and can be estimated for running at 3.0 m/s at about 2.0–2.5 Nm/kg. This resultant moment is related to a net tendon and muscle force of the quadriceps at about 3100–3800 N. Whilst running, the speed increases to >5.0 m/s and the resultant external knee joint moment increases up to 3.5 Nm/kg which gives an approximate tendon and muscle force of >70 N/kg or more than 7.0 times bodyweight. Downhill running is related to higher resultant knee joint moments and therefore to higher muscle and tendon loading than level or uphill running. In maximum sprinting, the resultant joint moment is reported at a maximum of about 4 Nm/kg which gives a net force to the quadriceps tendon and muscle of 90 N/kg in the eccentric phase and 3 Nm/kg or 65 N/kg in the concentric phase of stance. In order to present an overview on knee joint tendon and muscle loading during normal locomotion and exercise, Figure 11.5 summarizes data from different sources. The figure gives the net joint moment at the knee joint with respect to the body mass. The data show a strong linear relationship of the

force to the extensor MTU and the running speed. As shown in the figure, walking is related to much lower load to the quadriceps tendon; jumping exercises load as high as fast running.

Foot and Ankle

The AT is on one hand the strongest tendon of the human body and on the other hand it is frequently affected by injuries and overuse in sport and exercise. Therefore, the majority of research of tendon and muscle around ankle and foot is focussed on AT and the triceps surae muscle. Magnusson et al. (2003) reported during near-maximum plantar flexion, the human Achilles free tendon underwent a strain of approximately 8.0% while the aponeurosis showed a strain of 1.4%. Using cine PC-MRI, it was recently demonstrated the AT strain during voluntary contraction at 40% of MVC was 4.7% while that of the mid-region of the aponeurosis was 2.2% (Finni et al., 2003). These studies suggest that the free AT is more compliant than the aponeurosis.

In one-legged hopping, Lichtwark and Wilson (2005) measured in 10 subjects an average peak strain of the AT of $8.3 \pm 2.1\%$, an elastic modulus of $0.87 \pm 0.2 \text{ GPa}$, and a linear stiffness of $18.8 \pm 43.3 \text{ N/mm}$.

The tendon and the aponeurosis of the human triceps surae are of particular interest because of the substantial loading during locomotion and exercise. Particularly in running and jumping, peak tendon tensile stress up to 11 MPa have been measured in vivo (Komi et al., 1992, 1987) using buckle transducer or OF techniques. Figure 11.6 demonstrates the ATF measured with the OF device in heel-toe running. Maximum load is in the mid-stance when the heel is raising from the ground.

Table 11.1 summarizes the most important findings of in vivo ATF measurement in humans, sport, and exercise. The data demonstrates the large range of tendon loading in the various activities and the extreme tensile stress in jumping and running.

The highest tendon loading is in fast dynamic movement, the lowest tendon force was shown in bicycling. These results are in line with the quoted results of ACL loading during locomotion, running, active knee flexion, and extension and bicycling.

It becomes evident that the rather high risk of injury to both tendon and myotendinous junction

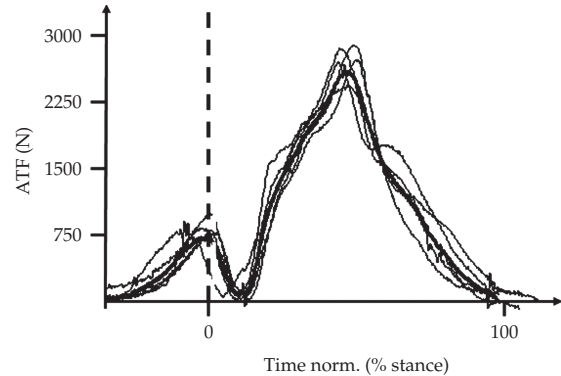


Figure 11.6 ATF in heel-toe running at 3 m/s measured with the OF technique. The graph gives the data of five trials of one subject and the average (bold). The broken line indicated the first contact with the ground.

remains a clinical challenge. The anatomical configuration of the triceps surae with three separate muscle compartments that merge via their aponeurosis into a common tendon represents a challenge in itself. The unique anatomical configuration leaves the possibility of three muscles contributing to the load occurring in the free AT. Differences in medial and lateral forces in cadaver AT has been observed when separate muscles of the triceps surae were loaded (Arndt et al., 1999). Asymmetric strain and strain distribution in the distal AT was demonstrated using an optical method when separate muscles were pulled differently (Brüggemann et al., 2007). The alignment of the calcaneus plays an important role in asymmetric AT strain. It was demonstrated in an in vitro experiment that a 7.5° calcaneal eversion leads to an increase of tensile strain at the medial column of the AT of more than 75%. A 15° eversion is related to a 100% increase of medial strain. In addition to medio-lateral axial strain differences, an intratendinous shear strain results and may cause sliding between tissue layers parallel to the acting forces. Asymmetric strain and shear between tissue layers should be discussed as a possible mechanism of AT local tissue overuse, damage, and failure to free tendon and aponeurosis.

The values of directly measured tendon forces (Table 11.1) indicate the very low tendon neuromechanical loading in bicycling, the moderate tensile force acting to the tendon during normal locomotion, and the increased forces in running, sprinting,

Table 11.1 ATFs and tensile stress in normal locomotion and exercise.

Activity	Author (year)	Achilles tendon force (N)	Tensile stress (MPa)
Walking	Finni et al. (1998)	1.300–1.500	19–22
Running	Komi (1990)	3.700–3.900	46–48
Sprint	Komi (1990)	–9.000	–110
SQ	Fukashiro et al. (1995b)	1.900–2.000	23–25
CMJ	Fukashiro et al. (1995b)	1.900–2.000	23–25
Hopping	Fukashiro et al. (1995b)	3.700–4.000	46–49
DJ	Brüggemann et al. (2000)	3.500–5.000	43–62
Bicycling	Gregor et al. (1987)	500–700	6–8

Data from direct measurements with the OF technique.

SQ, squat jump; CMJ, countermovement jump; DJ, drop jump.

and jumping. In jumping exercises, the peak tendon forces are clearly approaching the ultimate strength limits of the tissue. Due to the fact that TT has the capacity to adapt to a long-termed and well-organized training and adaptation concept in athletic training, it allows the tissue to withstand such extreme loads.

As already mentioned, the resultant joint moments can be used as indicators of neuromechanical loading. From the resultant plantar flexion moment at the ankle joint, one can derive the net tendon and muscle forces of the AT and the triceps surae muscle when the level arm of the AT is kept in mind. Some attempts even distributed the tendon force to the three muscle compartments.

Figure 11.7 summarizes data of the maximum net plantar flexion moment at the ankle joint in running at different velocities and jumping exercises. Up to a running speed of 6 m/s, the resultant joint plantar flexion moment and therefore the AT load increases linearly with an increase in running speed. At velocities higher than 6 m/s, a plateau of tendon loading can be identified. Maximum loading in jumping exercise is measured at this level of loading. Finally, Figure 11.8 gives the estimated peak forces of the AT derived from the net plantar flexion moments reported in different sources. The highest calculated force applied to the AT was reported for the takeoff of a (double) backward somersault after a handspring backward in gymnastics. The reason for the extreme forces are the high kinetic energy of the gymnast prior to takeoff, the transfer of angular momentum to linear momentum during the takeoff, and the >45° turn

round of the CoM movement during the 120 ms ground contact. An asymmetric AT tensile stress and the related tensile strain in floor exercise takeoffs have the potential to lead to an acute failure through tissue loading above the critical limits or tolerances. Such asymmetric loading actions are reported when gymnasts takeoff for a somersault with twists and when they initiate the longitudinal axis rotation during the contact to the ground.

The presented figures give an overview on peak tendon and muscle, as well as ligament loading in normal locomotion, exercise, and sports. Sport and exercise are related to high loading of the biological tissues in terms of peak tensile stress and tensile strain. Especially, activities at a high velocity and therefore at a high level of total body kinetic energy have the potential for extreme mechanical tissue loading, which can be close to the ultimate tissues' strain limits.

The data presented should contribute (1) to estimate the amount of activity-specific neuromechanical loading, (2) to avoid an individual overuse, and (3) to choose appropriate loading regimes to ensure tissue adaptation and an increase of tissue potential.

Concluding Remarks

Physical activity and exercise is related to biological tissue loading. The neuromechanical loading during locomotion and exercise is linked to force transmission to the skeleton and therefore to mechanical loading of connective tissue. The neuromechanical loading during sport and exercise has the potential to overload the biological structures and

Figure 11.7 Peak net plantar flexion moment at the ankle joint in running at different velocities and jumping. Data from Arampatzis et al. (1999) [A], Belli et al. (2002) [B], Kuitunen et al. (2002) [K], and Stefanyshyn and Nigg (1998) [S].

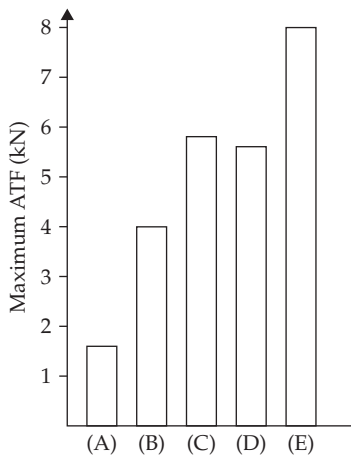
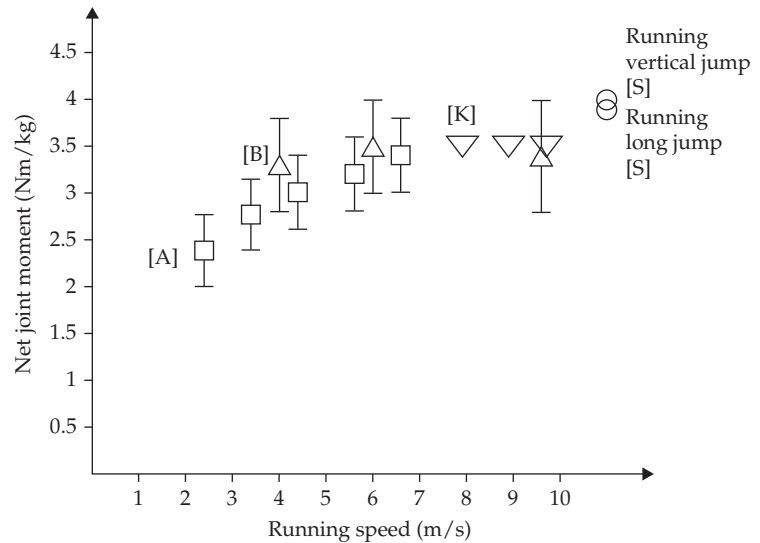


Figure 11.8 Calculated maximum ATFs in different sport activities. (A) Walking, (B) running, (C) sprinting, (D) stopping and landing, (E) backward somersault takeoff. Data from Brüggemann (1985, 1987).

to lead to injury and partial tissue damage or even complete tissue breakdown. Partial or entire tissue damage may interrupt training in sports and exercise which hinders and counteracts performance enhancement. Knowledge on exercise specific tissue loading may help to manage biological tissue loading during training and exercise and has the potential to support performance enhancement and injury prevention. A proper tissue loading is also of relevance in prevention and rehabilitation to allow tissue adaptation and to increase the tissue's potential to perform.

The chapter presented knowledge on neuromechanical loading of tendons, muscles and ligaments of the knee and ankle joint during sport and exercise. The data presented build the basis to estimate activity-specific and tissue-specific neuromechanical loading and have the potential to contribute to choose appropriate loading regimes which ensure tissue adaptation and avoid tissue overloading.

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Chapter 12

Stretch-Shortening Cycle Fatigue

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Introduction

There is no doubt that neuromuscular fatigue is a complex phenomenon. Initiation of the voluntary movement is in itself rather demanding with regard to the several steps required until the final “command” reaches the muscles. Figure 12.1a is an attempt to summarize these various phases. Theoretically, any of these steps in Figure 12.1a could be subject to certain levels of fatigue. In more general terms, the fatigue processes have been classified into two major categories and/or sites: peripheral and central fatigue. This is illustrated in Figure 12.1b, which demonstrates these processes as revealed by motor nerve stimulation. Other methods have also been widely used to characterize central and peripheral fatigue, and the reader is referred to Chapter 8 for additional details. In brief, however, it is often very difficult to separate the central (including supraspinal) and peripheral components from each other. They are likely to occur simultaneously, depending on the specific situation. The potential sites of impairments can be any one link in the long chain from the voluntary motor centers in the brain to the contractile filaments in the single muscle fibers. Any weak link could be responsible for the direct cause of muscle fatigue. Fatigue also involves pathways from the exercising muscle that “inform” the nervous system of the metabolic and mechanical states in the

periphery. It was Asmussen (1979) who opened the way to new thinking of the fatigue mechanisms. Although he did not have strong direct experimental evidence, he was able to propose that “some sort of pain-producing catabolites in the muscle are involved in the central fatigue, as they could elicit inhibitory nerve impulses from receptors (probably from some kind of chemoreceptors) in the fatigued muscles. The inhibition may act on the motor pathways anywhere from the voluntary centers in the brain to the spinal motor neurons.”

Asmussen’s early works (Asmussen, 1934, cited in Asmussen, 1979) revealed separate levels of susceptibility to fatigue in two different peripheral sites: the “transmission mechanism” (neuromuscular junction, muscle membrane, and sarcoplasmic reticulum) and the “contractile mechanism” (muscle filaments) (Figure 12.1a). These observations are still considered as a major principle to emphasize the task dependency of the site of impairments (Enoka & Duchateau, 2008). Figure 12.2 is another elegant demonstration of this task dependency. The classical ergograph task that consisted of lifting weights was performed with eyes closed until exhaustion. The simple instruction to open the eyes allowed the subjects to perform immediately 20–30% more extra work (Asmussen & Mazin, 1978). The reversed strategy (closing the eyes after reaching exhaustion with eyes opened) had no effect.

These observations and hypothetical explanations are very relevant to the neuromuscular fatigue of SSC exercise. The SSC-induced impairments are themselves very complex, as this type of exercise involves a large number of muscles, different forms of muscle actions, different intensities

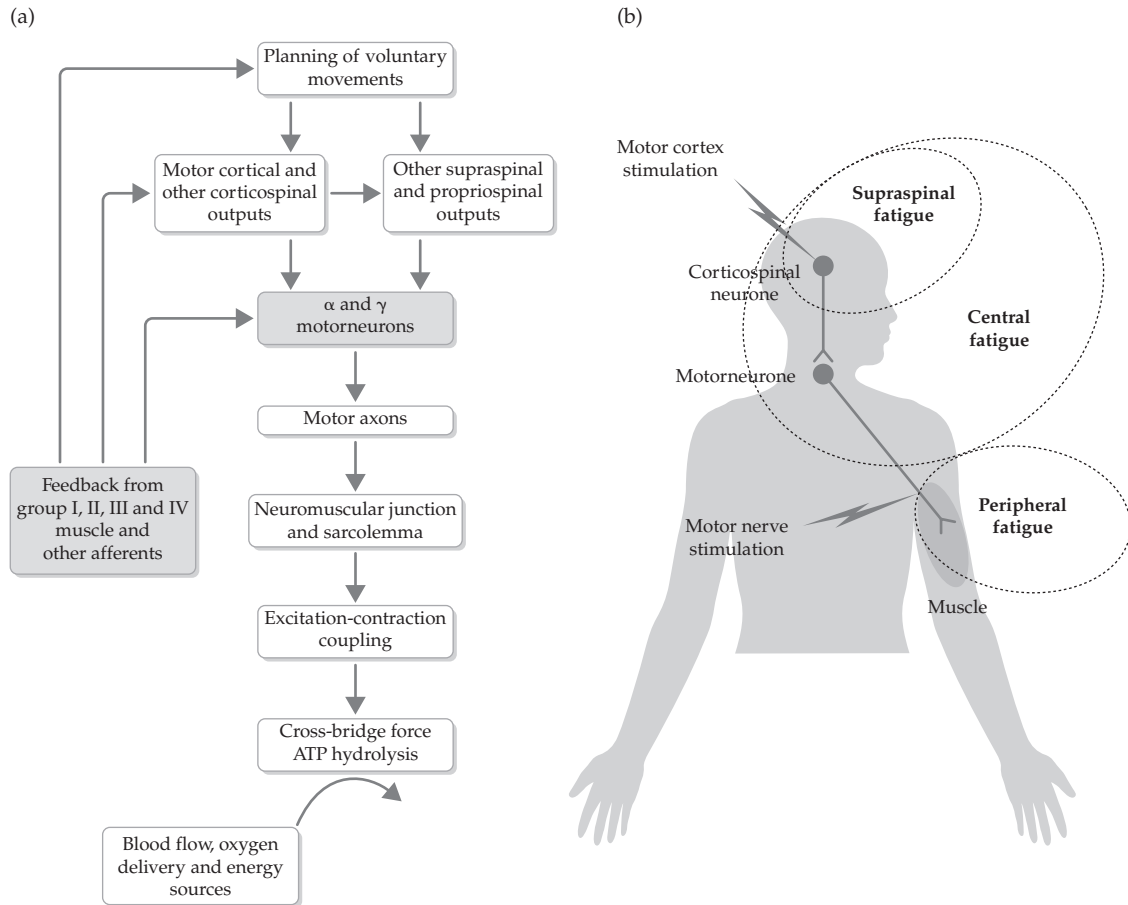


Figure 12.1 (a) The chain of processes leading to voluntary movement. Feedback from muscle afferents is shown going to the motor neurons, the motor cortex, and the premotor areas. (b) Division of muscle fatigue into peripheral and central fatigue by motor nerve stimulation. Supraspinal fatigue is a subset of central fatigue. (Modified from Taylor et al., 2008.)

and time durations (short and intensive versus prolonged exercises), and combined interventions of central and reflex neural components. Furthermore, recovery from SSC fatigue is characterized by the delayed occurrence of muscle pain sensation, defined as DOMS, which influences also the muscle activation profiles.

Neuromuscular Fatigue During Isolated Eccentric Actions

As has been discussed in Chapter 2, the eccentric phase is very dominant and important in SSC. This notion must naturally be seriously considered

when discussing the problems of SSC fatigue. Consequently, the following short presentation highlights some of the major differences between eccentric and concentric fatigue as measured in human experiments and using the specific force development protocol, in which the dynamic muscle actions (either eccentric or concentric) are always performed after full isometric action. This type of recording (and measurement) follows the same principle as used in measuring F–T characteristics of isolated muscle fibers (Edman & Tsuchiya, 1996). As described in detail in Chapter 1, the maximal eccentric action can produce much greater forces than the corresponding concentric one. This concept

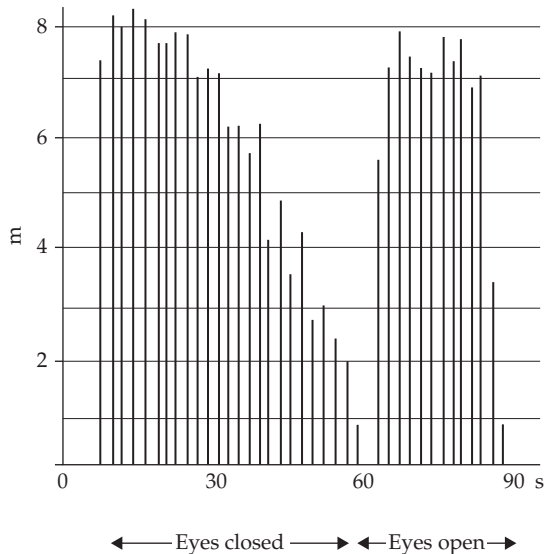


Figure 12.2 Illustration of the “task dependency” from arm ergograph recordings. The lifting task started with subject’s eyes closed till exhaustion, at which time he opened his eyes and continued to exercise. (With kind permission from Springer Science+Business Media: Asmussen, E. & Mazin, B. (1978) A central nervous component in local muscular fatigue. *European Journal of Applied Physiology and Occupational Physiology* 38, 9–15.)

of high mechanical tension in individual muscle fibers and connective tissues put Asmussen (1956) among the very first ones to quantify the performance and muscle soreness differences between fatiguing concentric (positive work) and eccentric (negative work) exercises. He was able to demonstrate for both arm (triceps brachialis) and leg (quadriceps femoris) muscles that the palpable muscular soreness was always present 1–2 days after negative work (eccentric exercise), but hardly ever after positive work (concentric exercise) of the same intensity. This finding has formed a basis—although often without reference to Asmussen’s work—for the concept of DOMS, typically occurring in relation to unaccustomed exercises, specifically including eccentric muscle actions. Asmussen also referred to the possibility of “production of pain-provoking substances in the connective tissue of the mechanically overstrained muscles.” Even before Asmussen (1956), Hough (1902) had suggested that exercise-induced muscle soreness might be due to some

rupture within the muscle, and especially in its connective tissue. Hill (1951) suggested that the soreness was due to mechanical injury, distributed microscopically throughout the muscle. Bøje (1955) as referred to by Asmussen (1956) was “inclined to believe that the pains are located in the intramuscular connective tissues, as they are most apt to develop after exercise that extended the muscles.”

These notions and experiments of Asmussen (1956) can be considered as a clear indication of the “mechanical stress concept” for exercise-induced muscle soreness. Using this principle, we designed a study in which EMG activity and force of the forearm flexors were registered during 40 repeated maximum concentric and eccentric actions (Komi & Rusko, 1974). The velocity of the dynamometer arm was the same in both conditions and the two exercise types (either eccentric or concentric) were separated by a minimum of 2 weeks. To produce the concentric or eccentric force, a special care was taken to secure that each repeated contraction started always with full preactivation before the dynamometer lever arm was activated upward in the eccentric mode and downward in the concentric mode. Thus, in both conditions the starting maximal EMG activity was the same, but due to the difference in the force-producing capacity of the muscle, the maximal concentric force was about 60% of the maximal eccentric force similarly as shown in basic comparison of the forearm flexor concentric and eccentric forces (Komi, 1973). The fatigue experiments showed then that muscle force declined by 50% in eccentric and by 80% in concentric exercise, when the last actions were compared with the initial ones (Figure 12.3). Interestingly, maximal EMG activity of the studied muscles declined continuously during the entire work period of 40 contractions, and the pattern and magnitude of this decrease were the same in both types of exercise modes. In all subjects, the eccentric type of fatigue caused DOMS, which was felt primarily at the myotendinous junction level. Often it was so severe that the subjects could not extend their elbow for a few days after the testing. The recovery from DOMS, which was associated with muscle swelling, lasted for 1 or, sometimes even, for 2 weeks. In none of the cases was any substantial

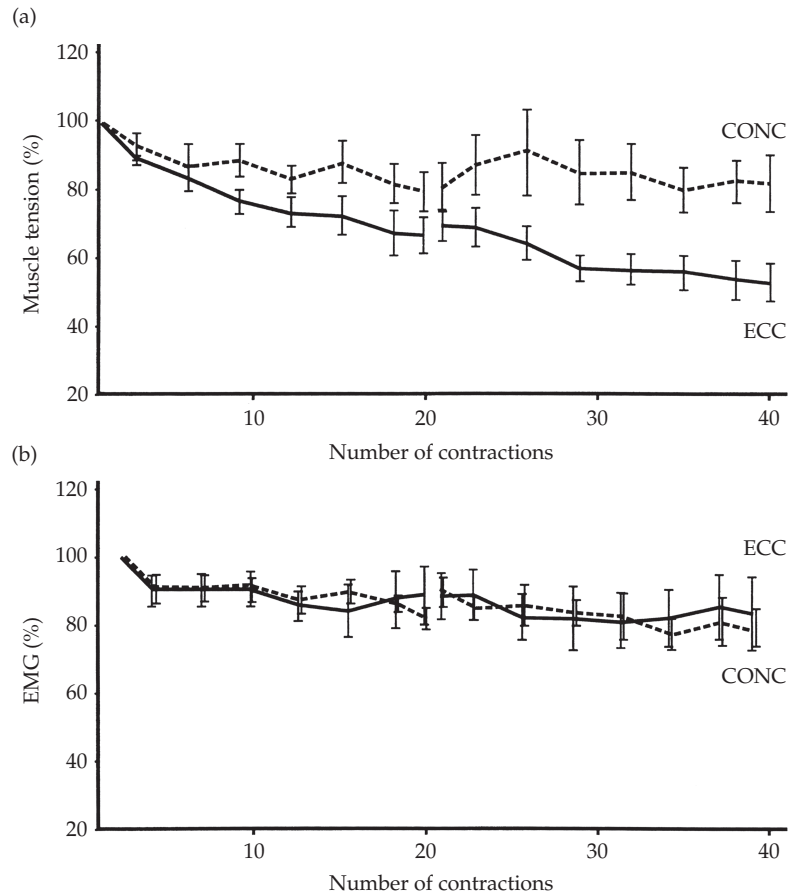


Figure 12.3 Decrease of muscle tension (a) and electromyography (EMG) (b) during the course of 40 repeated maximal contractions of either eccentric (ECC) or concentric (CONC) actions. At the start of the exercise, the absolute maximal forces were 440 N and 245 N, in eccentric and concentric exercise, respectively. The absolute EMG values were the same in both types of maximal actions. Note that despite similar reduction in EMG, the eccentric exercise caused much greater reduction in force. (Komi & Rusko, 1974.)

soreness felt after concentric loading. Symptoms of soreness were very much similar to those observed in an earlier training study (Komi & Buskirk, 1972), when the severe muscle soreness prevented any positive training adaptation for more than 1 week. After disappearance of DOMS, the eccentric training resulted in rapid force increase. It was then quite soon demonstrated that eccentric-induced reduction in performance and associated DOMS was also present in the leg extensor muscles (Komi & Viitasalo, 1977). This can be demonstrated in Figure 12.4, which shows that both the F–T characteristics and EMG/force ratio presented a delayed recovery from eccentric fatigue but not from concentric fatigue.

Today, we know many more details about fatigue influences of eccentric exercise. It has been studied quite extensively in many laboratories both in

animal and human models. A common feature of all these studies is DOMS and the progressive damage process, from which the muscles recover at a slow rate. We now know both the mechanisms of muscle damage and the recovery in many details, although the final answers are yet to be given. This information has also been fundamental in helping the exploration of SSC fatigue.

Fatigue Definition That May Apply to Repeated SSC Loading

From the foregoing discussion, it is evident that to define muscular or neuromuscular fatigue would be almost impossible so that the needs and interests of all working in these problems could be satisfied. Many researchers adopted, however, the definition of fatigue given by Bigland-Ritchie and

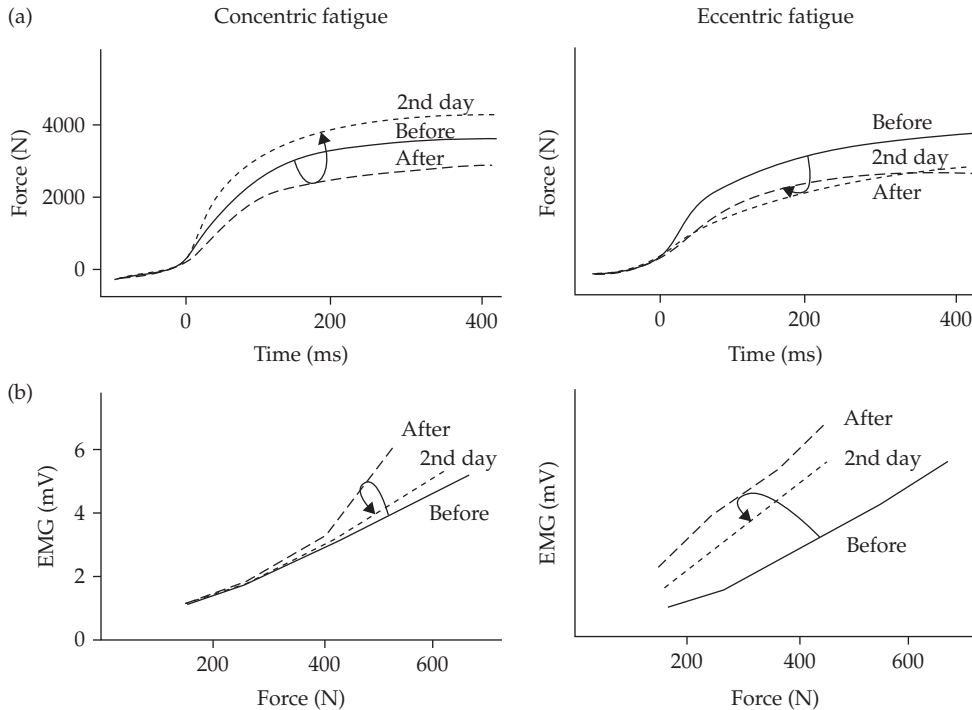


Figure 12.4 Influence of 40 repeated maximum concentric and eccentric actions on the isometric F–T curve of the bilateral leg extension (a) and on the electromyography (EMG)–force relationship in unilateral knee extension (b). Note the delayed recovery of muscle performance following the eccentric fatigue. (From Komi & Viitasalo, 1977.)

Woods (1984): “Any exercise-induced reduction in the ability to exert muscle force or power, regardless of whether or not the task can be sustained.” Accordingly, the inability to continue a task is often termed “exhaustion” and considered as the culmination of these ongoing fatigue processes. The time to exhaustion is clearly dependent on the exercise intensity and on its mode (continuous or intermittent). In a submaximal exercise, fatigue may then occur without any decrement in the task performance as other motor units or muscles are recruited to compensate for those that are fatiguing.

With regard to the SSC task specificity defined in Chapter 2, one can easily see that the loading characteristics during this exercise are different from isolated isometric, concentric, or eccentric actions. SSC is a natural but complex activity, which in itself is often difficult to understand in its all features. First, SSC is characterized by high impact forces that are often repeated over long durations (e.g., during a

marathon). In fatiguing SSC exercises, the impact loads are repeated over time, stressing metabolic, mechanical, and neural components. Intensive and/or unaccustomed SSC-type exercises may thus result in reversible ultrastructural muscle damage and DOMS. Secondly, the impact loads and the nature of stretches involved in the active braking phase of SSC are usually very fast, of short duration and controlled simultaneously by reflex and central neural pathways. The SSC fatigue model is of particular interest as it causes disturbances in stretch-reflex activation and, thus, provides an excellent basis for studying neuromuscular adaptation to exhaustive exercise. Finally, the magnitude of metabolic stress is dependent on the velocity of stretch and the coupling time between stretch and shortening. Extending then the traditional fatigue thoughts to the SSC fatigue requires rearranging of the thinking of the possible mechanisms involved. It also requires complementary techniques of investigation.

Experimental Settings

Short- and Long-Term SSC Fatiguing Protocols

In SSC fatigue protocols, a muscle or group of muscles are subjected to repeated impact/rebound loads for a defined duration and intensity. Repeated hopping on place is the simplest example of such a fatigue situation. In some cases, the fatiguing exercises are of fixed duration, but usually the exercises are performed until exhaustion. Our recent review (Nicol et al., 2006) included 45 SSC fatigue studies performed during the last 20 years on human subjects. Many additional studies have since been published on this topic. Figure 12.5 shows examples of SSC fatiguing protocols varying from prolonged running (30 min to 6 h) to shorter but more intensive rebound series on a specific sledge apparatus (Kaneko et al., 1984; Komi et al., 1987).

The long-lasting SSC performances serve for the purpose to characterize more specifically the effects of a great number of repeated SSC actions on various aspects of the neuromuscular function.

The running protocols may vary from a 10 km run by nonendurance runners (Nicol et al., 2003) to a 65 km ultramarathon race with an altitude difference of 2500 m (8 h 30 min) by well-trained runners (Millet et al., 2002). The cross-country skiing protocols vary similarly, from a skiing marathon (≈ 2 h 30 min) (Millet et al., 2003b) to an 85 km skiing race (≈ 4 – 10 h) (Viitasalo et al., 1982). The braking (or impact) phases are naturally different depending on the activity. Cross-country skiing is a typical form of locomotion, in which the braking phase is long and relatively smooth in both free and traditional techniques. In the latter technique, the vertical ski forces seldom exceed 1.5 times body weight (Komi & Norman, 1987; see also Chapter 3). Consequently, the repeated SSC actions in this event will have much smaller functional, structural, and damaging effects on muscle if compared to running, for example. Except perhaps for ultramarathon run, the impact peak in running is much higher and the duration of the braking (“eccentric” phase) is short (50–120 ms). The repeated loading will consequently have greater stretch-induced

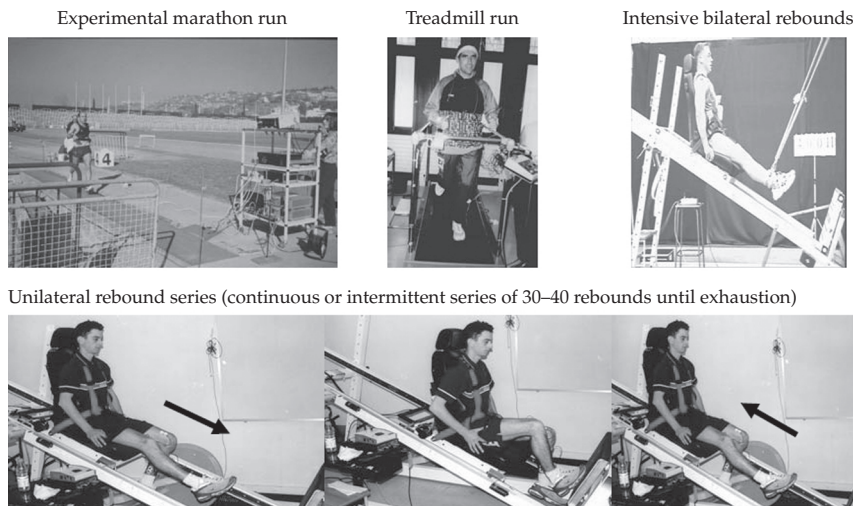


Figure 12.5 Presentation of the different fatiguing SSC protocols. The running protocols may be of either fixed duration (e.g., marathon) or run performed until exhaustion. The bilateral and unilateral rebound protocols performed on the sledge consist of either continuous or intermittent series until exhaustion. The rebound height is usually set at either 70% or 80% of the maximal rebound height. The lower knee flexion reached at impact is also preset at 90–110°, and controlled during the entire exercise by visual feedback. The net duration of the rebound exercise varies from 1–5 min in the continuous protocol to 6–20 min for the intermittent one.

effects than in cross-country skiing (Millet & Lepers, 2004). Thus, the nature of the braking phase is different in cross-country skiing as compared to running. During this phase, both activities induce stretches in the MTU of leg extensor muscles, but the stretch velocities are much higher in running as compared to skiing. These two events represent a good comparison, especially regarding the long-term SSC fatigue effects. It is not possible for a runner to repeat the marathon race in 1-week interval due to the typical SSC fatigue-induced structural damage. In cross-country skiing, even when performed with the same intensity and duration as the marathon run race, the recovery processes from

possible muscle damage take place more rapidly, and the athletes are usually ready to repeat the 50km race after a few days only. This emphasizes that SSC, when repeated long enough and with high intensity, causes reversible neural, structural, and functional disturbances, severity and duration of which are dependent on the nature of SSC task.

The short-term SSC exercise protocols consist usually of intensive and exhaustive series of rebounds performed on the sledge apparatus. By adjusting the subject's position on the gliding sledge, fatigue can be induced selectively in the upper-limb muscles (Figure 12.6) or in the lower-limb muscles (Figure 12.7). The upper-limb protocol

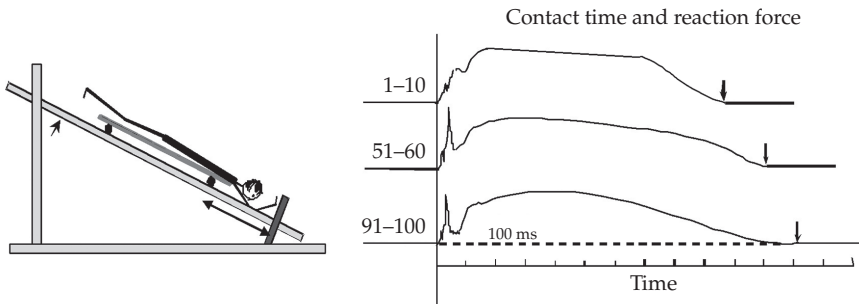


Figure 12.6 Relative changes in kinetics (contact time and reaction force) during the time course of 100 submaximal rebounds with the upper limbs. (Data from Gollhofer et al., 1987b.)

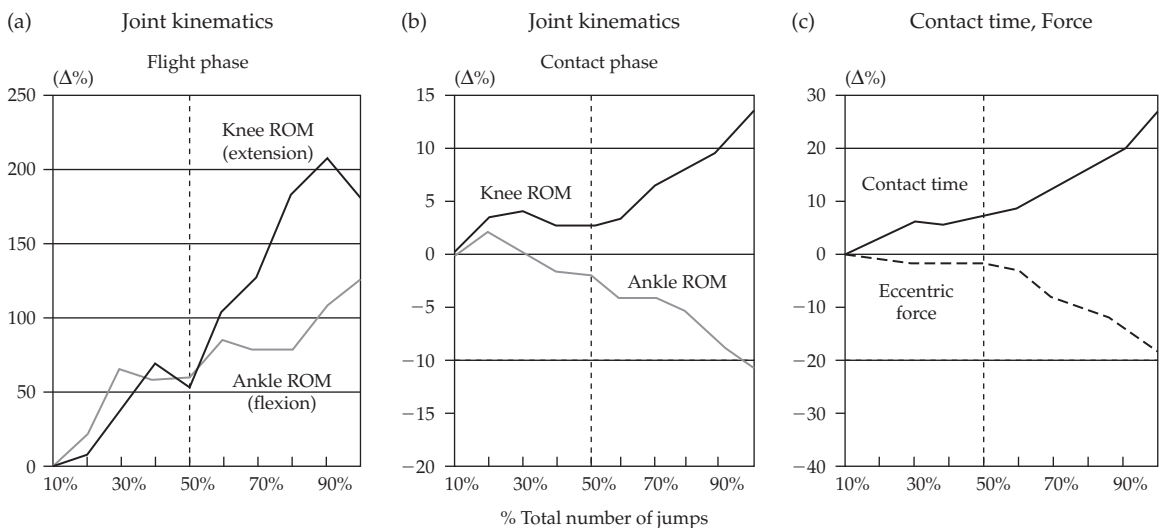


Figure 12.7 Relative fatigue-induced changes in kinematics (a and b) and in kinetics (c) during the time course of SSC rebound exercise performed with the lower limbs. Range of motion (ROM). (Data from Horita, 2000.)

used by Gollhofer et al. (1987a,b) includes 100 submaximal SSC rebounds with both arms. In the basic fatiguing protocol of the lower-limb extensor muscles, the exercise is performed in a sitting position by rebounding as long as possible to a given submaximal rising height. Exhaustion is usually reached after 100–400 consecutive submaximal rebounds (Horita et al., 1996; Nicol et al., 1996b), for a total of 1–5 min of intensive work. The severity of the fatigue protocol can be increased by adding 100 maximal DJs prior to the exhaustive exercise (Nicol et al., 2003). However, facing the premature stop of some of the subjects possibly due to central fatigue and/or rapid metabolic fatigue, the continuous rebound exercise has been recently replaced by an intermittent rebound exercise that consisted of series of 30–40 successive rebounds with intermediate recovery periods of 3 min (Regueme et al., 2005). This resulted in a systematic increase of the net SSC exercise duration (from 6 to 20 min) that secured in most subjects the occurrence of delayed fatigue effects.

Testing Problematics

Although not necessarily typical only for exhaustive SSC exercise, the experiments with this form

of activity have already clearly implied that fatigue responses need to be studied during the course of the exercise itself and during the follow-up period of several postexercise days. Practice has also shown that the immediate postexercise tests repeated at frequent intervals are also very useful. Compromise has to be exercised, however, and therefore the most often used testing times after completion of fatiguing exercise include such time periods as immediately after and 2 h, as well as 2 days, 4 days, and in some instances even 7 or 8 days postexercise. This makes it possible to explore the SSC fatigue mechanisms during the exercise itself, during immediate (acute) recovery phase as well as during the longer-term (delayed) recovery. The 2 h testing point is of particular interest as it reveals in most subjects temporary, but clear functional improvements between the acute and delayed recovery phases (Nicol et al., 2006).

Fatigue During the Course of the Exercise

The fatigue responses during exercise are expectedly very individual, although certain general patterns can be described. The experiments of Horita et al. (2000) (see Figures 12.7 and 12.8) can be used as concrete examples of how the kinematics, EMG,

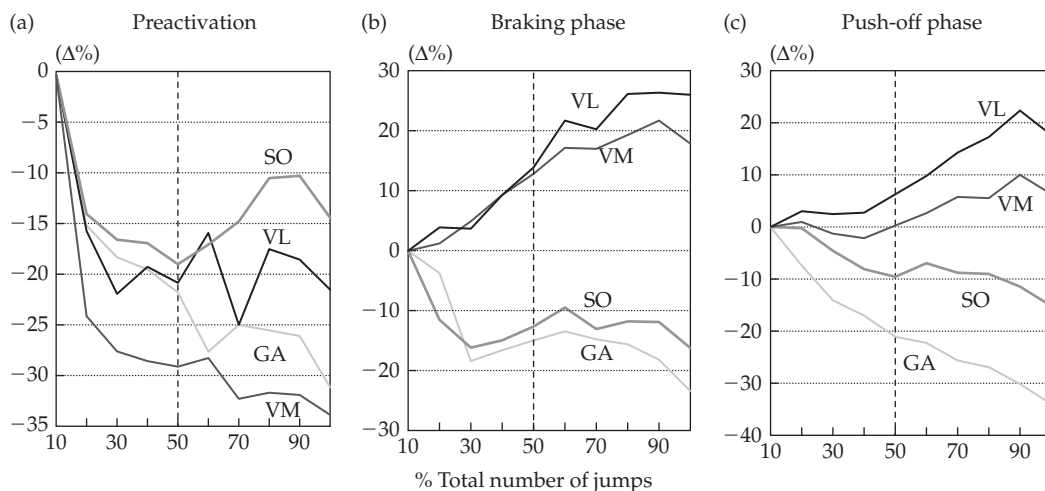


Figure 12.8 (a)–(c) Relative group averaged EMG changes along exhaustive SSC rebound exercise performed with the lower limbs. EMG activity from vastus lateralis (VL), vastus medialis (VM), gastrocnemius (GA), and soleus (SO) muscles. (Data from Horita, 2000.)

and kinetic parameters change during the time course of the fatiguing SSC exercise on the sledge apparatus. During the first half of the exhaustive SSC exercise, the kinetic and kinematic changes were relatively minor, especially in the contact phase, but became clear during the second half of the exercise. Progressive increase in the contact time (see also Figure 12.6) and gradual increase in the initial force peak and its subsequent decrease (fall in the braking force) are characteristics to the fatigue process. EMG activities can also be measured during the contact phase, and their changes are likely to be variable in this multijoint exercise. Reduction in muscle preactivation seems to be very obvious in all muscles in the early phase of the exercise (Figure 12.8), but this common observation is mostly attributed to the improvement of the rebound technique (Regueme et al., 2005). Thereafter, the braking and push-off phase EMGs show additional reduction in the ankle plantar flexors. One can also observe decrease in the SSC efficacy, especially during the braking phase, and the associated increase in the knee extensor EMG activity and EMG/force ratio in the subsequent push-off phase seems to confirm this notion. The push-off phase is then performed with progressively increasing efforts. In such sub-maximal rebound exercises, the subjects make efforts to compensate for the reduced force output by increasing the contact time, relying then more on the push-off phase work output. The push-off phase is more metabolically costly (ME is low, see

Chapter 7), and the failure to continue the work will eventually become evident. This is in line with the progressive increase (up to 30%) in contact time reported by Gollhofer et al. (1987b) (Figure 12.5) at the end of short and intensive SSC rebound exercises performed with the upper limbs.

The marathon run model has also shown similar increases in the ground contact duration (Komi et al., 1986). The fatigue state is characterized by a drop in the force after the impact peak that is likely to be related to the observed faster and longer flexion movement (Nicol et al., 1991b; Horita et al., 1996). In case of the arm exercises, the dramatic increase in the impact peak (Figure 12.6) resulted, most likely, from increased preactivation of the arm extensor muscles (Gollhofer et al., 1987a,b). The increase in preactivation is, indeed, usually associated with more extended limbs and increased muscle-tendon stiffness prior to impact. The subsequent drop in force after impact is currently considered as an important indicator of reduction in tolerance to repeated stretch loads as fatigue progresses (Nicol et al., 2006). A logical consequence of this is that in order to maintain the same SSC performance, e.g., a constant marathon speed or a constant sub-maximal rebound height, the subject must perform greater work during the push-off phases leading to even faster progression of fatigue. Depending upon exercise intensity, this represents a vicious circle leading to a progressive reduction of the capacity to maintain the task. Figure 12.9 represents in more

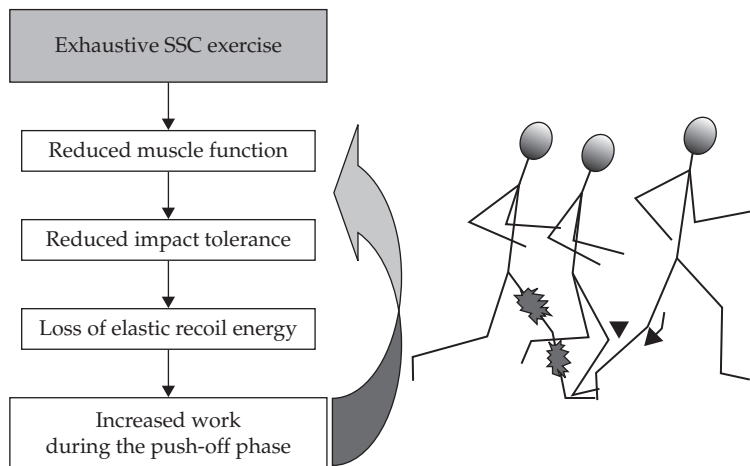


Figure 12.9 Schematic representation of the sequence of events expected to take place during fatiguing SSC exercise. SSC, stretch-shortening cycle. (Modified from Nicol & Komi, 2000.)

practical terms the sequence of events expected to take place during fatiguing SSC exercise.

These overall findings emphasize the pertinence of the SSC model to study the neuromuscular adjustments to the contractile failure and to reveal potential neural limitations. It is suggested that in the nonfatigued state, the muscles are able to damp the impact in SSC by a smooth force increase and smooth joint motion. During submaximal SSC exercises, the exhaustion time is delayed by the intervention of different neural strategies that take place both during the pre- and the postimpact phases.

Intermediate Maximal and Submaximal Testing Tasks

A common protocol used to quantify development of muscle fatigue is to interrupt the ongoing exercise with brief maximal contractions (voluntary or electrically evoked) to estimate the decline in maximal force capacity and voluntary activation (Merton, 1954). It is only recently, however, that SSC fatigue studies used such tests during the exercise.

Place et al. (2004) introduced brief tests every hour during a 5h running exercise performed at moderate velocity. Their findings suggested that maximal voluntary contraction (MVC) capability of the knee extensor muscles was depressed in the final stages of a 5h running exercise, and clear evidence existed for central activation failure as well as for alterations in muscle action potential transmission. Subsequently, Millet and Lepers (2004) compared these results with those obtained during a cycling exercise (Lepers et al., 2002). This comparison revealed a similar time course of MVC reduction throughout the first 3h, but differences occurred after the fourth hour where MVC declined by 14% in running and by only 6% in cycling. As expected, the 5h running exercise ended up with a greater (−28%) loss of maximal knee extension strength capacity than the 5h cycling exercise (−18%) (Lepers et al., 2002).

Girard et al. (2008) examined the time course of impairment in neural and contractile processes every 30 min during a prolonged (3h) high-intensity intermittent exercise (prolonged tennis playing).

Voluntary activation was assessed through three methods: the normalized EMG response (root mean square, RMS/M-wave), the superimposed twitch on MVC, and the 80Hz tetani. The latter technique was applied only before and after the tennis match and to those subjects who could tolerate it. The results revealed progressive but moderate reductions in the maximal voluntary knee extension torque (−9%) that were mainly attributed to central activation failure and alterations in excitation–contraction coupling. The latter hypothesis is well supported by the recent study of Skurvydas et al. (2008) who observed a progressive decrease in both MVC and electrically evoked muscle force at low (20Hz) as compared to high (100Hz) frequencies (P20/P100 ratio) when tested repeatedly after the 10th, 50th, and 100th DJ of an intensive and intermittent jumping exercise.

Progressive development of fatigue may also be investigated by repeating submaximal and/or maximal SSC tests during the course of the exhaustive exercise (Nicol et al., 1991a,b; Skurvydas et al., 2008). For example, the marathon protocol of Nicol et al. (1991a,b) included submaximal and maximal running tests performed on the track every 10 km of the marathon run. Kinetic analysis of the track tests did not reveal any change in the submaximal run performed at the individually fixed initial marathon running velocity (Nicol et al., 1991a). In contrast, however, the maximal running test revealed a parabolic decrease of the sprinting velocity after the first 20 km (Figure 12.10a) with associated decrement in the resistance to the impact load and subsequent increased work during the push-off phase (Figure 12.10b) (Nicol et al., 1991a). The tests of higher loading level (sprint run) may thus reveal a more homogeneous deterioration of the muscle function than the submaximal ones.

Functional Influences

Basic Biphasic Pattern

Intensive and/or unaccustomed SSC exercises induce various impairments of neuromuscular function that are usually biphasic (bimodal) in nature (Figures 12.11 and 12.13). This pattern is

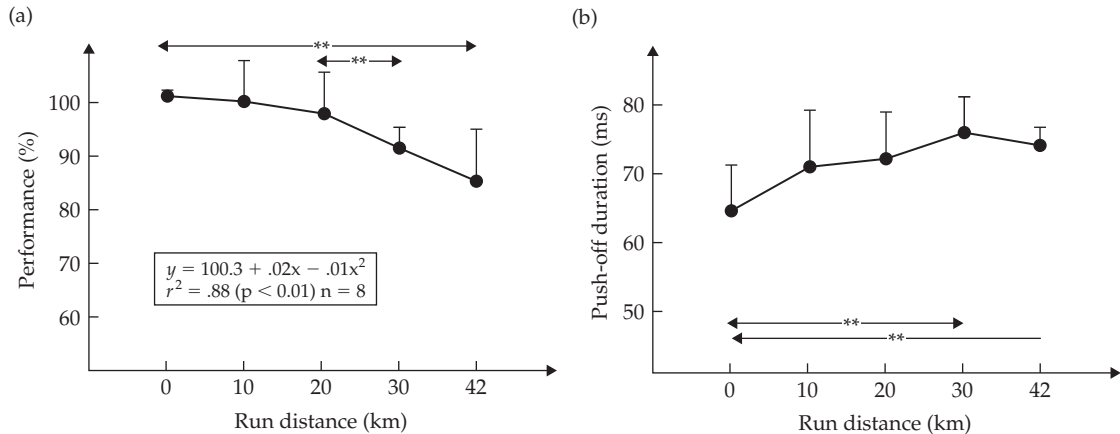


Figure 12.10 Progressive changes in a sprint performance performed every 10 km during the course of a marathon run. (a) Relative change (mean + SD) of the maximal sprint velocity (100% = before marathon value). (b) Duration of the push-off phase of the sprint runs along the marathon. ** $P < 0.01$: significantly different from the pre-fatigue absolute values. (From Nicol et al., 1991c.)

characterized by four different phases of performance reduction and recovery (as shown schematically in Figure 12.11): (1) In this first phase, the performance is reduced toward the end of the exercise (exhausting point) (2) which is then followed by an acute recovery (within a few hours) prior to (3) a delayed reduction and (4) a subsequent slow recovery. The entire duration of the recovery may last up to 7–8 days or in some instances even for 2 weeks. This biphasic pattern is particularly apparent when the SSC exercise has been exhaustive enough. The original concept of biphasic pattern was introduced by Faulkner et al. (1993) for eccentric-type exercise, and then demonstrated in our exhaustive SSC fatigue studies (Nicol et al., 2006).

Similar to eccentric-type fatigue, the exhaustive SSC exercise results in DOMS, which is characterized by a sensation of dull pain and discomfort, increasing in intensity during the first 2 days, remaining symptomatic for 1–2 days, and usually disappearing 5–7 days after exercise. Soreness is not constant, being mostly felt when the exercised limbs are extended or fully flexed, or when the muscles are palpated deeply (Howell et al., 1993). Sore muscles are often stiff and tender, and their ability to produce force is reduced for several

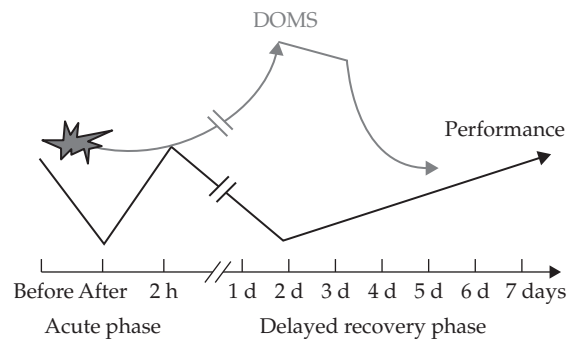


Figure 12.11 Schematic representation of the general trend of changes in performance and delayed onset muscle soreness (DOMS) sensation after exhaustive SSC exercise.

days or weeks (Asmussen, 1956; Komi & Rusko, 1974; Sherman et al., 1984; Nicol et al., 1996a, 2003; Murayama et al., 2000). Particularly important in terms of injury prevention is the timing of DOMS disappearance, as it occurs prior to complete structural and functional recoveries (Figure 12.12). Despite clear occurrence of DOMS, it must be realized that neither the degree nor the timing of ultrastructural damage per se correlates well with the respective changes in DOMS sensation (Howell

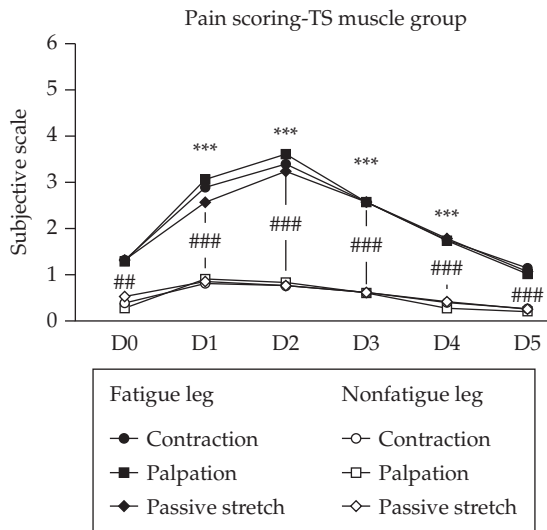


Figure 12.12 Sensation of pain as tested in three different conditions (during palpation, contraction, and passive stretch). The pain scores were reported on a six-level scale—from no pain (0) to extremely sore muscle (6). Regardless of the pain testing procedure, pain was reported only for the fatigued triceps surae muscle group. *** $P < 0.001$: significantly different from the prefatigue (PRE) absolute values; ### $P < 0.01$, #### $P < 0.001$: significant interleg difference. (Adapted from Regueme et al., 2007b.)

et al., 1993; Newham et al., 1983b; Paulsen et al. 2010b). The associated hypothesis of a causal relationship between the inflammatory process and DOMS (Smith et al., 1989; MacIntyre et al., 1995) also remains questionable (Newham, 1988; Malm et al., 2004, Paulsen et al. 2010a,b). DOMS cannot therefore be used to reflect detailed recovery processes precisely.

“Time-Dependent” Effect: A Parallelism Between Neural and Mechanical Recoveries

Based on the published literature until 2008, it appears that approximately 60% of the SSC fatigue studies concentrated on the acute recovery phase (<2h postexercise), whereas the other studies extended their investigation to the delayed recovery phase (up to 8 days). Figure 12.13 shows their combined results in terms of both acute and delayed changes in MVC, voluntary EMG activity, and voluntary activation (by twitch interpolation

technique). These overall results follow the notion of Asmussen (1979), who was among the first ones to suggest that fatigue-induced changes occur in parallel between mechanical and neural factors. The general observation of a biphasic recovery pattern along the several days of the recovery period supports the concept of “time-dependent” effect. In SSC fatigue situations, there is enough evidence to consider this as adjustments that would compensate for contractile failure or protect fatigued muscles from injury (Nicol et al., 2006). These adjustments may be both central and peripheral. Before going into the mechanisms themselves, the following paragraphs will present some major functional changes observed along the biphasic recovery.

Maximal Force Production and Muscle Activation

This notion can be observed simply by following the maximal force and EMG activation over a period of 4–8 days postexercise. The usual finding is that there is an immediate ($21 \pm 8\%$) drop in MVC. When reexamined 2h postexercise either partial or complete recovery of MVC is frequently observed ($-10 \pm 8\%$) (Figure 12.13). Most of these fatigue studies reported an immediate drop in both MVC and voluntary EMG activity followed by secondary reductions on day 2 (2D). On the average, a parallelism is observed between activation and force changes along the 8 days of the recovery period. For the acute drop in MVC, one may conclude that it is quite independent of the fatiguing protocol duration (prolonged versus short and intensive that may vary from 3 min to a few hours). For the delayed recovery phase, however, it is unfortunate that only Avela et al. (1999b) examined these two factors after a prolonged (marathon) SSC exercise. In other studies, the fatiguing protocol was short and intensive, consisting of exhaustive rebounds on the sledge or of a downhill treadmill run. If the fatiguing SSC protocol is intermittent, one may not see any clear drop in MVC or EMG in the acute recovery phase, but delayed fatigue effects still persist in both parameters (Regueme et al. 2005; Girard et al., 2008).

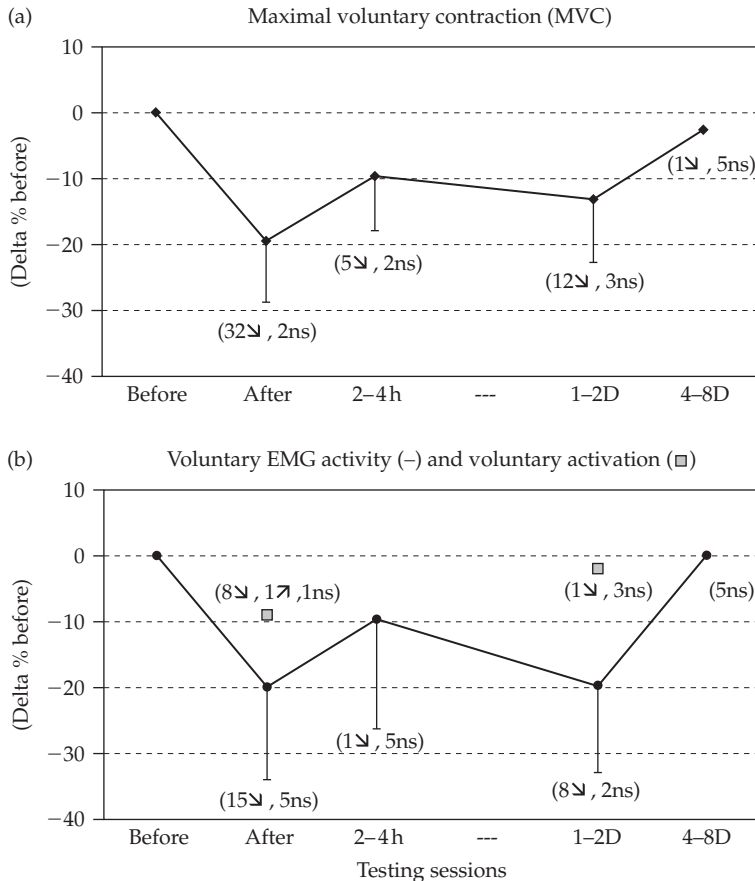


Figure 12.13 Relative acute and delayed reductions in maximal voluntary isometric force (MVC) and electromyographic (EMG) activity (RMS or IEMG) following SSC exercises. (a) MVC data compiled from 31 articles using either knee extensor or plantar flexor muscles and reporting either decreased (\searrow) or nonsignificant (ns) changes. (b) Corresponding changes reported by some of these studies in voluntary EMG activity measured by surface EMG electrodes and in electrically evoked voluntary activation. For individual references refer to Nicol et al. (2006).

The rate of force development (RFD) is also reported to be dramatically reduced and accompanied by a slow recovery (Pullinen et al., 1999) (Figure 12.14). In accordance again with the concept of parallel neural and mechanical changes with SSC fatigue, recovery of both maximal EMG and RFD remained incomplete until the sixth day postexercise (Pullinen et al., 1999).

Although recordings of the EMG demonstrate persisting reductions for a few days after exhaustive SSC exercise (for a review see Nicol et al., 2006), caution should be exercised when interpreting surface EMG changes during muscle fatigue. In particular, it cannot be concluded from the sole RMS or IEMG data that the measured decreases result entirely from the occurrence of central fatigue. To take into account potential impairments in neuromuscular transmission and action

potential propagation in muscle fibers, one of the common techniques consists in expressing voluntary EMG activity over the M-wave (e.g., RMS/M). Immediately after a ski-skating marathon (Millet et al., 2003b), RMS was thus found to decrease but RMS/M was unchanged. Minimizing the role of a reduced M-wave in the acute recovery phase after prolonged running, however, both RMS and RMS/M parameters were significantly reduced (Millet et al., 2003a; Place et al., 2004; Racinais et al., 2007). In the running study of Gauche et al. (2006), this was still the case when checked 24 and 48 h postexercise. Furthermore, the extended follow-up of the recovery period (over 6–8 days) after a marathon run (Avela et al., 1999b) as well as after exhaustive rebound protocol on the sledge (Kuitunen et al., 2004; Dousset et al., 2007) revealed very limited and nonsignificant changes in maximal

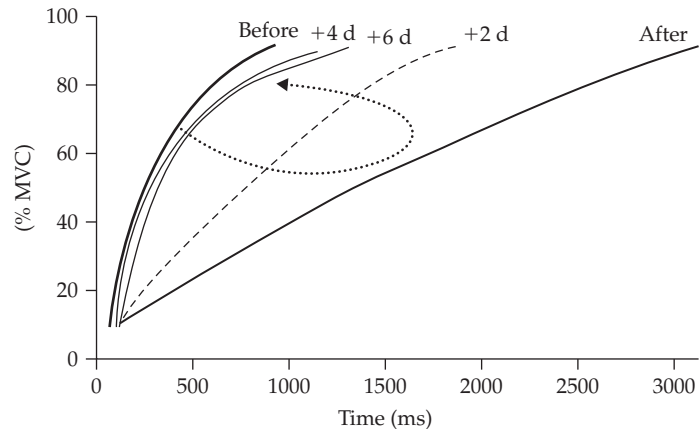


Figure 12.14 Relative F-T curves of the maximal knee extension measured in isometric condition immediately before and after a marathon run as well as 2, 4, and 6 days later. Note the large and immediate decrease in the relative RFD and its slow recovery toward the fourth day post-marathon. MVC, maximal voluntary contractions. (Data from Pullinen et al., 1999.)

M-wave whereas biphasic decrements were observed in maximal RMS and/or in voluntary activation level. Finally, it is noteworthy that the reported changes in the M-wave amplitude are quite variable among SSC studies and between subjects (for a review see Millet & Lepers, 2004). Consequently, further studies are needed to confirm the potential contribution of M-wave changes to the SSC recovery phenomena.

Another way to assess the “central” versus the “peripheral” origins of the decreases in voluntary EMG activity and force production is to estimate “voluntary activation” by the use of the twitch interpolation technique. In the SSC literature, it is only recently that some studies used this technique and their combined results are presented in Figure 12.13. In the acute SSC recovery phase, no significant change was observed after continuous and intermittent 90min runs (Racinais et al., 2007), but an activation deficit was reported after longer ones (Millet et al., 2002, 2003a; Place et al., 2004; Saldanha et al., 2008). Supporting our introductory comments on the running versus cross-country skiing comparison, no deficit in voluntary activation was found after a freestyle ski-skating marathon despite of a similar drop in force than after a 30km run of same duration (Millet et al., 2003a). For the delayed recovery phase, the lack of estimation of voluntary activation is more problematic as only a few SSC fatigue studies have examined this phase with the EMG technique (Kuitunen et al., 2004;

Martin et al., 2004; Dousset et al., 2007; Ross et al., 2007). From these publications, only one (Dousset et al., 2007) reported a significant (6%) drop in voluntary activation at 2D whereas no change was found in the other reports.

Maximal and Submaximal SSC Testing Conditions

Although maximal isometric tests often reveal the effects of SSC fatigue, maximal dynamic tests seem more meaningful mechanistically. In dynamic tests involving slight or no ground impact such as in CMJs and SJs, only a few studies (Lepers et al., 2000) reported a significant change after SSC fatigue. However, when those tests are maximal and involve high ground impact peaks (DJ, five jumps, and sprint), significant acute (Nicol et al., 1991b) (Figure 12.15) and delayed (Nicol et al., 1996b; Horita et al., 1996, 1999, 2003) reductions in performance have been demonstrated after exhausting SSC exercise. After fixed fatiguing rebound protocols including 100 maximal DJs, however, Skurvydas et al. (2002) reported similar acute reductions in SJ, CMJ, and DJ performances. This type of protocol may still lead to a significant reduction in maximal DJ up to 72h postexercise (Skurvydas et al., 2008).

In these maximal situations, where stretch loads are high and muscle stiffness must be well regulated to meet the external loads, the decline in

performance is typically characterized by a large peak force reduction (PFR) after impact. These findings have then led to the hypothesis that the observed loss of tolerance to ground impact could

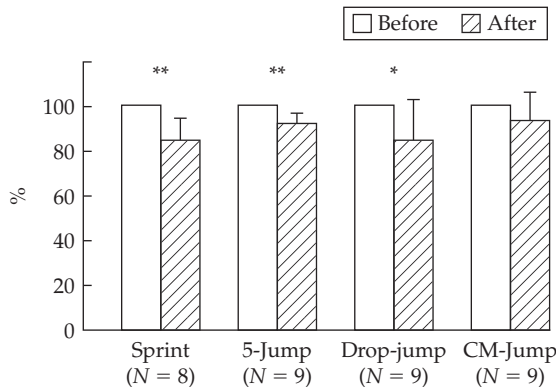


Figure 12.15 Relative changes in the before–after marathon of the maximal running, jumping, and isometric performances. SPRINT: 10 m sprint, 5-JUMP: 5 jump series, DROP-JUMP: drop-jump from a 50 cm height, CM-JUMP: countermovement jump. These subjects showed a mean 22% reduction in maximal isometric contraction. *,** $P < 0.05$, and $P < 0.01$ in before–aftermarathon comparison. (Modified from Nicol et al., 1991b.)

result from a reduced contribution of the monosynaptic stretch-reflex to the leg stiffness (Nicol et al., 1991a,b) (Figure 12.16).

In SSC-type performances, both central and reflex adjustments are indeed operative and expected to contribute to the observed changes in the post-landing stiffness along the recovery period. The submaximal SSC tests emphasize the flexibility of the neuromuscular adjustments between the acute (POST) and the delayed (2D) recovery phases. In a submaximal rebound test, Regueme et al. (2005) demonstrated varying neural changes immediately after (POST) and 2 days after (2D) an exhaustive SSC exercise (Figure 12.17). In the POST testing session, large increases in SOL activation occurred both during the preactivation and the early braking phase. Differing from these POST exercise observations, the increased SOL preactivation at 2D was not associated with any increased activation during the early braking phase, but during the late push-off phase. This shift of muscle activation may thus be considered to reflect a neural strategy to protect the recovering muscles at 2D point from the stressful stretching phase, while securing the rebound performance by increased work during the push-off phase.

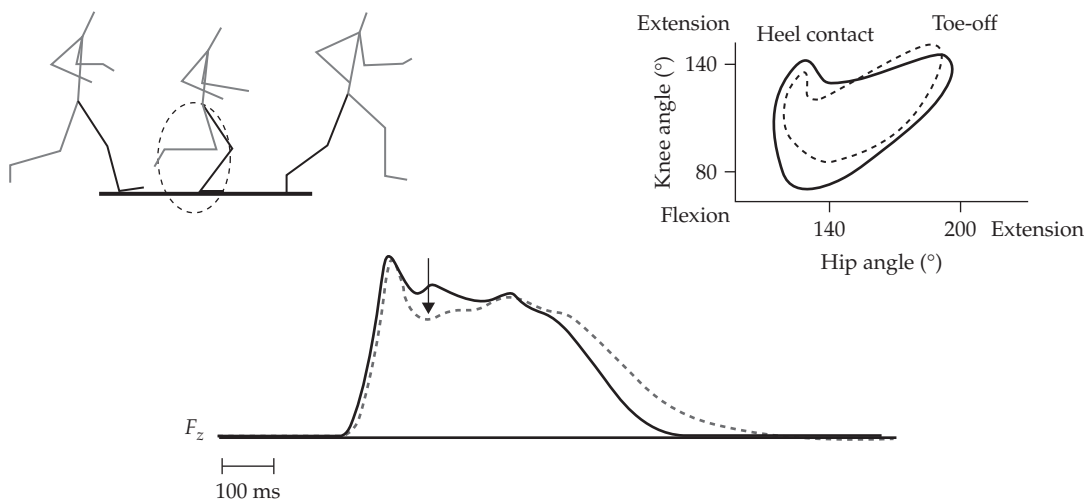


Figure 12.16 Schematic presentation of the hypothesis of the fatigue-induced loss of resistance (tolerance) to impact. This is likely to result at least partly from a reduced contribution of the short-latency stretch reflex (M1) during the early braking phase. (Modified from Nicol et al., 1991a,b.)

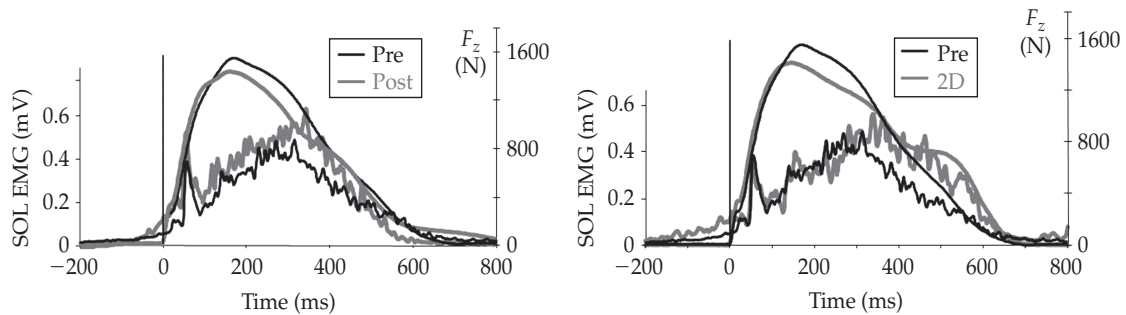


Figure 12.17 Comparison of the group-averaged records of vertical reaction force (F_z) and soleus (SOL) electromyographic (EMG) activity recorded in a submaximal rebound test before and after exhaustive SSC exercise (PRE–POST) as well as 2 days later (PRE–2D comparison). (With kind permission from Springer Science+Business Media: Regueme, S.C., Nicol, C., Barthélemy, J. & Grélot, L. (2005) Acute and delayed neuromuscular adjustments of the triceps surae muscle group to exhaustive stretch-shortening cycle fatigue. *European Journal of Applied Physiology* 93, 398–410.)

Differing from the increase in central activation (preactivation and braking phase activation prior to the stretch-reflex M1 response) in POST, the M1 reflex response of SOL did not increase at 2D. The M1 reflex response was even found to decrease despite an increased preactivation. These data give some support to the hypothesis of a biphasic reduction of the stretch-reflex response. Furthermore, the 2D test revealed a 36% increase in GA muscle preactivation. In line with the earlier mentioned results of Horita (2000) (Figure 12.17), this may indicate progressive intermuscular compensations for the exercise-induced contractile failure. Finally, even though the EMG analysis of submaximal SSC tasks is shown to reveal the existence of a contractile failure, this may not necessarily be detected through the kinematic and kinetic analysis of the movement (Finni et al., 2003; Kyröläinen et al., 2000).

Stretch- and H-Reflex Responses

As it is not always easy to isolate the stretch-reflex EMG response from the global EMG recordings, potential effect of SSC fatigue on the EMG reflex response have been examined, at first, in *passive reflex testing conditions* (Nicol et al., 1996b). A powerful engine was used to induce passive stretches of the shank muscles at slow and intermediate angular velocities (0.44–1.9 rad/s). Intensive SSC rebound exercises were found to result in a

biphasic trend of decline in the peak-to-peak EMG reflex response to passive stretches after a marathon run (Avela et al., 1999a) as well as after a very intensive rebound exercise on the sledge (Nicol et al., 1996b).

This notion gets support from the findings that the active M1 response (measured during maximal rebound test) also demonstrates a biphasic trend of recovery (Figure 12.18a and b). Figure 12.19 is drawn for the purpose to combine the results of available literature and, consequently, to convince the reader that this biphasic pattern is not only an isolated phenomenon, but a very common finding in connection with exhaustive SSC exercise.

Attempts have been made to include H-reflex to the category of “neural” parameters in the follow-up measurements. After running exercises, only acute reduction of the normalized H-reflex response (H/M ratio) have been observed (Bulbulian & Bowles, 1992; Avela et al., 1999b; Kuitunen et al., 2004) whereas biphasic responses of SLR, M1, and H/M ratio are reported after intensive rebound exercise on the sledge (Doussset et al., 2007). This suggests that the spinal excitability may also be influenced during both acute and delayed phases of the SSC recovery. When exhaustive SSC exercise was performed repeatedly on days 0, 5, and 10, the H/M ratio was found to remain decreased at the level of –40% when measured 15 days after the first bout of exercise (Nicol et al., 1996a).

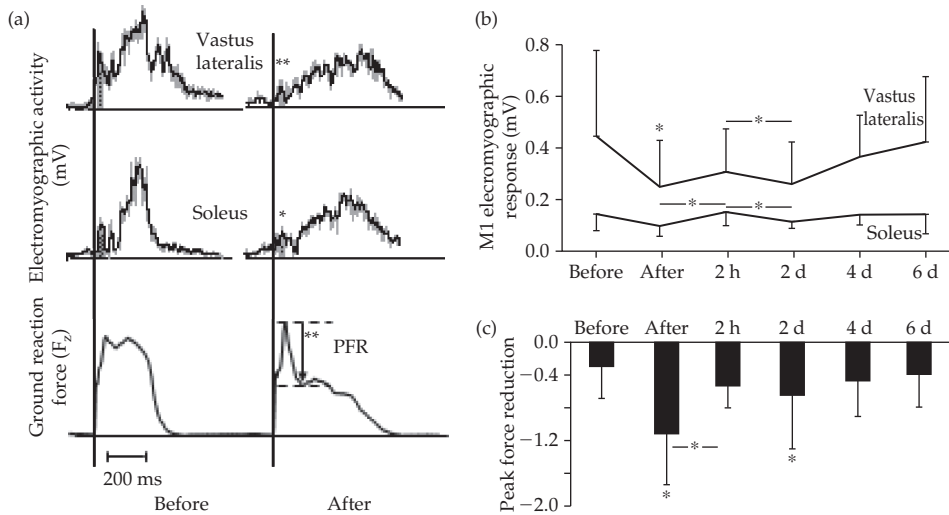


Figure 12.18 SSC fatigue effect on the active short-latency stretch-reflex (M1) response and associated changes in the rebound kinetics. (a) Biphasic recovery of EMG activity and vertical GRF over time, during 10 successive sledge rebounds performed before and after marathon running. (b) Corresponding active short-latency reflex component (M1) of EMG activity. (c) Postlanding stiffness regulation as reflected by peak force reduction (PFR) measured from the normal component of the GRFs. (Adapted from Avela et al., 1999b.)

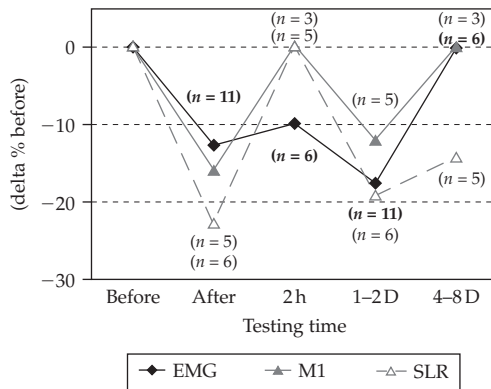


Figure 12.19 Relative acute and delayed decrements following SSC exercises in maximal voluntary EMG activity (EMG), in the passive short-latency stretch-reflex response (SLR), and in the active (M1) one. Data compiled from 14 published articles testing either knee extensors or ankle plantar flexors. For the group mean calculation (n = number of studies), the nonsignificant (ns) values have been replaced by the null value. For individual references, refer to Nicol et al. (2006). Reproduced from Nicol, C., Avela, J. & Komi, P.V. (2006) The stretch-shortening cycle: A model to study naturally occurring neuromuscular fatigue. *Sports Medicine* 36, 977–999. With permission from Adis, a Wolters Kluwer business (© Adis Data Information BV 2006). All rights reserved.

Finding of the parallelism between neural and mechanical responses is a very common phenomenon, and other examples besides those shown in Figure 12.18 are available. Figure 12.20 is an illustration of the biphasic recovery pattern demonstrated in the passive mechanical reflex response after prolonged (10 km run) as well as after short but intensive (rebounds on the sledge) SSC exercises (Nicol et al., 2003).

As expected, however, both measures demonstrate great interindividual variability, expressing therefore the individual nature of the recovery patterns across subjects. The interindividual variability needs to be taken seriously also for the reason that some subjects may even show reflex facilitation in the acute recovery phase (after 2 h), in both passive and active stretch-reflex responses (Kuitunen et al., 2004; Nicol et al., 2003). Consequently, the biphasic shape of recovery should not be generalized as being similar in all subjects. Some subjects simply recover faster than others and experimental evidence supports this notion (Nicol et al., 2003). Timing of the biphasic pattern is expected to vary among individuals.

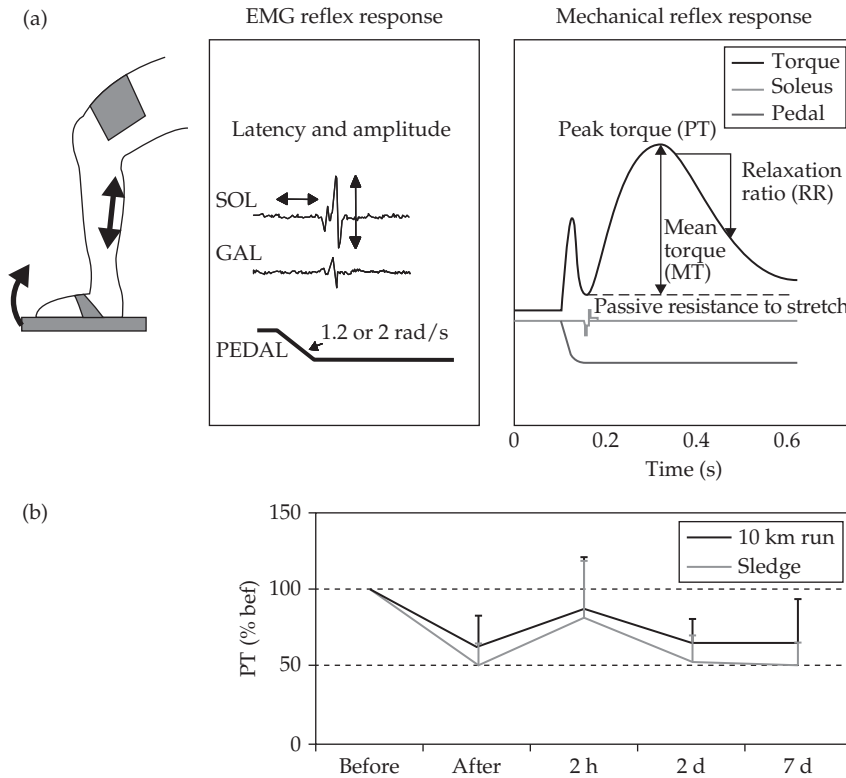


Figure 12.20 (a) Schematic presentation of the passive stretch-reflex test with the analysis of the associated electromyographic (EMG) (left) and mechanical (right) reflex responses. (b) Biphasic recovery patterns of the peak torque (PT) of the mechanical reflex response when recorded after a 10 km run or an intensive rebound exercise on the sledge ergometer. (Data based on Nicol et al., 2003.)

These overall results demonstrate that muscle function and stiffness regulation may be disturbed in a delayed, but individual manner after exhaustive-type SSC exercise. On the other hand, a clear parallelism exists between the respective changes in performance, in neural (central and peripheral) activation. This implies existence of potential coupling between the contractile type of failure and the central and peripheral adjustments that take place along its recovery.

“Testing-Task” Influence on the Activation Pattern

If the “time dependency” (biphasic pattern) is a factor to take into consideration in SSC fatigue

studies, the “testing task-dependent” effect should be equally considered.

Maximal Versus Endurance-Type Isometric Tests

In sustained submaximal isometric contractions, EMG amplitude is reported to increase during the course of the exercise, and to remain less than maximal at the limit of endurance (Gandevia, 2001). According to our knowledge, only one study (Nicol et al., 1991b) has examined the influence of an exhaustive SSC exercise (marathon run) in both maximal and submaximal voluntary isometric tasks. The post-marathon test was found to start at a higher EMG activity level and to end at a similar terminal value than the pre-marathon

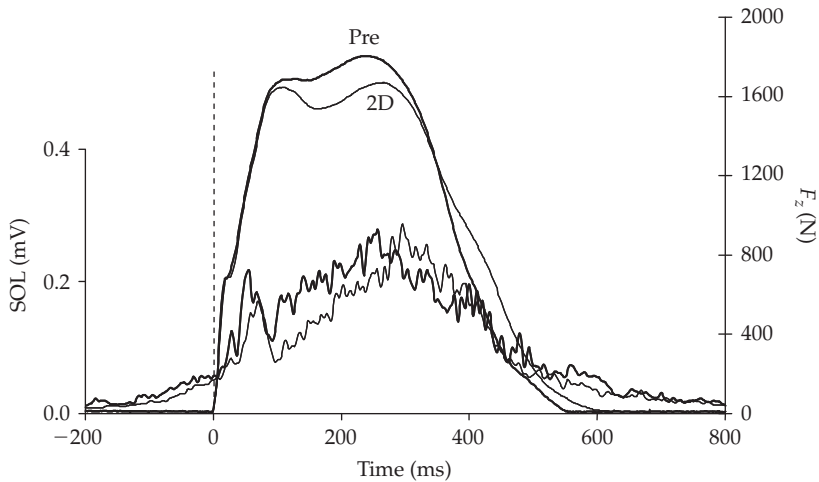


Figure 12.21 Comparison of the group ($n = 10$) averaged records of vertical reaction force (F_z) and soleus (SOL) EMG activity recorded in maximal DJ on the sledge ergometer at two measurement points after exhaustive SSC exercise: prior to (PRE) and 2 days after the exercise (2D). (From Regueme et al., 2007b.)

one. Although this led to an earlier stop of the exercise, the initial increase of the neural drive is likely to reflect the recruitment of additional motor units in order to compensate for fatigue of active muscle fibers. This is in contrast with the parallel and systematic drops in maximal force and voluntary activation observed in the MVC test.

Maximal Versus Submaximal SSC Dynamic Contractions

The hypothesis of “testing task-dependent” effects is supported by the opposite trends of neural adjustments to fatigue reported in submaximal (Regueme et al., 2005) as compared to maximal (Avela et al., 1999b; Regueme et al., 2007b) rebound test conditions.

In maximal DJ performances that require an appropriate muscle stiffness regulation to meet the impact load, the decline in performance is typically characterized by a loss of resistance to impact (PFR after impact) (Figure 12.18a and c). As illustrated in the left graph of Figure 12.21, this is usually associated with clear EMG reductions during both the centrally programmed preactivation phase and the subsequent braking phase. Simultaneously, observed reductions in the active short-latency stretch-reflex response (M1) suggest that the observed EMG changes in SSC tasks are both central and reflex in nature. These observations support the existence of neural attempts to protect the

fatigued muscles in maximal SSC (e.g., DJ) conditions. Based on the limited reductions observed in maximal SSC performances as compared to those measured in single-joint MVC tests (Nicol et al., 1991b,c; Regueme et al., 2007b), this might include compensatory intervention of less fatigued synergist muscles.

In submaximal SSC tests, the contractile failure of the fatigued muscles may be successfully compensated by the increase of their activation during the preactivation phase (Bonnard et al., 1994; Regueme et al., 2005) as well as during the braking and/or the push-off phases (Nicol et al., 1991a). Consequently, SSC testing tasks must be selected carefully, when attempts are made to reveal the variety of neural adjustments that may compensate for the “exercise-induced reduction in the ability to exert muscle force or power, regardless of whether or not the task can be sustained.” Both the task itself and its timing along the recovery phase need to be considered simultaneously.

It must be added here that centrally programmed and reflex activation changes may be opposite in a given task (central facilitation versus peripheral inhibition). They may vary depending on the task complexity (single- versus multijoint), task intensity (maximal versus submaximal), and recover at different pace (acute versus delayed). It is very likely that in order to observe true changes in the kinematic parameters, one may need to fatigue the subjects exhaustively as this was the case (Figure 12.22c) for

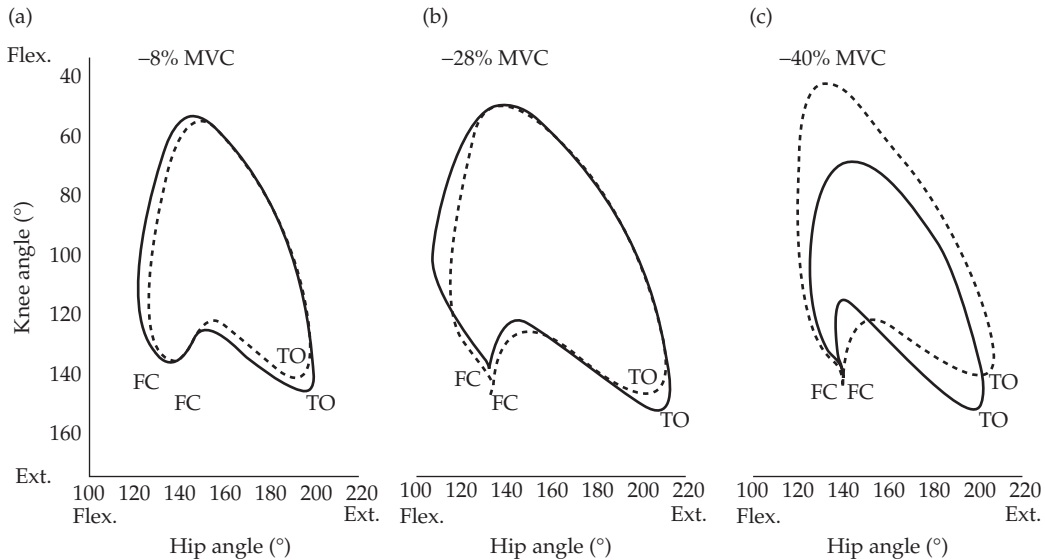


Figure 12.22 Examples (subjects a, b and c) of hip-knee angle cyclograms measured before (cut line) and after (black line) the marathon at the fast (4.5–6 m/s) treadmill running speed. Their respective drops in maximal knee extension (MVC) test are mentioned earlier in the cyclograms. MVC, maximal voluntary contractions. (Adapted from Nicol et al., 1991a.)

the most fatigued runner of our marathon study (Nicol et al., 1991a).

Any Contralateral Influence?

The preceding paragraphs have included material with the assumption that the SSC fatigue effects are primarily unilateral in case the exercise has been performed with one limb only. There is, however, information in the literature that could imply contralateral effects as well. The general existence of “contralateral irradiation” of unilateral activation has been known for a century (Sherrington, 1906). Since then, growing evidence exists that contralateral improvements in motor skill and strength rely on “crossover effects” of unilateral motor and sensory activity (Hortobagyi et al., 2003; Lee & Carroll, 2007).

As muscle fatigue is typically associated with motor and sensory changes, fatiguing voluntary efforts on one side may thus be expected to alter voluntary activation of homologous muscles on the other side of the body. Leading to short-term muscle disorders, with reversible muscle damage, inflammation, and pain, exhaustive SSC exercise

are thus of particular interest. However, the available knowledge regarding the contralateral effects of fatiguing unilateral SSC exercise is not very consistent. Even when Regueme et al. (2007b) applied a multiple set of tests, the overall fatigue effects were restricted to the exercised leg in both uni- and bilateral maximal SSC tests. Based on these results, it seems that acute muscle disorders induced by SSC exercises do not lead to contralateral changes in performance in either single or intermittent maximal motor tasks.

However, some evidence of crossover of central fatigue exists (Regueme et al., 2007b), thus emphasizing the needs to be explored further. As suggested by Zijdwind and Kernell (2001) and recently confirmed by Rattey et al. (2006), it seems that “crossover” of central fatigue is more likely to occur in moderate but sustained contractions than in maximal but brief motor task.

Delayed Influence on the Sense of Position and Velocity

Exhaustive SSC exercise is also expected to result in modification of the static position acuity. Regueme

et al. (2008) attempted to provide evidence that this indeed happens. The position sense test was carried out before an exhaustive rebound exercise on the sledge, and was repeated at the expected times of major inflammation and pain (2D), and at complete recovery (day 8, 8D). In contrast to the control (nonexercising leg), testing of the fatigued leg revealed constant coding errors at 2D, suggesting systematic overestimation of the target dorsiflexed position held with the exercised ankle. The EMG analysis performed for both agonist and antagonist muscles supported these findings and showed especially at 2D significant increase of the agonist/antagonist EMG ratio as compared with the pre-fatigue testing session. In agreement with the antagonist (stretched) muscle influence on the position sense (Roll et al., 1989; Ribot-Ciscar et al., 2003), the overestimation of large dorsiflexed position is mostly attributed to potential SSC fatigue effects on ascending proprioceptive afferents issued from the exercised/inflamed antagonist muscles. Regueme et al. (2007a) had earlier observed that perception of movement velocity can also be decreased during the delayed recovery phase from the typical SSC exercise.

Consequently, and despite a relatively few interest to study sensing of position and velocity in connection with exhaustive SSC exercise, it can be suggested that SSC fatigue can result in proprioceptive disturbances lasting for a few days. This notion gets support from either animal (Pedersen et al., 1998) or human (Paschalis et al., 2007) observations and suggests that there is a decrease in sensitivity of the primary endings during the delayed phase of the SSC recovery period.

Fascicle–Tendon Interaction

Muscle (fascicle)–tendon interaction has received considerable interest during the last 10 years (for a review see Ishikawa & Komi, 2008, and also Chapter 10). Unfortunately and to the best of our knowledge, ultrasound research has not been used much at all for the purpose to reveal possible disturbances and their recovery in relation to SSC fatigue. Ishikawa et al. (2006) are perhaps the only ones who applied the SSC fatigue model to measure fascicle length changes in resting (passive) and

isometric (active) conditions. They found that the fascicles were lengthened in a passive (resting) situation immediately after the exercise and remained lengthened until 2 days (2D) postexercise. During maximal isometric action, the final fascicle length measured at peak force was also longer at 2D, and recovered completely to the original length at 8D postexercise. It is too early to interpret these findings in terms of functional mechanisms. More studies are needed with the ultrasound method that should also take into consideration the entire fascicle–tendon interaction.

Potential Mechanisms Related to Performance Deterioration and Recovery in Connection with Exhaustive SSC Exercise

The preceding sections have emphasized the biphasic pattern of the performance deterioration after exhaustive SSC exercise. To answer the questions of possible mechanisms for this pattern, we would like to concentrate on the situations where the SSC fatigue has indeed been exhaustive in nature. As previously discussed, SSC exercises load the neuromuscular system in a more complex way than any isolated form of muscle actions. DOMS is often discussed in connection with muscle damage, as already introduced in the section on “Basic Biphasic Pattern” and in Figure 12.11. Due to limitations in available space, the reader is also referred to specific reviews on that topic (Cheung et al., 2003; Taylor et al., 2008).

Stretch-shortening cycle fatigue may thus be considered as a model to examine the potential neural adjustments to short-term and reversible structural changes of the MTC. As in the case of isolated forms of muscle action, we are dealing with both central and peripheral neural adjustments to fatigue that may be thought to differ between the acute (<2h) and the delayed recovery phases. The delayed and perhaps the more relevant recovery phase is, in particular, usually related to the natural time course of inflammatory and/or remodeling processes related to muscle damage. As the biphasic pattern particularly applies to exercise-induced changes in stretch-reflex response, we will then concentrate on the potential “alteration in the

fusimotor-muscle spindle function." Considering the combined testing time and testing task effects on the neural strategy, special emphasis will be put on the potential influence of sensory inputs at spinal and motor cortex levels.

Structural Changes and Subsequent Inflammation/Remodeling Process

The overall process may be considered on the one hand as following the classical "Armstrong's model" (Armstrong, 1990; Armstrong et al., 1991) that differentiated four subsequent phases for exercise-induced muscle damage and ensuing inflammation: the initial, autogenetic, phagocytic, and regenerative phases. As the exercise-induced ultrastructural muscle damage is reversible, these phases include the whole process from initial damage to repair. At the anatomical level, injuries are commonly divided into shearing injuries, in which both myofibers and framework are torn, and injuries in situ, in which only myofibers are damaged, such as after repeated eccentric contractions. Shearing injuries result mainly from direct trauma to skeletal muscle, strain injuries, while in situ injuries follow exhaustive exercise, the application of local anesthetics, or are caused by diseases (Bigard & Fink, 2002). Exercise-induced injuries per se may be regarded as a continuum from mild to pathological-like changes, the latter stage being possibly induced by intense eccentric protocol in untrained subjects (Lauritzen et al., 2009; Paulsen et al., 2010b).

In line with Armstrong's model, muscular responses to exercises involving intense and/or unaccustomed eccentric muscle actions are usually explained by the classical "damage–inflammation–repair" pathway (MacIntyre et al., 1995; Clarkson & Sayers, 1999). After SSC exercise, it is noteworthy that cytoskeletal and myofibrillar abnormalities are lower immediately after exercise, as compared to those observed 2–3 days later (Fridén et al., 1983; Newham et al., 1983a; Hikida et al., 1983). The delayed amplification of muscle damage gives support to the rapid functional recovery, usually observed 1–2h after SSC exercise, and before a secondary decline

1–2 days later. Provided that mechanical coupling exists between extra- and intrafusal fibers, this may give support to the possibility of disturbances in the intrafusal fibers themselves (Komi & Nicol, 2000). Unfortunately, only a few SSC fatigue studies measured functional changes as well as indirect markers of the development of an inflammatory reaction. The results of Dousset et al. (2007) indicated an immediate postexercise increase only in interleukin 6 (IL-6) while prostaglandin E2 (PG E2) and leukocyte concentrations peaked at 2h postexercise (Figure 12.23a and b). Increases in C-reactive protein (CRP), substance P concentrations, and serum creatine kinase (S-CK) activity as well as muscle thickness were more delayed (Figure 12.23c and d). These changes occurred concomitantly with biphasic changes in MVC, voluntary activation, H/M ratio, and stretch-reflex EMG amplitude.

The validity of the inflammatory model after eccentric contractions has been challenged, however, as immunological studies have produced evidence for a remodeling theory without preceding muscle inflammation (Pedersen & Toft, 2000; Malm et al., 2000, 2004; Yu et al., 2002, 2003, 2004). This theory argues that exercise-induced disruption of the cytoskeleton is not a sign of damage, but rather remodeling of the myofibrillar structure. For instance, the frequently reported Z-band streaming (Fielding et al., 1993) is considered as a desmin resynthesis (Yu et al., 2002). In addition, Malm et al. (2000) reported that with respect to infiltrating neutrophils and macrophages, satellite cell activation and IL-1 β detection, eccentric cycling exercise, and multiple biopsies might cause similar changes in adult human skeletal muscle.

The comparison of these two theories is limited by the restricted number of human studies including direct muscle damage quantification, the variety of the damaging exercise protocols, and the lack of common methods for muscle damage quantification. Interestingly, however, recent emphasis has been put in the human literature to combine methods to improve quantitative and qualitative assessments of damage, inflammation, and/or remodeling at the ultrastructural level (Paulsen et al., 2007, 2009a,b,c; Lauritzen et al., 2009; Raastad

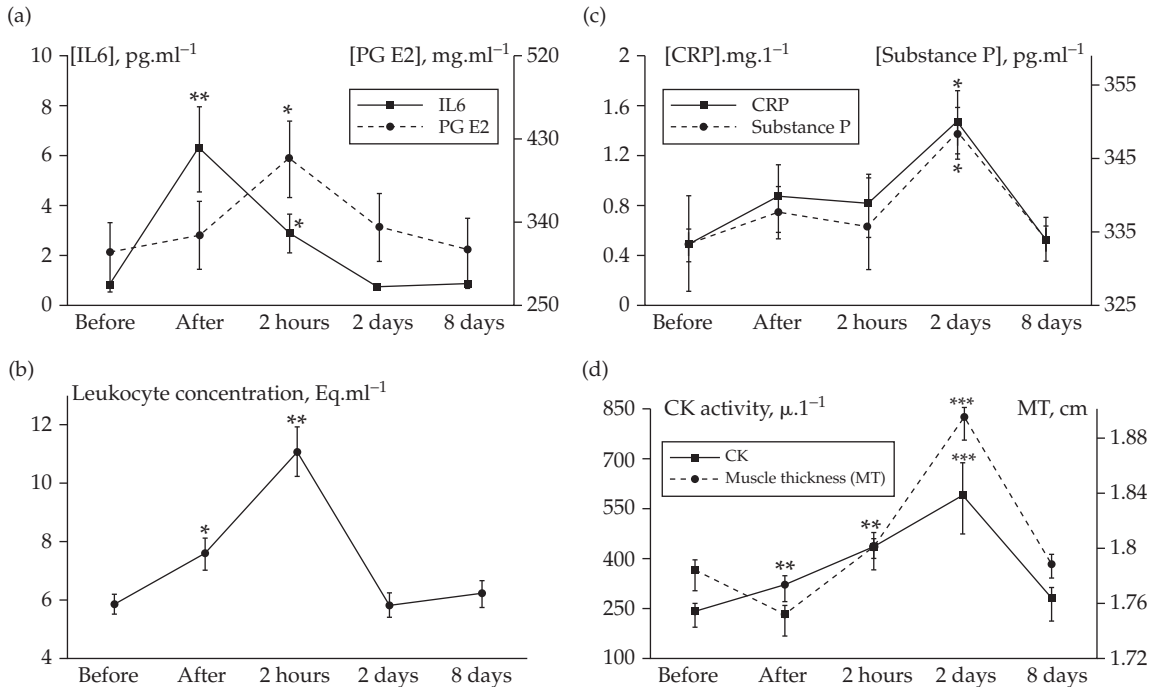


Figure 12.23 Changes in blood concentrations of indirect markers of muscle damage and inflammation before exhaustive SSC exercise and along 8 days of its recovery period. (a) IL-6 (solid line) and PG E2 (dashed line). (b) Leukocyte concentration. (c) CRP, substance P concentrations. (d) Serum creatine kinase activity (S-CK) and muscle thickness (MT). * $P < 0.05$; ** $P < 0.01$: Significant changes between pre-SSC exercise value and other values. (From Dousset et al., 2007.)

et al., 2010). These data bring additional insight into the discussion of both theories and the understanding of the associated DOMS and neuromuscular changes along the postexercise recovering period.

Potential Sources of Neural Changes

As suggested by the complexity of the SSC fatigue effects, the possible neural mechanisms involved in SSC-type fatiguing exercises are great in number. During the time course of fatiguing SSC exercise, a significant interaction has been demonstrated between prelanding activation and kinematics, and the resulting postlanding stiffness adjustment (see section on "Maximal and Submaximal SSC Testing Conditions"). These results emphasize the plasticity as well as the efficacy of neural adjustments during

the early parts of fatiguing exercise. They may also explain why, in some studies that used very moderate intensity exercise, the fatigue effects were minimal. After exhaustive SSC exercise, contribution of the central and reflex neural adjustments to the induced contractile failure has been clearly shown to vary depending on the testing task (see section on "Maximal Versus Submaximal SSC Dynamic Contractions"). The variation in neural adjustments has been classically addressed to several mechanisms: (1) to compensate for the contractile failure (Gollhofer et al., 1987b), (2) to optimize the neural drive in proportion to the contractile failure (also known as "muscle wisdom," Bigland-Ritchie et al., 1983a,b), and (3) to induce contractile failure with inadequate neural drive (Gandevia et al., 1995). It is not very well known why neural drive itself should be a limiting factor for the force output of the

muscle, but this has been repeatedly observed after intense or prolonged running and jumping exercises (see section on “Maximal Force Production and Muscle Activation”).

Specific Alteration in the Fusimotor-Muscle Spindle Function

As presented in “Stretch- and H-Reflex Responses” section, large acute and/or delayed reductions in stretch-reflex responses have been measured in both passive and active conditions after exhaustive SSC exercises. In case of muscle fatigue, various spinal and/or peripheral feedback mechanisms may occur (Gandevia, 2001). In general, these mechanisms can be related to either presynaptic or postsynaptic inhibitory effects coming from decreased net facilitation (disfacilitation) of the Ia-afferents originating from the muscle spindle or from different feedback sources. Some direct evidence comes from microneurographic recordings that reflex facilitatory influence provided by muscle afferents indeed declines as fatigue develops (Bongiovanni & Hagbarth, 1990).

Although the exact mechanism inducing the reduced Ia-afferent activity has not yet been thoroughly explained, three major possibilities have been presented: (1) withdrawal of the fusimotor support to the muscle spindles (Bongiovanni & Hagbarth, 1990; Hagbarth et al., 1986) and/or (2) intrafusal fiber fatigue itself (Emonet-Dénand & Laporte, 1974), and/or (3) mechanical unloading of the muscle spindles (Fowles et al., 2000; Avela et al., 2004). Methodologically, it is very difficult to separate the effect of fusimotor support to muscle spindles from the effects of muscle spindles themselves in fatigue. At present, however, it can only be hypothesized that both direct and indirect fatigue effects on the fusimotor-muscle spindle system exist.

Reflex Adjustments of the Neural Activation: Special Emphasis on Groups III and IV Afferents

It was originally suggested by Asmussen and Mazin (1978) and later supported by Bigland-Ritchie et al. (1986) that reduction in the neural activation could depend on some reflex response

from the contracting muscle itself. This hypothesis has been later challenged by Löscher et al. (1996) for sustained isometric contractions. The nature of SSC fatigue could, however, favor the intervention of such mechanisms. During the delayed phase of the recovery, structural and chemical changes associated with induced damage and inflammation should, in particular, have logical consequences on the afferent sensory pathways and, consequently, on the efferent activities.

The afferents most likely to change during and after fatiguing SSC exercise include muscle spindle afferents (groups Ia and II), GTO afferents (group Ib), groups II and III afferents (mechanically sensitive), and groups III and IV afferents (sensitive to non-noxious and noxious chemical and metabolic events). It is of interest for the present discussion on the functional effects of SSC-induced muscle damage that small-diameter, groups III and IV, muscle afferents could be likely candidates for such influences.

Can Sensitization of Small-Diameter Afferents Contribute to the Biphasic SSC Recovery?

These free nerve endings are particularly dense in the regions of connective tissues, but also between intra- and extrafusal muscle fibers as well as near blood vessels, in the GTOs and at the myotendinous junction (Stacey, 1969; Kaufman et al., 2002). They are known to be mostly polymodal (Kaufman & Rybicki, 1987), being sensitive to several parameters associated with either metabolic fatigue (Decherchi & Dousset, 2003), muscle injury (Kniffki et al., 1978; Rotto & Kaufmann, 1988) or with the inflammatory process (McMahon & Koltzenburg, 1990; Ge & Khalsa, 2003). In humans, the mean conduction velocity for groups III and IV fibers have been reported by Simone et al. (1994) to vary within the following ranges: (3.1–13.5 m/s) and (0.6–1.2 m/s). Consequently, these afferents carry messages in a continuous manner as long as the relevant stimuli exist.

Only part of these receptors is of nociceptive type (Mense & Meyer, 1985), but they are likely to contribute to the DOMS sensation. Most group IV afferents are nociceptors, whereas group III afferents also respond to non-noxious mechanical

stimuli, such as muscle contraction and stretch (Mense & Meyer, 1985; Kaufman & Rybicki, 1987). Group IV fibers have been suggested by Armstrong (1984) to be primarily responsible for the sensation of DOMS, but group III may be expected to be involved as well when the muscle becomes swollen during the delayed SSC recovery phase. Furthermore, once activated, the nociceptors also release neuropeptides, which cause vasodilation, edema, and release of histamine and substance P (Cuesta et al., 1999). These processes then lead to a further and long-lasting activation of some of the sensory endings (Jessel & Kelly, 1991; Millan, 1999).

Facilitatory and/or Inhibitory Influence?

The exact influence of small-diameter muscle afferents on the neural activation is not clearly established in fatigue conditions. In particular, the literature presents two major and contradictory trends in case of pain and damage.

As early as 1942, Travell and collaborators proposed the hypothesis further defined by Johansson and Sojka (1991) of a "vicious circle" in which groups III and IV muscle afferents are believed to activate gamma motor neurons and thereby to increase the background firing of the muscle spindles and/or their sensitivity to stretch. In the cat study of Jovanović et al. (1990), intra-arterial injection of algescic agents and lactic acid were found to induce increase of fusimotor discharge rate, which was mainly attributed to the activity of groups III and IV muscle afferents. In the case of SSC fatigue, this mechanism has been suggested to play a role in the large stretch-reflex facilitation observed in some of the "fast recovering subjects" (Nicol et al., 2003). At the end of fatiguing contractions, evidence also exists for a reflexively induced increase of the fusimotor discharge rate to the contracting muscle, its close synergists (Ljubisavljević et al., 1992, 1995), and to other muscles (for a review, see Ljubisavljević & Anastasijević, 1996). Additional complication comes from the pain studies conducted in humans by Matre and collaborators (1998, 1999), in which the infusion of hypertonic saline resulted in stretch-reflex facilitation at rest, but had no effect when the muscle was functionally active (during sitting and walking).

The hypothesis opposite to the "vicious circle" one refers to the sparing and protective effects of the fatigued muscle (Bigland-Ritchie et al., 1986; Garland & Kaufman, 1995; Jammes & Balzamo, 1992). Some convincing, although indirect evidence exists in favor of a presynaptic inhibitory effect on alpha motor neurons (Garland & McComas, 1990; Garland, 1991). For instance, Duchateau and Hainaut (1993) observed that the decrement in H/M reflex response did not recover as long as the fatigue-induced metabolic accumulation was maintained by ischemia. Avela et al. (2001) were able to show the same phenomenon after long-lasting SSC fatigue. Additional support to the involvement of groups III and IV muscle afferents comes from the finding that selective damage of these afferents (by application of capsaicin) abolishes the depression of the monosynaptic reflex induced by muscle fatigue (Brunetti et al., 2003).

These observations give support to the hypothesis of a potential functional effect of groups III and IV muscle afferents, especially during the inflammatory process that characterizes the delayed SSC recovery period. This is in line with the recent findings of Dousset et al. (2007) that the delayed changes in reflexes and isometric force occurred concomitantly with an increase in muscle thickness, CRP, and substance P concentration as well as S-CK activity. Thus, it is likely that PSI triggered by the groups III and IV muscle afferents may contribute to the biphasic pattern observed along the recovery period.

Influence of Sensory Input at Motor Cortex Level

As DOMS is characteristic of the SSC recovery, one would expect that pain research would bring additional information on the central influence of nociceptive groups III and IV muscle afferents.

Although pain influence has not been measured directly in connection with SSC fatigue, modifications in motor control strategies have been demonstrated using experimental pain protocols. With regard to the inflammation effects, Besson et al. (1975) demonstrated inhibitory influence of mesencephalic areas on neurons located in the lamina V from the dorsal horn when they were previously

sensitized to bradykinin. Andersen et al. (1995) reported that a central summation of nociceptive and non-nociceptive afferent activity can occur once secondary hyperalgesia is present. The introduction in man of a new method of evoking tonic pain discharge through intramuscular injection of hypertonic saline may bring new insights into the comprehension of the immediate and delayed pain effects (Rossi et al., 1998, 1999; Qerama et al., 2005). For instance, Le Pera et al. (2001) demonstrated that tonic muscle pain can induce an inhibition of the primary motor cortex as reflected by a reduction in motor-evoked potentials (MEPs) in the resting muscle. During the peak pain, the absence of effects on the H-reflex response suggested that the observed reduction in size of the MEPs was probably due to decreased excitability of the motor cortex. Twenty minutes after pain, the MEP amplitude was found to be further depressed and the H-reflex amplitude was also reduced suggesting an inhibition of the spinal motor neurons, possibly overlapping the cortical inhibitory processes. Furthermore, when the firing of small-diameter muscle afferents is maintained by muscle ischemia at the end of a fatiguing contraction, force and voluntary activation remain reduced, although MEP and the silent period (SP) recover (Gandevia et al., 1996) (Figure 12.24). The dissociation of impaired voluntary activation from changes in the responses of the motor cortex as long as the muscles were held ischemic suggests that small-diameter muscle afferents may play a role in supraspinal fatigue.

Furthermore, and confirming animal studies, Martin et al. (2006) provided evidence that motor neurons of extensor and flexor muscles of the human elbow are not uniformly affected by inputs from groups III and IV muscle afferents. As shown in Figure 12.25, afferent inputs from homonymous and antagonist muscles inhibit extensor motor neurons, whereas motor neurons innervating flexors are facilitated. This suggests that extensor muscles might require greater cortical output to generate a given force during fatigue (Martin et al., 2006). Thus, small-diameter muscle afferents may

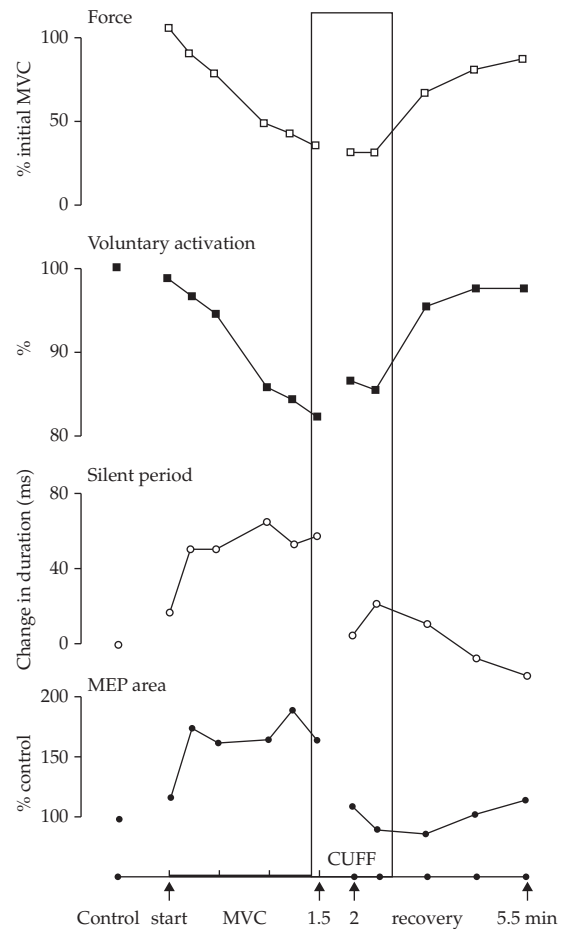


Figure 12.24 Effect of a maintained ischemia after sustained MVC on maximal voluntary contraction, voluntary activation, and MEP characteristics (MEP area and SP). The upper two panels show maximal voluntary contraction and voluntary activation (estimated by the force increment elicited by TMS). Both of them fell during the sustained MVC, did not recover during the cuff-induced ischemic period, but recovered when blood flow resumed. The bottom two panels show changes in the TMS-elicited EMG responses. Both the SP duration and the MEP area increased during the sustained MVC. Unlike voluntary force and activation, they recovered despite muscle ischemia. MVC, maximal voluntary isometric contraction; MEP, motor-evoked potential; SP, silent period; TMS, transcranial magnetic stimulation; EMG, electromyography; CUFF. (Adapted from Taylor & Gandevia, 2001.)

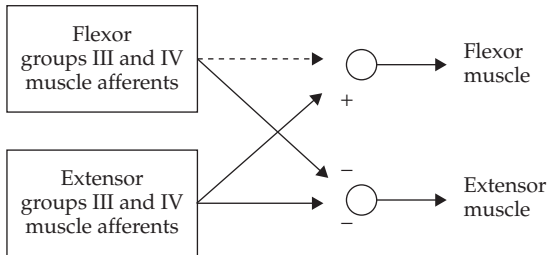


Figure 12.25 Schematic diagram showing apparent effects of firing of groups III and IV muscle afferents on the flexor and extensor motor neuron pools. The figure is based on findings that indicate facilitation of flexor afferents and inhibition of extensors by flexor or extensor afferents. A previous study (Butler et al., 2003) showed nonsignificant facilitation of the flexors by flexors afferents (dashed line). (From Martin et al., 2006.)

contribute to the inhibition of motor neurons in some muscles, but not in others.

Based on these overall observations, it is suggested that groups III and IV muscle afferents are likely to contribute to the biphasic pattern observed along the SSC recovery period. On the other hand, both animal and human studies show mixed effects of groups III and IV muscle afferents on motor neurons. It is therefore possible that the spinal and supraspinal effects of the sensitization of small muscle afferents could vary depending on the level of the imposed musculotendinous fatigue. Variation in receptor types and in the ability to modulate pain at multiple levels in the nervous system could explain part of the intersubject variability in soreness perception.

Conclusions

This chapter made an attempt to demonstrate that naturally occurring, but exhaustive, SSC exercise induces often dramatic reduction in force and power production. The complex features of SSC loading is represented as the biphasic nature of performance deterioration and recovery.

When sufficiently exhaustive, SSC exercises may lead to reversible muscle damage, with subsequent inflammation/remodeling processes associated with DOMS. These events have considerable influence on neuromuscular function including indices such as muscle structure, muscle mechanics, and joint and muscle stiffness. In connection with these variables, the neural modifications are of considerable mechanistic interest. This is demonstrated by the parallelism between the neural and mechanical changes during the SSC exercise and along its subsequent recovery.

In the acute recovery phase, the monosynaptic stretch reflex is one of the most dramatically reduced parameters immediately after SSC exercise. The immediate reduction is thought to result from the short-term effects of metabolic fatigue and from the decrease in muscle-tendon compliance. The delayed recovery is more complex in nature. It is suggested that the delayed recovery results from the influences of structural damage/remodeling of the muscle-TTs. Structural and functional recovery is long lasting, and prevents an individual from performing normal exercise routines for several days. In some severe cases, the recovery period may exceed 10 days. After fatiguing SSC exercise, inadequate neural drive may be considered as an attempt of the neuromuscular system to protect the MTU from additional damage. Many studies suggest influence of exhaustive SSC fatigue on the fusimotor-muscle spindle function as well as at the supraspinal level. Activation of small (III and IV) afferents is proposed as an attractive factor to cause PSI with subsequent reduction in the stretch-reflex response, but also to result in inhibition and/or facilitation at the supraspinal level. The functional neural consequences and their coupling with muscle damage/remodeling induced by SSC exercise are illustrated in Figure 12.26.

Finally, based on the reported SSC fatigue studies using maximal isometric and/or SSC testing tasks, it is emphasized that the SSC type may allow distinct examination of the central and

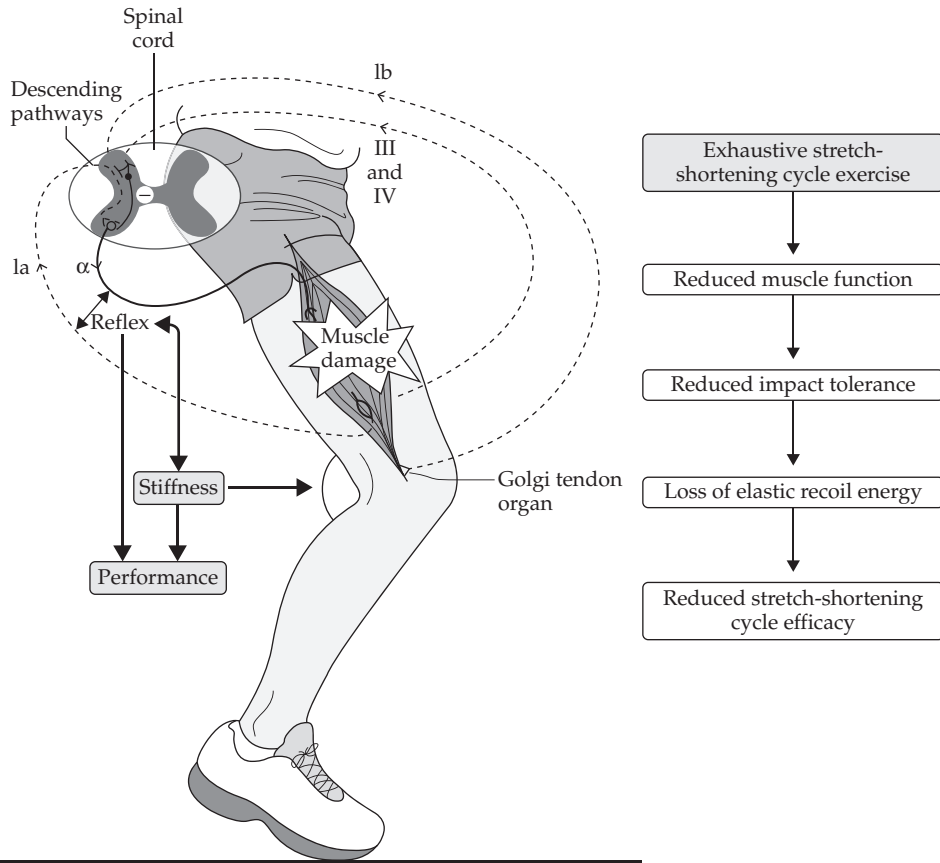


Figure 12.26 Schematic presentation of the possible interaction between neural pathways and the events of mechanical failure during the delayed recovery phase from exhaustive SSC exercise. PSI pathway is not shown in this graph. When the muscle fatigues, it is characterized by reduced tolerance to repeated stretch loads, by a deterioration of elastic recoil and increased work during the push-off phase, so that the same functional outcome can be maintained. SSC, stretch-shortening cycle. (Modified from Taylor et al., 2008.)

stretch-reflex EMG changes with fatigue. For instance, the maximal SSC tests confirmed the functional role of the stretch-reflex EMG response (discussed in Chapter 1) as their reduction with fatigue was found to result in large decrease in SSC performance. Even more meaningful are the submaximal SSC testing tasks as they may reveal a group

of changes (adjustments) of the central and reflex EMG components along the recovering period. As the SSC tasks involve several joints, they also favor the examination of intermuscular compensations. These remarks apply particularly to SSC exercises performed on the sledge exercise as they are characterized by a high reproducibility.

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Chapter 13

Training Adaptation of the Neuromuscular System

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Introduction

Although almost all sport disciplines involve the muscle to be successively stretched and shortened within a very brief period of time, only few studies have directly investigated the training effect on the “SSC” and the associated mechanisms. As a consequence, most of this chapter will be dedicated to the adaptations that occur within the nervous and muscular systems that underlie the improvement of strength and power in response to classical training programs. Such a review is nevertheless relevant as it provides the methods, and the associated adaptations, to develop strength and power from which the performance of SSC exercises can be maximized. We will describe successively: (1) the basic theories of strength and power training; (2) motor units characteristics and muscle activation level; (3) the main mechanisms for neural and (4) muscular adaptations.

Basic Theories of Strength and Power Training

Definitions

Before discussing the principles of strength and power training, it is necessary to define basic

notions commonly used in the field. Furthermore, to avoid confusion among nonspecialists when we report experimental data from human in natural conditions and animal preparations, we will use the term “force” throughout this chapter. As in human in vivo conditions, force is developed through a lever arm, the appropriated term would be “torque.”

Muscle Strength

Muscle strength is the maximal amount of force a muscle or a muscle group can generate in a specified muscle contraction or movement. It is commonly measured in three different ways: (1) as the maximal force that can be produced during an isometric (static) contraction; (2) the peak force produced during isokinetic shortening (concentric) or lengthening (eccentric) contractions; or (3) the maximal load that can be lifted once. The isometric contraction is classically referred to as an MVC. For isokinetic actions, force is either reported as the maximal (peak) or the average force exerted during the movement and can be expressed as percentage of the maximal force recorded at slow velocity. The load that can be lifted once is known as the 1-repetition maximum (1-RM).

Muscle Power

Muscle power is equal to the product of the load (or force) and the velocity of movement. This product induces that, for a specific movement, muscle

power can be increased either by performing the movement in a briefer period of time or by lifting a heavier load without extending the duration of the movement. Consequently, both the increase in maximal force or speed of shortening may augment the maximal power produced by a muscle. Peak or average power can be quantified by measuring the speed of a movement performed against a constant load or to record the force produced during a movement performed at constant velocity (isokinetic action). For a single muscle electrically stimulated, peak power is classically obtained for a load corresponding to 1/3 of the maximal force or when the muscle shortens at 1/3 of the maximal shortening velocity (Hill, 1938). For voluntary contractions, however, peak power is usually reached at higher values (~40–50% of maximum force or velocity; see Kaneko et al., 1983; Baudry & Duchateau, 2007).

Rate of Force Development

In many sport disciplines, a great amount of force must be produced rapidly, and only a brief period of time (~100–200ms; Zatsiorsky, 2003) is available to develop the greatest possible force (slope of the F–T curve); this refers to as the RFD (Figure 13.1). Because longer time (>300ms) is usually necessary to attain the maximal force, a higher RFD would permit to produce a greater percentage of the maximal muscle force (Figure 13.1). Although the notion of RFD is closed to those of muscle power, it differs in that the RFD relies on the time course of the force production rather than on the effect of the velocity of muscle shortening on the load displacement. Such distinction is highly relevant as the RFD has been reported to be a better determinant of the performance than the maximal force or power (Komi, 1986; Aagaard et al., 2002). This leads some scientists to measure preferentially the maximal RFD (Duchateau & Hainaut, 1984; Van Cutsem et al., 1998) or the RFD at different time points of the F–T curve of a rapid isometric contraction (Häkkinen et al., 1985; Aagaard et al., 2002; de Ruyter et al., 2004; Gruber & Gollhofer, 2004) as an index of change in the muscle contractile properties associated with a training program.

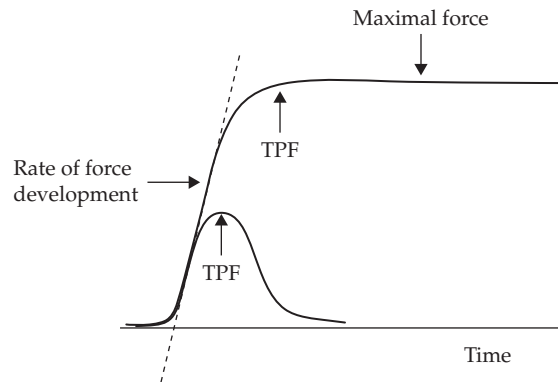


Figure 13.1 Comparison of F–T curves during a contraction aimed to reach the maximal voluntary force of the individual and a contraction during which the individual intended to develop his force as fast as possible (ballistic contraction). The dashed line represents the rate at which the force is developed. Although the two contractions exhibit a similar RFD, the briefer time-to-peak force for the ballistic contraction does not allow to exert the maximal force of the individual, as observed in many sport disciplines. Specific training programs have been designed to increase the RFD and as a consequence the peak force that an individual can reach during ballistic contraction (see text). TPF, time-to-peak force.

Overload Principle

Progressive resistance exercise or progressive overload refers to the practice of increasing the stress placed on the muscle as it becomes more capable of producing greater force or power (Hellebrandt & Houtz, 1956). The muscle can be progressively overloaded by increasing the loads that are used or by increasing the total training volume (number of sets and/or repetitions of a particular exercise). A reasonable guideline is to increase the resistance for a particular number of repetitions or training volume by no more than 2.5–5% at one time (Fleck & Kraemer, 2004). Although the recovery between sets of an exercise is largely determined by the objectives of the training program, the reduction of the time between two sets can also contribute to increase the stress imposed to the muscle. However, when the goal is to increase power or maximal strength with very high loads (>90% of 1-RM), a longer recovery period is needed to maintain a high quality of movement (rate and intensity of activation).

Main Parameters for Strength and Power Training

As already mentioned, the increase in muscle strength is based on the overload principle (Hellebrandt & Houtz, 1956), and many methods have been developed for this purpose. Because the description of the training methods is beyond the scope of this chapter, the reader is invited to consult more specialized literature (Schmidtbleicher, 1992; Fleck & Kraemer, 2004; Bompa & Carrera, 2005). Nevertheless, we will briefly evoke the essential parameters that determine the characteristics of the load during a training program: (1) the magnitude of the load; (2) the number of repetitions and sets of a given exercise (training volume); (3) the type of contraction and loading technique; (4) and the RFD or speed of movement.

Magnitude of the Load

Except for untrained individuals who can already experience strength adaptation with load below 60% of their maximum force, the optimal loading of a muscle to increase its force ranges between 60% and 100% of MVC or 1-RM (McDonagh & Davies, 1984). This is commonly defined as the strength-training zone (Figure 13.2; Sale & MacDougall, 1987). Because the maximal number of repetitions that can be completed at a given load declines with the increase of the load, the number of repetitions within each set will therefore be related to the magnitude of the load. Depending of the muscle group and training status of the individual, the average number of repetitions that can be performed is: 12–15 at 60%, 10–12 at 70%, 6–8 at 80%, and 3–4 at 90% of 1-RM (Sale & MacDougall, 1987). Well-trained athletes commonly use a narrow range of loads (1–10 RM), and for some of them (weightlifters; throwers and jumpers in athletics) the focus is on heavy load (1–6 RM). The use of such heavy load is also known to improve the performance of SSC exercises performed under high-impact loadings such as in jumping.

Training Volume

Moderate-volume training with various combinations of repetitions and sets produces greater

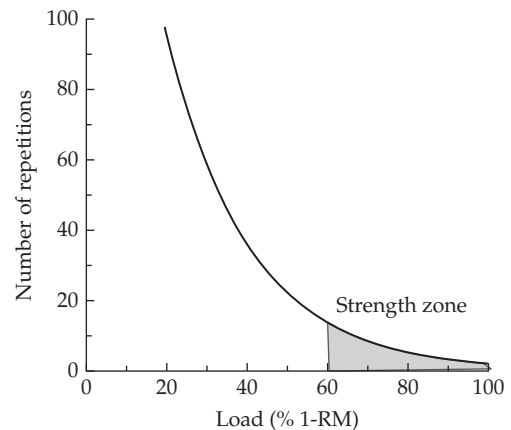


Figure 13.2 Relation between the load and the number of repetitions that can be performed. Load is expressed as percentage of the heaviest load that can be lifted for one repetition (1-RM). The strength training zone indicates the range of loads (60–100% of 1-RM) that favors the increase in maximal force. (Redrawn from Sale & MacDougall, 1987.)

improvements in strength activities compared with low- and high-volume training (Fleck & Kraemer, 2004; Gonzalez-Badillo et al., 2005). For trained athlete, it is usually accepted that a total of 30–50 repetitions for a given exercise is necessary to increase substantially the strength of a muscle group. When the strength-training program focuses on muscle hypertrophy, a greater number of repetitions and sets should be performed (80–100 repetitions per exercise; Tesch, 1992; Schmidtbleicher, 1992). Depending on the training method and the objective, a rest period of 2–5 min between sets is commonly used. As strength gain depends of the chronic stress of the motor system, several training sessions per week has to be performed. The frequently recommended “dose” is 2–3 days per week for novice and average level athletes and 4–5 days per week for elite athletes (Kraemer & Ratamess, 2004).

Type of Contraction and Loading Technique

During a contraction, muscle may shorten (concentric contraction), lengthen (eccentric contraction), or keep the same length (isometric contraction).

Except for isometric exercises, strength training involves length change of an active muscle over a defined range of motion. Because the force that a muscle can produce is varying with joint angle (either shortening or lengthening), it is necessary to determine the range of motion and the type of contraction required by the sport discipline. On the basis of the F–V relation determined either on single muscle fiber or on whole muscle in animal preparation, the maximal force produced during eccentric contractions is greater than that produced during isometric and concentric contraction (Katz, 1939; Edman, 1988). Despite similar observations *in vivo* in human muscles (Komi & Buskirk, 1972; Aagaard et al., 2000; Klass et al., 2005), the force produced during eccentric contractions does not always differ significantly from isometric force (Westing et al., 1990; Amiridis et al., 1996; Colson et al., 1999), due presumably to a submaximal neural activation (see later). The greater force developed during eccentric contraction compared with concentric contraction should be taken into account when choosing the type of contraction as it means that a same absolute load imposes a greater relative load (% of maximum force) to the muscle group during the raising than the lowering phase of the movement.

Another classical distinction is whether the load applied to the limb remains constant or varies over the range of motion. This is referred to as constant load and variable load training, respectively. Movements with free weights (i.e., barbells and dumbbells) correspond to the so-called “constant loads” whereas machines with cam provide loads that vary over a range of motion. The advantage of such machines is to produce a variable load that matches the strength capability of the individual throughout a prescribed range of motion (Enoka, 2008). Similarly, exercise machines in which load is controlled by gear or friction systems, hydraulic or pneumatic systems provide an accommodating resistance that allows a nearly constant (isokinetic movement) velocity of the displaced body segment. The advantage of movement performed at constant velocity is that voluntary activation can be maintained at a high level throughout the whole range of motion which is not the case with a

constant load. Although these machines are useful for strength gain, their major limitation, however, is that neither they do not mimic natural movements nor allow the acceleration of the load during the movement similarly to what occurs in many sport disciplines (Iossifidou et al., 2005).

RFD and Speed of Movement

As already mentioned, in explosive sports, the ability to rapidly develop force is more important than the maximal force itself. It is known for many years that to increase the RFD or power of a muscle group, a high rate and intensity of activation is needed. Depending on the requirements of a sport discipline, small or medium to heavy load can be used (Duchateau & Hainaut, 1984; Häkkinen et al., 1985; Schmidtbleicher, 1992; Van Cutsem et al., 1998; Aagaard et al., 2002; Beck et al., 2007). It has been shown by Behm and Sale (1993) that isometric and concentric isokinetic training induced similar adaptations. This means that the intention to contract quickly a muscle group may produce similar changes in the RFD than the actual speed of movement. The exercises are usually performed either from a resting condition, a sustained submaximal contraction, preceded by a countermovement or a rapid prestretch (SSC; Figure 13.3). In the latter case, the exercise is called “plyometrics.” This type of exercise does not intend to increase the maximal strength but is known as effective at increasing muscle power and the RFD (Kyröläinen et al., 2005; Kubo et al., 2007). Another advantage of plyometric exercises is that they enable an individual to train more specifically the “stretch-shortening” component of a movement, which is difficult to accomplish with any other technique. It is recommended, however, to limit the number of repetitions within a set and to give sufficient rest between two sets to avoid fatigue and keep the “explosive” aspect of the movement.

Specificity of Training Effects

A widely accepted principle in strength-training theory is the specificity of the adaptations. This means that the effects of training are observable mainly in the practiced exercise. As a consequence,

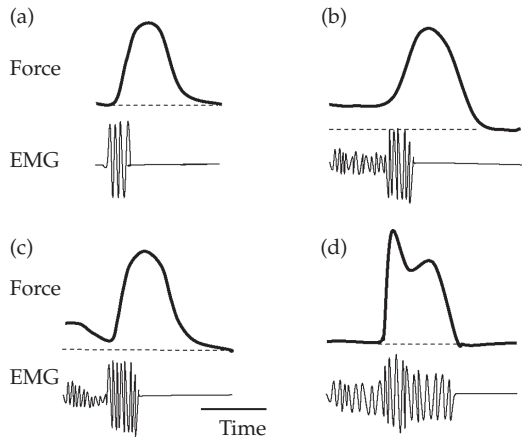


Figure 13.3 Schematic representation of F–T curves of ballistic contractions performed from different initial conditions. The four panels illustrate ballistic contractions performed from: (a) a resting state, (b) a sustained submaximal contraction, (c) a countermovement, and (d) a “plyometric” contraction (DJ). Note that the pattern of electromyographic (EMG) activity and the RFD vary during these different conditions with a faster RFD exhibited for the plyometric condition.

the training exercises should simulate the sport task parameters as closely as possible to induce adaptations that could be transferred to the sport movement. The main parameters that need to be considered are: (1) the movement pattern and position, (2) the contraction type, and (3) the magnitude of the load and the speed of contraction or movement (Sale & MacDougall, 1987; Figure 13.4).

Movement Pattern and Position

Although strength training increases the maximal strength of an individual, the maximal force that can be exerted varies across tasks (Rutherford & Jones, 1986; Aagaard et al., 1996; Wilson et al., 1996). There are many examples showing that the improvement in strength is greatest when training and testing modalities are the same. For example, 8 weeks of training that consisted in barbell-squat exercise produced a larger increase when the 1-RM was tested in the squat exercise than when tested in a nonspecific test such as a leg press (Thorstensson et al., 1976). Similarly, a training program that involves simultaneous extension of the two knees

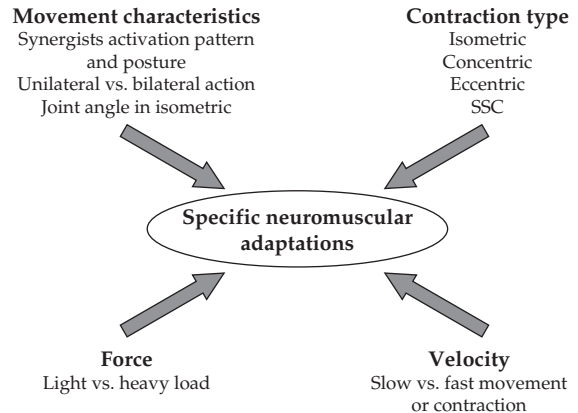


Figure 13.4 Schematic presentation of the different parameters that can influence the specificity of the neuromuscular adaptations with strength and power training.

(bilateral movement) improves the strength of knee extensor muscles to a greater extent during bilateral extension than when the movement is performed by one limb alone (unilateral movement; Coyle et al., 1981; Enoka, 1988). It is also well known that training with maximal isometric contraction at a given joint angle increases the maximal strength substantially for the trained angle but much less for adjacent joint angles (Thépaut-Mathieu et al., 1988; Kitai & Sale, 1989). It is interesting to note that the specificity of strength gains is most pronounced for complex tasks, in either less constrained movements (Thorstensson et al., 1976; Chilibeck et al., 1998; Rutherford & Jones, 1986; Wilson et al., 1996) or involving eccentric contractions (Higbie et al., 1996; Hortobagyi et al., 1996; Laidlaw et al., 1999; Aagaard et al., 2000). Moreover, multijoints exercises seem to induce greater transfer to the sport specificity than single-joint exercises (Enoka, 2008).

Contraction Type

When an individual trains with one type of contraction and the progress is evaluated with the same type of muscle action, a substantial increase in strength maybe apparent. However, if the same individual's progress is determined using another modality of contraction, little or no increase in

strength maybe demonstrated. This testing specificity indicates that gains in strength are specific to the type of muscle action used during training. This effect is often demonstrated by comparing training-induced gains in peak force measured either by an MVC (isometric force) or a 1-RM test (dynamic force). For example, a 12-week training program consisting in leg extension against a load of approximately 80% of 1-RM (concentric contractions), increased the 1-RM load to a greater extent (200% and 240% for men and women, respectively) than the increases in isometric MVC force (20% and 4%) (Rutherford & Jones, 1986). Similarly, training with concentric isokinetic actions of the leg extensor muscles at 100°/s induced a greater peak force gain when tested during a concentric than an eccentric contraction (16.7% vs. 8%; Tomberlin et al., 1991). The same contraction-type specificity was true when training involved eccentric contractions (50.8% and 28.7% when tested by eccentric and concentric contractions, respectively). Nevertheless, a lower degree of specificity after training performed with concentric contractions has been reported by Hortobagyi et al. (1996). In this study, the subjects who trained the knee extensor muscles with eccentric contractions experienced a much greater gain in force during similar contraction type (116%) compared with isometric (48%) and concentric (29%) contractions. In contrast, subjects who trained with concentric contractions displayed a relatively similar strength gain in this type of contraction (53%), than in isometric (34%) and eccentric (46%) contractions. Despite a lack of specific adaptation with concentric contraction in this particular study, it is usually recommended for a training program of a given sport to include the specific types of muscle actions encountered in this sport (isometric, concentric, and eccentric contractions or stretch-shortening actions).

Load and Speed

Training with heavy load (>60% of maximum) increases mainly the maximal force and power with limited change in maximal velocity of movement or RFD (Duchateau & Hainaut, 1984; Häkkinen et al., 1985), although the study by Aagaard and

colleagues (2002) reported an increase in the RFD after training with load greater than 70% of 1-RM. In contrast, training using exercise with light load (30–40% MVC) but performed with maximal velocity (e.g., ballistic actions) increases mainly the maximal RFD with limited increase in maximal force (Duchateau & Hainaut, 1984; Häkkinen et al., 1985; Van Cutsem et al., 1998). Furthermore, specific adaptations of the load–velocity relation were found following training with different loads in elbow flexion actions (Kaneko et al., 1983). Light loads which are moved with high velocity ($\leq 30\%$ of 1-RM) are more effective to increase the maximal velocity of shortening compared with heavier loads ($\geq 60\%$ of 1-RM). In contrast, heavy loads are associated with a lower speed and augment predominantly the velocity of shortening for mechanical resistance close to the muscle maximum force. Similarly, several studies have found velocity-related specificity in the gain of force when training was performed at constant speed (Coyle et al., 1981; Caiozzo et al., 1981; Kanehisa & Miyashita, 1983). Training at low velocity caused a substantial increase in strength developed at low velocity but had little effect on the strength produced during high velocity movement. In contrast, training at high velocity induced greater increase in strength at high than at low velocity. Finally, training with mixed (high and low velocities; Coyle et al., 1981) or at intermediate (Kanehisa & Miyashita, 1983) velocity produced intermediate results. This knowledge of speed-related adaptations has important practical implications when designing resistance training programs for specific sports.

In conclusion, the amount of carryover from a strength-training program to a specific sport performance depends on the transfer specificity between the training exercise and the sport skills. For beginners, however, training does not have to be as specific as for elite athletes, at least in the first few months of training. Although the increase in strength and power results mainly in changes within the muscular system, the specificity of adaptations to strength training also includes changes that occur primarily in the nervous system, such as those associated with learning and improvements in coordination (see later). Such adaptations

are particularly clear in SSC type exercises after a program of plyometric training (Schmidtbleicher & Gollhofer, 1982; Voigt et al., 1998).

Motor Units Characteristics and Muscle Activation Level

In human experiments, the strength of a muscle is usually estimated as the peak force achieved during an MVC or by a 1-RM exercise. Changes in maximal strength can be attributed to adaptations in the force capacity of the muscle fibers and the activation characteristics of the neurons that supply muscle fibers. In this section, we will evoke briefly the main characteristics of the activation of motor unit that consists of a neuron and the muscle fibers it innervates, and their specific behavior during different types of contraction.

Motor Unit Recruitment and Rate Coding

As defined by Sherrington (1925), the motor unit is the common final pathway of the motor system that comprises a motor neuron in the ventral horn of the spinal cord, its axon, and the muscle fibers that the axon innervates. Although the distribution of synaptic inputs can influence the order in which motor units are recruited, the most important determinant is the size of the motor neuron. The association between the size of a motor neuron and the order at which it is activated is known as the “size principle” (Henneman, 1957). The influence of size on recruitment order is attributable to its effect on input resistance. According to Ohm’s law, the change in membrane potential in response to a synaptic current is proportional to the input resistance of the motor neuron. Because small motor neurons have a high input resistance, they are the first to be recruited in response to an increase in depolarizing synaptic currents. As a consequence, smaller motor units are activated before larger units. Due to the relation between the size of the motor neuron and the properties of the muscle fibers it innervates, this recruitment sequence results in slow-contracting and fatigue-resistant motor units being recruited before fast-contracting and fatigable motor units. Although there is some

variability in the recruitment order of motor units with similar thresholds (Thomas et al., 1986; Feiereisen et al., 1997), the recruitment order of motor units is essentially the same for isometric and dynamic contractions, including shortening and lengthening contractions (Duchateau & Enoka, 2008), and during rapid (ballistic) isometric (Desmedt & Godaux, 1977, 1978) and shortening (Ivanova et al., 1997) contractions.

The force that a muscle exerts depends on the amount of motor unit activity, changing with the number of motor units that are active (motor unit recruitment) and the rates at which motor neurons discharge action potentials (rate coding). The relative contributions of recruitment and rate coding to the force exerted by a muscle vary with the level of muscle force and the muscle performing the contraction. In most muscles, the upper limit of motor unit recruitment is about 80–90% of the maximal force (Kukulka & Clamann, 1981; De Luca et al., 1982; Van Cutsem et al., 1998). In some hand muscles, however, the upper limit of motor unit recruitment is 50–60% of maximum (Milner-Brown et al., 1973; De Luca et al., 1982; Duchateau & Hainaut, 1990; Moritz et al., 2005). However, one important feature of motor unit recruitment is that the absolute force level at which it is recruited is not fixed and varies with the speed and type of muscle contraction. For example, the recruitment threshold of motor units in the TA decreases progressively with the increase in the RFD (Figure 13.5a; Desmedt & Godaux, 1977). As a consequence of this adjustment, motor units are activated earlier during rapid contractions, and approximately 3 times as many motor units are recruited to produce a given peak force during a rapid contraction compared with a slow contraction (Desmedt & Godaux, 1977). Due to this effect, most motor units are likely to be recruited with a load equivalent to 1/3 of maximum when performing a rapid contraction. The extent of the reduction in recruitment threshold of motor unit, however, is greater for slow-contracting muscles (e.g., SOL) compared with fast-contracting muscles (e.g., masseter) (Desmedt & Godaux, 1978). The greater reduction in recruitment thresholds for slow muscle likely facilitates their ability to perform fast contractions. Furthermore, recruitment

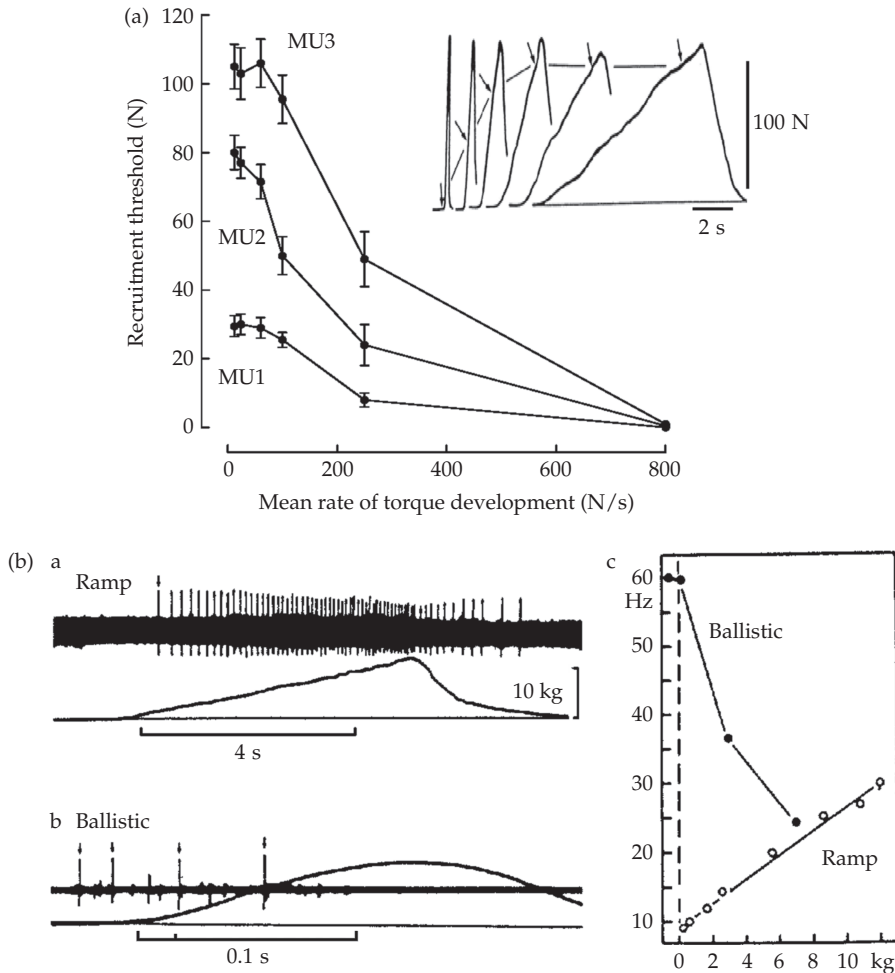


Figure 13.5 Changes in motor unit recruitment threshold and discharge frequency with increased rate (speed) of torque (force) development. (a) Illustrates the reduction in recruitment threshold (mean \pm SD for ten trials) for three motor units in the TA muscle with an increase in the rate of torque development during ballistic isometric contractions performed with the ankle dorsiflexor muscles. The inset shows the six different rates of increase in torque to the target force of 120 N ($\sim 50\%$ MVC), with the most rapid contraction (0.15 s to peak force) at the left-hand side. Note the decrease in recruitment threshold for all units, but without change in recruitment order predicted by the size principle. The thresholds become close to zero for the most rapid contraction. (b) Displays the discharge frequency of a motor unit during a slow increase in force (ramp; a) and during a contraction of similar force but performed as fast as possible (ballistic; b). The change in motor unit discharge rate during the two contractions is illustrated in c. Note the opposite change in instantaneous discharge rate during the two contraction types and the much larger frequency (60 vs. 30 Hz) attained during the ballistic contraction. MU, motor unit. (Adapted from Desmedt & Godaux, 1977.)

thresholds can be lower during dynamic contractions compared with isometric contractions (Tax et al., 1989), and at short muscle lengths compared with long lengths during isometric contractions (Pasquet et al., 2005). As a consequence of the

upper limit in the recruitment threshold of motor units, the increase in muscle force beyond this limit is accomplished entirely by rate coding.

Although the discharge rate of a motor neuron increases linearly with the depolarizing current it

receives (Kernell, 1965), there is a sigmoidal relation between discharge rate and muscle force (Monster & Chan, 1977; Erim et al., 1996; Macefield et al., 1996; Moritz et al., 2005). The minimal rate at which most motor neurons discharge action potentials repetitively during voluntary contractions is 5–8 Hz (Sogaard et al., 1996; Van Cutsem et al., 1998), but the maximal discharge rates vary across muscles. Average rates of 30–50 Hz have been recorded for most muscles during isometric contractions (for a review, see Enoka & Fuglevand, 2001), whereas rates of approximately 10 Hz have been recorded for a slow-contracting muscle as the SOL (Bellemare et al., 1983). Instantaneous discharge frequencies at the initiation of rapid contractions, however, can reach values of 60–120 Hz in untrained subjects (Figure 13.5b; Desmedt & Godaux, 1977; Van Cutsem et al., 1998; Van Cutsem & Duchateau, 2005).

In animal studies, the maximal discharge rate usually matches the fiber-type composition of the muscle with muscles that contain a high percentage of slow fibers displaying lower maximal rates (Bakels & Kernell, 1993). In humans, the general slope of the relation between discharge rate and muscle force has been reported to vary as a function of recruitment threshold of the motor unit in some studies (Freund et al., 1975; Monster & Chan, 1977), but not in others (Milner-Brown et al., 1973). There is also no consensus on the relation between motor unit recruitment threshold and its maximal discharge rate. Some studies found a negative correlation between peak discharge rate and recruitment threshold during ramp isometric contractions (De Luca et al., 1982; Duchateau & Hainaut, 1990), whereas a recent study reported that peak discharge rate increased with recruitment threshold when subjects performed discrete isometric contractions at various target forces (Moritz et al., 2005). In addition to technical problem in the recording of discharge rate at maximal force, the discrepancy between studies may reflect the inability of some individuals to drive high-threshold motor units to their maximal discharge capacity.

Maximal Capacity for Muscle Activation

As evoked in the previous section, the maximal force or RFD of a muscle is achieved when the

whole population of motor units is recruited and activated at maximal frequency. However, full activation during an MVC or at the onset of a fast voluntary contraction may not be reached in untrained individuals, leading to a suboptimal activation of their muscles.

Maximal Force

A common approach used to assess whether a muscle can be fully activated by the descending drive during a maximal contraction is the “interpolated twitch method” (Merton, 1954; Gandevia, 2001). Briefly, the method consists in superimposing to an MVC a single (Merton, 1954; Gandevia, 2001; Taylor, 2009), doublet (Behm et al., 2002; Klass et al., 2005) or short train of supramaximal electrical stimuli (Strojnik, 1995; Kent-Braun & Le Blanc, 1996). It is assumed that the superimposed stimulation will recruit muscle fibers that are not activated by the voluntary effort and thereby will produce an extra force that is superimposed on the ongoing voluntary force (Figure 13.6a). Although the technique presents some limitations as a method to quantify voluntary activation of muscles in humans (see de Haan et al., 2009), it is the only method currently available to estimate the extent of voluntary activation. With this technique, it has been shown that many untrained individuals exhibit submaximal activation during an MVC (Figure 13.6b), at least when the superimposed stimulation involves a brief train of stimuli (Kent-Braun & Le Blanc, 1996; Behm et al., 2002; Stevens et al., 2003). Interestingly, muscle activation appears to be markedly less than maximal during eccentric contractions (Figure 13.6c; Westing et al., 1990; Amiridis et al., 1996; Pinniger et al., 2000). It is not clear whether submaximal activation is due to an incomplete recruitment, submaximal discharge rate of individual motor units, or both mechanisms.

Maximal RFD

Whereas it has been observed in different muscles that motor unit discharge rate during ballistic contractions reached maximal values of approximately 120 Hz in untrained subjects (Desmedt & Godaux, 1977), the maximum RFD during tetanic contractions is reached at an electrical stimulation of

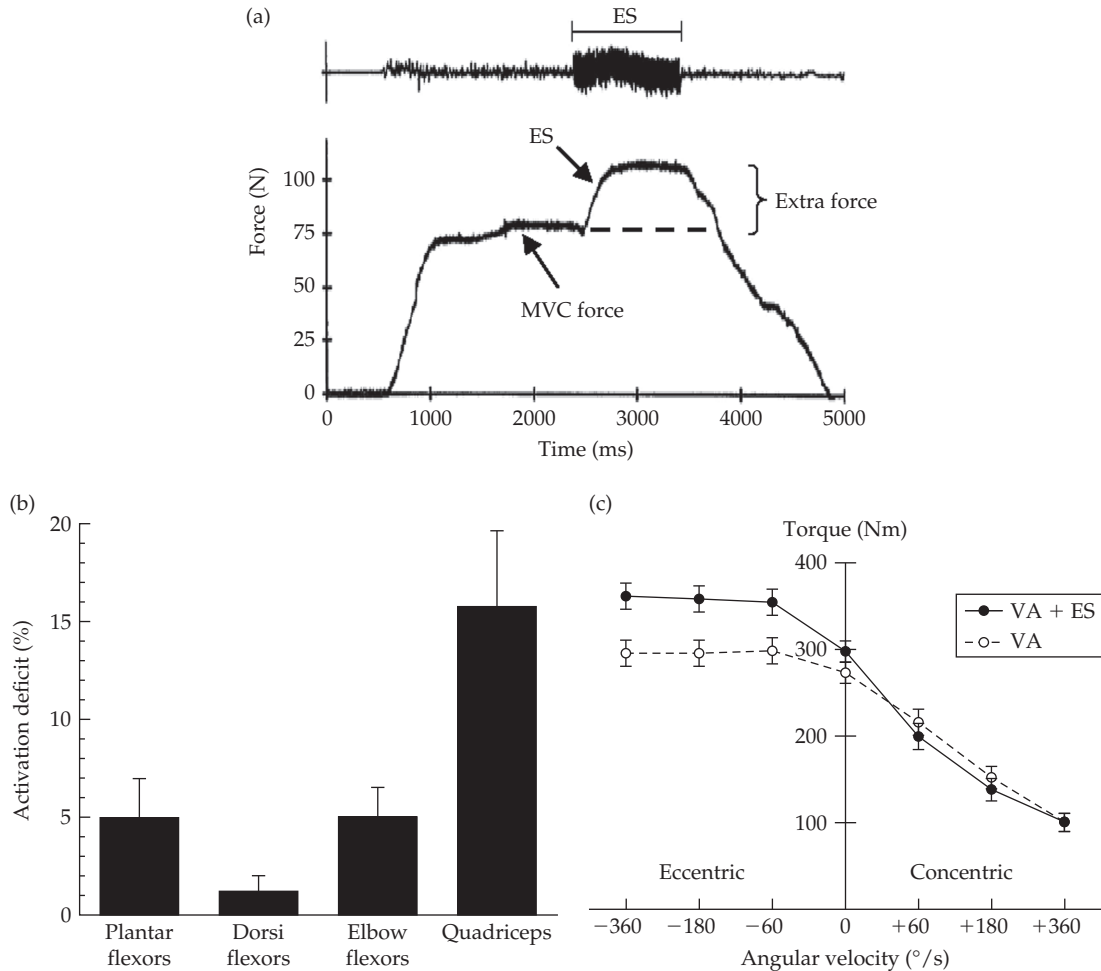


Figure 13.6 Assessment of muscle activation using the interpolated twitch. The method consists in superposing a single supramaximal electrical stimulus or a short train of electrical stimuli to an MVC (a). When extra force is produced by the superimposed electrical stimulation (ES) on the ongoing MVC force, this indicates that the subject is unable to produce a maximal activation of his muscle and a deficit in voluntary activation can be calculated. In (b), the histograms show that the mean deficit in activation (expressed in % of maximum) during an isometric MVC varies between muscles. In (c), the torque-velocity relation of the knee extensors is compared for voluntary activation alone (VA) and when electrical stimulation (ES) is superimposed (VA + ES). Note that the deficit in voluntary activation is mainly observed in eccentric and isometric contractions but absent during concentric contractions. (The first two panels (a and b) are adapted from Kent-Braun & Le Blanc, 1996 and Behm et al., 2002 and panel (c) was redrawn from Westing et al., 1990.)

200–300 Hz (Miller et al., 1981; de Ruiter et al., 2004; Duchateau, unpublished observation). Although no difference between the maximal RFD during fast voluntary and electrically induced (8 pulses at 300 Hz) contractions was found for the knee extensor muscles, the force integral (index for the time course of the force production) over the first 40 ms

of a fast voluntary contraction represents, on average, only 40% of its value in maximal electrically induced contraction (de Ruiter et al., 2004). This would suggest that the voluntary activation does not allow reaching the maximal intrinsic RFD of the muscle. Although the capacity for fast voluntary force development varies considerably among

subjects, the ability to reach high RFD seemed to depend mainly on the rate at which muscle activation increases at the initiation of the contraction.

These observations indicate that the level of activation is not always maximal in untrained subjects and that a program of strength training may increase the level of voluntary activation, especially for maximal isometric and eccentric contractions, and RFD. This might be of particular interest for stretch-shortening type performances.

Neural Adaptations

As mentioned in the previous section, strength gains may result, in part, from adaptations within the nervous system. Indirect evidence of neural adaptations is the dissociation between structural (muscle size) and functional changes. For example,

it has been shown by Staron et al. (1994) that a program (8 weeks) of strength training increased the 1-RM up to approximately 100%, without any significant changes in the CSAs of muscle fibers. It is well documented, moreover, that the time course of adaptation differs between neural and muscular factors (Sale, 1988). At the early part of a training program, strength increases before any detectable changes in muscle CSA (Narici et al., 1989) suggesting that adaptations should occur in the nervous system. More recently, Woolstenhulme et al. (2006) also reported that knee extensors strength increased before any change in either cytoskeletal proteins or fibers CSA. This dissociation is often interpreted as evidence of a contribution to voluntary strength gains by the so-called “neural factors” (Figure 13.7; Sale, 1988; Duchateau et al., 2006; Enoka, 2008).

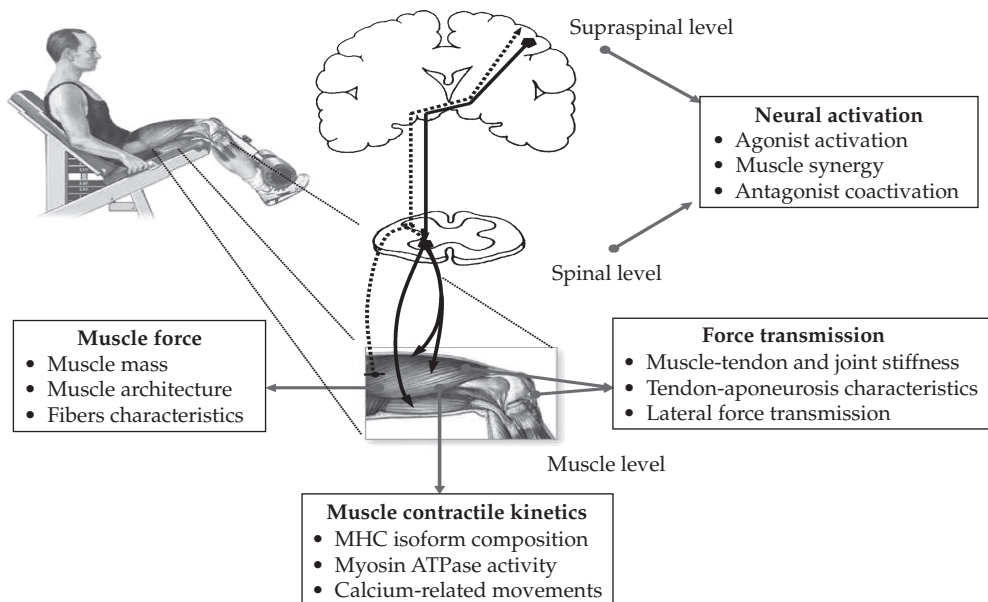


Figure 13.7 Schematic presentation of the possible loci of neural and muscular adaptations during the course of a strength or power training program. Potential sites within the nervous system include supraspinal and spinal centers leading to an increase in neural activation of the main agonist muscles, a reduction in antagonist coactivation, and/or an improvement in synergy between muscles. The continuous lines represent the descending drive to the muscles whereas the dotted lines correspond to the afferent feedback from the muscle receptors. At muscular level, potential adaptations can occur at both muscle structure and tendon characteristics. Depending on the training program, these adaptations are susceptible to increase the maximal force-generating capacity of the muscle, the muscle contractile kinetics, and to improve force transmission to the skeleton.

Change in Agonist Muscles Activation

The dissociation between changes in voluntary contractions and in muscle size does not inform, however, as to whether the intensity of the neural drive to muscle during maximal contraction is really increased after strength or power training.

Whole-Muscle Level

The changes in voluntary activation during maximal contraction or at the onset of a fast contraction after a training program has been quantified by measuring changes in the surface EMG activity (Moritani & de Vries, 1979; Häkkinen et al., 1998; Aagaard et al., 2000); interpolated twitch (Amiridis et al., 1996; Knight & Kamen, 2001); and ratio of evoked tetanic force to MVC force (Davies et al., 1985; Duchateau & Hainaut, 1988).

At the whole muscle level, the classic approach consists in recording changes in surface EMG activity during a maximal contraction. For example, it has been found that the average EMG during an MVC often increases concurrently with maximal force after a strength-training program (Moritani & de Vries, 1979; Häkkinen et al., 1998; Aagaard et al., 2000). These results contrast with those of other studies that did not find that EMG increased with MVC force (Carolan & Cafarelli, 1992), even

when the EMG was normalized to the maximal M wave, i.e., the compound maximal action potential recorded from surface electrodes in response to nerve stimulation. This normalization procedure is often used (Keen et al., 1994) to avoid the confounding effect of training-related changes of the muscle fiber membrane and to assess more objectively neural adaptations. Training program using fast contractions has been shown to induce earlier muscle activation (Van Cutsem et al., 1998) and greater surface EMG at the onset of a ballistic contraction (Figure 13.8; Häkkinen et al., 1985; Van Cutsem et al., 1998; Gruber et al., 2007). Despite a change in the EMG pattern between the eccentric and concentric phases of a stretch-shortening exercise after plyometric training has been reported (Kyröläinen et al., 1998; Kubo et al., 2007), the authors concluded that the increase in jump height after training cannot be mainly explained by such neural adaptations but rather relies more on changes occurring in the MTC. The increase in muscle preactivity prior to the landing phase in DJ after training, however, is reported to increase the musculo-tendinous stiffness (Gollhofer & Kyröläinen, 1991), and consequently the amount of stored and reused elastic energy (Horita et al., 2002; see Chapter 7).

An alternative approach is to assess the level of muscle activation (amount of motor drive)

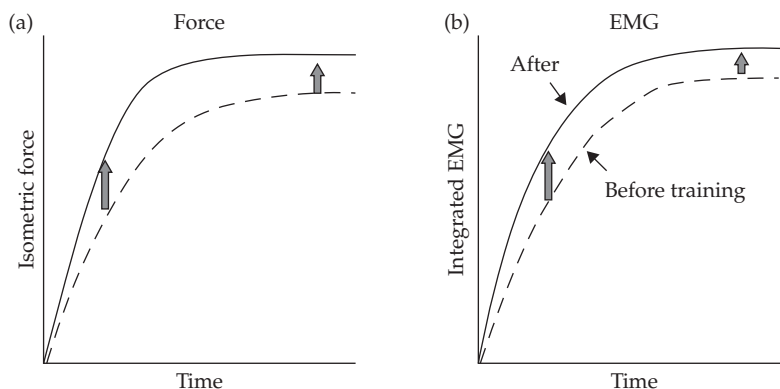


Figure 13.8 Change in the RFD (a) and corresponding iEMG (b) of the leg extensor muscles after a training program of fast (explosive) contractions (jump training). (a) Indicates that the maximal rate of isometric force development increased more (big arrow) than the peak force (small arrow). (b) Shows a similar adaptation for voluntary activation as the iEMG increased proportionally to a greater extent (big arrow) at the onset of muscle activation than during the force plateau (small arrow). Data are from Häkkinen et al., (1985) and the figure redrawn from Sale (1988).

by stimulating electrically the muscle during an MVC (twitch interpolated technique; Merton, 1954; Gandevia, 2001). However, because activation seems to be near maximal in many muscles when assessed with this technique before training (Figure 13.6b), there are minimal changes after strength training when the activation of the motor unit pool is estimated with the twitch superimposition technique (Harridge et al., 1999; Knight & Kamen, 2001; Pucci et al., 2005). Another method to investigate the neural drive to the agonist muscle consists of comparing the MVC force with the force developed in response to a train of stimuli (tetanic force). When using this method, Duchateau & Hainaut (1988) observed an increase in the ratio of MVC force to tetanic force for the adductor pollicis muscle after 6 weeks of strength training. The training program involved voluntary contractions and loads that were 60–65% of maximum. The greater increase in MVC force (22%) compared with tetanic force (15%) suggests that the training produced an adaptation that resulted in a 7% increase in the activation of the motor unit pool for the hand muscle (Figure 13.9). It is, however, not excluded that training may also have improved the contribution of the

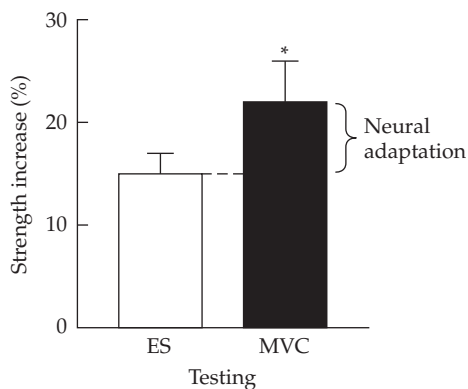


Figure 13.9 Comparison of the increase in strength of the adductor pollicis muscle when tested by a maximal voluntary contraction (MVC) and a maximal electrical stimulation (ES) after a 6-week strength-training program with a load of approximately 65% of maximum. The difference between the increase in MVC force (22%) and tetanic force (15%) indicates the extent of the increase in neural adaptation. (Data from Duchateau & Hainaut, 1988.)

synergist muscles to increase postural stabilization (Le Bozec & Bouisset, 2004) and thereby increasing the efficiency of force transmission from the hand muscle during the MVC (see also Baudry & Duchateau, 2007).

In conclusion, even if a decrease in the deficit in voluntary activation detected with the interpolated twitch technique and an increase in the ratio of tetanic and MVC forces may indicate that muscle activation is enhanced after strength training, it remains some uncertainty about the exact source of the improvement.

Multi-Unit Level

A major limitation of the previously evoked techniques, however, is that they cannot distinguish between the respective contribution of the recruitment and rate coding in the observed increase in MVC force. Because the upper limit of motor unit recruitment is about 80–90% MVC for most of limb muscles (Kukulka & Clamann, 1981; De Luca et al., 1982; Van Cutsem et al., 1997), increases in force above this level can only be achieved with adaptations in discharge rate. However, Pucci et al. (2005) reported that strength training increased voluntary activation from 96% to 98%, but without any change in discharge rate as measured with multi-unit recordings. Direct recording of motor unit activity, although challenging, provides more accurate evidence of the effect of training on motor unit recruitment and rate coding.

The potential mechanisms that might explain the increased activation of the agonist muscles after training include subtle changes in the pattern of motor unit recruitment and increases in the neural drive (Semmler & Enoka, 2000; Enoka, 2008). Although the timing of motor unit activation can be slightly changed with training (Cracraft & Petajan, 1977), it has been shown that the recruitment order of a motor unit population still followed the size principle during a ramp contraction, after either isometric or dynamic training (Hainaut et al., 1981; Van Cutsem et al., 1998). Furthermore, the order of recruitment during ballistic contractions did not change after 12 weeks of dynamic training with loads of 30–40% of maximum in the TA (Van Cutsem et al., 1998).

The discharge frequencies of motor units during high-force contractions appear to place the motor units on the upper region, but not on the plateau, of the force–frequency relation obtained from electrically induced contractions (Macefield et al., 1996). Accordingly, the maximal RFD during a tetanic contraction is usually obtained at a frequency that is greater than the average discharge rate of motor units observed during fast voluntary contractions (Miller et al., 1981; de Ruiter et al., 2004). These observations suggest that the MVC force and its maximal rate of development are less than the intrinsic capacity of the muscle and can thus be improved by increasing motor unit discharge rate. Consistent with this expectation, changes in discharge rate with training have been observed at the onset of a fast contraction (Van Cutsem et al., 1998) and during MVCs (Patten & Kamen, 2000; Kamen & Knight, 2004). At the end of a training program involving the knee extensor muscles in groups of young and old adults, for example, MVC force and discharge rate in the VL increased for both the young and elderly adults by 29% and 36% and 15% and 49%, respectively (Figure 13.10; Kamen & Knight, 2004). Interestingly, the increase in discharge rate was mainly observed in the first part of the training program whereas the MVC force increased until the end of the training program, likely underscoring the

combined effect of neural and muscular adaptations and their different time courses.

As demonstrated by Van Cutsem and colleagues (1998), dynamic training also changes motor unit behavior during fast (ballistic) contractions. In this study, initial discharge rates of motor units in the TA was compared before and after 12 weeks of training with ballistic contractions of the dorsiflexor muscles against a moderate load (30–40% MVC). Although no change was observed in the recruitment order of motor units, the instantaneous discharge rate of the first four action potentials increased from 69 to 96 Hz with training (Figure 13.11a–c). Furthermore, training caused a significant increase in the number of motor units (from 5% to 33%) that discharged with brief interspike intervals (<5 ms; Figure 13.11d). Thus, the increase in the RFD during rapid contraction appears to have been achieved by an adaptation in motor unit discharge rate. Such possibility has been assessed by computer simulation program developed by Fuglevand et al. (1993) and data showed that discharge rates comprised between 100 and 400 Hz can increase the RFD in the TA muscle. Due to technical limitations, yet no study has recorded the behavior of single motor unit during SSC exercises.

Motor unit synchronization is another frequently proposed mechanism to explain the increase in

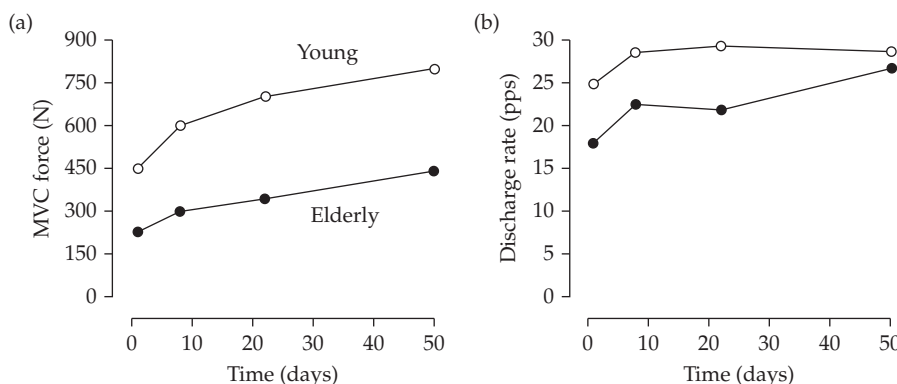


Figure 13.10 Changes in MVC force of the quadriceps femoris muscles (a) and the discharge rate of motor units in the VL (b) of young (open circles) and old (filled circles) adults during the course of a strength-training program with maximal isometric contractions. Motor unit discharge rates were recorded during a maximal (100%) isometric contraction. Note the rapid increase in discharge rate and in MVC force at the beginning of the training program. (Data from Kamen & Knight, 2004.)

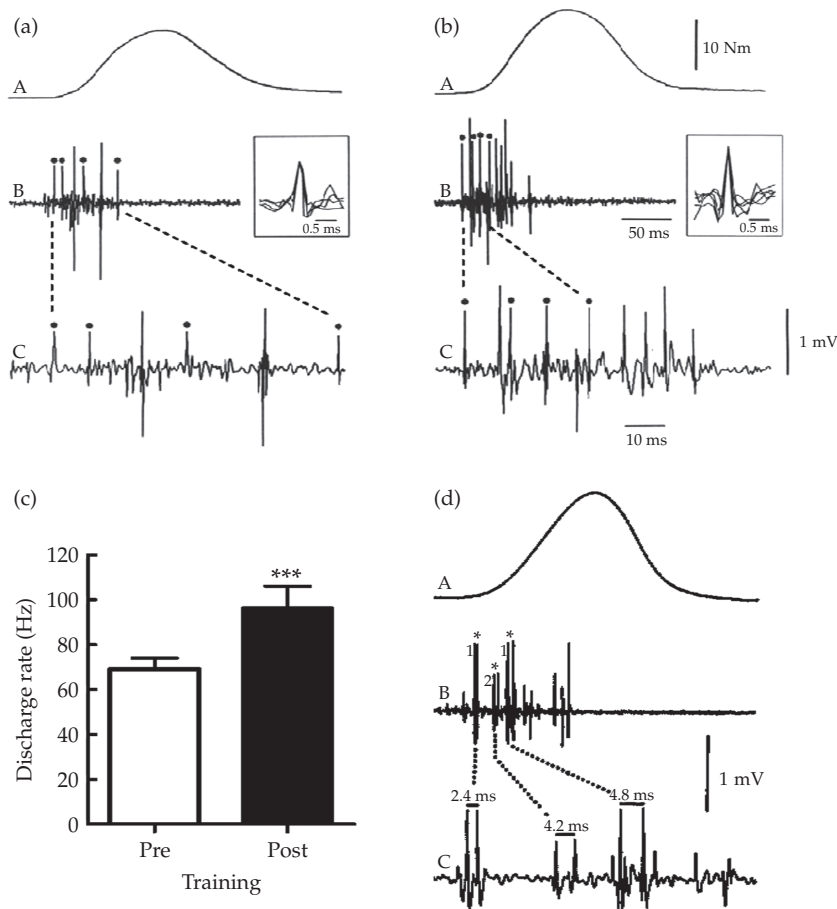


Figure 13.11 Comparison of motor units behavior from the TA during ballistic contractions of similar force levels (~40% MVC), before (a) and after 3 months of dynamic training (b). Traces correspond to the ankle dorsiflexors force (A) and intramuscular EMG plotted at slow (B) and fast (C) speeds. (a) shows the typical discharge pattern of a motor unit in untrained muscle, consisting in a brief time elapse between the first two spikes followed by longer interspike periods. (b) illustrates the usual motor unit behavior in trained muscle showing that the high onset of instantaneous discharge frequency is maintained during the subsequent spikes. The asterisks indicate the discharge frequency of the same motor unit and their traces are superimposed with an extended display (B). In (c), the histograms illustrate the mean maximal discharge rate averaged for the first three interspike intervals of ballistic contractions for the whole motor units population analyzed before and after training. ***Denote significant difference ($P < 0.001$) before and after training. (d) shows doublet discharges of motor units during the ballistic contraction after the training program. Two different motor units which started to discharge with a doublet are illustrated. Motor unit 1 fired two doublets (2.4 and 4.8 ms, respectively) while motor unit 2 discharged with a double spike of 4.2 ms interval. The asterisks indicate double discharges. (Adapted from Van Cutsem et al., 1998.)

force through neural adaptations (Milner-Brown et al., 1975). Synchronization of motor unit impulses is usually defined as the coincidental discharge of impulses from two or more motor units. The most widely accepted mechanism responsible for the

short-term synchronization is related to branched inputs from last-order neurons that increase the probability of simultaneous discharge in the motor neurons sharing these inputs (Nordstrom et al., 1992; Schmied et al., 2000; Semmler, 2002).

In their pioneering study, Milner-Brown et al. (1975) reported that weightlifters have a higher synchronization index compared with untrained subjects and that strength training increases synchronization between motor units. However, their method based on a surface EMG-based population index to examine synchronization in the FDI muscle has several limitations. A more reliable technique to assess the level of motor unit synchronization in human muscles is the cross-correlation of the discharge times of two concurrently active motor units (Farmer et al., 1997). The analysis involves constructing a cross-correlation histogram and calculating an index of synchronization determined from the peak of the histogram. Although the amount of motor unit synchronization can vary with the contraction type (Semmler et al., 2002) and the type of habitual activity performed by the individual (Semmler & Nordstrom, 1998; Semmler et al., 2004), it does not appear to change with strength training. Indeed, a recent study of Kidgell et al. (2006) showed that a 4-week training program increased the strength of a hand muscle by 54% without any change in the amount of synchronization between pairs of motor units. In addition, computer simulations by Yao et al. (2000) indicated that motor unit synchronization does not increase the maximum force exerted by muscle during steady-state isometric contraction. These findings suggest that synchronization is not an important parameter in the expression of muscle strength.

Changes in Antagonist Coactivation and Coordination Between Muscles

In addition to an intensified neural drive to the motor neuron pools of the prime mover muscles with strength training (Figure 13.7), changes can involve both the relative activation of different motor neuron pools and the connectivity within and between pools. These adaptations can modulate the amount of antagonist coactivation and the level of activation of the different synergist muscles.

Changes in Antagonist Coactivation

Coactivation is classically defined as the concurrent activity of the antagonist muscles during the

activation of the agonist muscles. The level of coactivation is classically quantified by computing the ratio between the agonist and antagonist EMG activities. Coactivation increases joint stability and stiffness, and varies with factors such as the intensity and type of the contraction, movement speed, the amount of fatigue, and the level of training (for a review, see Kellis, 1998). Because excessive coactivation should reduce the net force produced by the agonist muscles, the level of coactivation has to be adjusted by the nervous system to not impede the performance of the agonist muscles. Alterations in coactivation with training maybe related to a change in the ability to focus the motor command to the appropriate muscles involved in the task and/or to improve the timing between agonist and antagonist activities. For example, elite athletes exhibit reduced coactivation of the semitendinosus muscle compared with sedentary subjects when performing isokinetic contractions with the knee extensor muscles (Amiridis et al., 1996). This is consistent with the reduced coactivation of one of the antagonist (BF) muscles within the first weeks of strength training of the knee extensor muscles using isometric MVCs (Carolan & Cafarelli, 1992). Similarly, Pensini et al. (2002) found that substantial increases in plantar flexor force after 4 weeks of training with eccentric contractions was accompanied with a significant decrease in antagonist coactivation during concentric (−27%) and eccentric (−22%) actions. Kyröläinen and colleagues (1998) also found a decrease in coactivation during the course of a 12-week training program with plyometric exercises involving upper limbs in untrained women. In contrast, Häkkinen et al. (1998) found that an increase in isometric knee extensor force (36% and 57% in elderly men and women, respectively), after 6 months of training consisting of lifting and lowering a load that increased progressively from 50% to 80% of 1-RM during the training program were accompanied by decline (13% and 22%, respectively) in coactivation of the antagonist (BF) muscle with no change in middle-aged men and women. Although additional experiments are needed to understand the reasons of this apparent discrepancy between studies in young adults, they nevertheless suggest that when coactivation is

reduced, the net force about the joint can increase due to the removal of the negative force produced by the antagonist muscle.

Change in Coordination Between Muscles

Among the potential neural factors, a major contributor to the improvement in performance is the coordination of activity among the synergist and postural muscles involved in the task (Carroll et al., 2001; Carson, 2006). As already mentioned, this factor is often considered to play a major role in the specificity of strength gains. It has been indeed reported by Carroll et al. (2001) that resistance training can improve the ability of individual to perform rhythmic tasks. In their study, the authors compared the ability of individual to perform flexion or extension movements of the index finger at different cadences imposed by an auditory metronome, before and after 4 weeks of training. The training program, which consisted in lifting and lowering a load of approximately 70% over a 45° range of motion with the index finger extensors, increased the maximal isometric strength by 26%. Before training, the pattern of extension phase of the index finger during rhythmic movements at imposed cadences was highly variable and further the pattern of coordination compromised as the frequency of movement was increased. After training, the performance was improved and was associated with a more consistent activation pattern of the muscles during the task as attested by the changes in surface EMG activity. These observations indicate that strength training produces functional adaptations of the neuro-mechanical constraints that underlie the control of the task.

Presumably, early gains in a strength-training program are related to learning the appropriate activation pattern for the task, especially for a novel task. Such adaptations would influence two aspects of a strength manoeuvre: the postural support for the task and the goal-directed movement itself. As with coactivation, however, these adaptations have not been examined at the level of the motor unit.

Potential Locations for Neural Adaptations

Despite the evidence that suggests a significant role for neural mechanisms in strength-training

adaptations, the specific sites of adaptations along the neuraxis have seldom been identified experimentally, and most of the evidence for neural adaptations following strength training remains indirect (Duchateau & Enoka, 2002). The changes that can be evoked in neural circuits with training are produced, in general, by adaptations at one or both of the following levels (see Figure 13.7): (1) supraspinal level (corticospinal neurons, subcortical neurons, and inhibitory and excitatory intracortical interneurons); (2) spinal level (motor neurons and inhibitory and excitatory interneurons).

Supraspinal Level

Some studies involving short-term motor skill training have reported that changes within the primary motor cortex were associated with extension of the cortical representation of the muscles involved in the task and with an increased excitability of the descending (corticospinal) pathway (Figure 13.7; Pascual-Leone et al., 1995; Classen et al., 1998), possibly due to selective alterations in intracortical inhibition (Liepert et al., 1998). Despite the observation that several weeks of skill training can increase corticospinal excitability (Pascual-Leone et al., 1995; Jensen et al., 2005), strength training does not seem to be accompanied by similar adaptations (Carroll et al., 2001; Jensen et al., 2005). For example, Jensen et al. (2005) found that skill training 3 times per week for 4 weeks increased the maximal MEP induced by TMS and decreased the minimal stimulus intensity required to elicit MEPs at rest and during a contraction. In contrast, the maximal MEP and the slope of the input–output relation both decreased significantly at rest, but not during contraction in strength-trained subjects. Similarly, Carroll et al. (2001) found that the 33% increase in maximal isometric force of a hand muscle after 4 weeks of strength training was not accompanied by changes in corticospinal excitability measured at rest and was reduced when tested during contractions. They also observed that the degree of reduction in MEPs evoked by TES and TMS was similar. Because TMS largely excites cortical neurons through interneurons and TES excites the corticospinal fibers at the axon hillock (Rothwell, 1997), these results suggest

that strength training changed the functional properties of spinal cord circuitry, but not the output from the motor cortex. In contrast, Beck and colleagues (2007) reported that MEP size increased in the TA following 4 weeks of ballistic training of the dorsiflexor muscles against a load of 30–40% of 1-RM. This change was not associated with changes in SICI and ICF. Since spinal excitability tested by the Hoffmann reflex (H-reflex) remained unaltered, it was suggested that changes most likely occurred at the supraspinal level. Although there are no clear explanations for the increase in MEP size after ballistic training, results further showed that the adaptations were task-specific because no changes were observed at rest or in nontrained movements. This specificity of the adaptation may likely explain the apparent discrepancy between the different studies. Indeed, specific training effect maybe observable provided that the training and testing conditions are the same. Moreover, the absence of increase in corticospinal excitability after strength training does not mean that no adaptation occurred at the supraspinal level. As shown from functional imaging (fMRI) studies (Doyon & Benali, 2005), acute changes during the process of motor learning are accompanied by an increase in activity at the cortical level, which subsequently shifts toward subcortical structures such as the basal ganglia and the cerebellum during the final stage of automatic movement performance (Floyer-Lea & Matthews, 2004; Puttemans et al., 2005). Consistent with that discussion, it has been shown that unilateral strength training increases voluntary activation of the opposite untrained limb (“cross education” effect) so that, in addition to adaptations located at spinal and cortical levels, subcortical mechanisms are presumably involved (Carroll et al., 2006).

Spinal Level

The motor unit activity depends both on descending drive and peripheral input that maybe indirectly investigated by recording the changes occurring in reflex responses after a training intervention (Figure 13.7). Limited studies have investigated the effect of training on the spinal stretch reflex. Among them, Häkkinen and Komi (1983)

reported that the EMG amplitude of the stretch reflex in the knee extensors elicited by tendon tap decreased after 16–24 weeks of heavy strength training. In contrast, Voigt and colleagues (1998) observed an increase in the tendon tap for the SOL but no change for the MG after 4 weeks of jumping (hopping) training. Although these studies indicated training- and muscle-specific adaptations, tendon reflex was recorded at rest. Therefore, care must be taken when these data are used for interpretation of reflex changes in response to training intervention (Zehr, 2002). In the study by Voigt et al. (1998), however, change in spinal excitability has also been analyzed by means of the SLC of the stretch reflex in the SOL and MG during hopping. The data showed no change in both muscles after training. In addition to the discrepancy between these stretch-induced methods, the results reinforce the assumption that training effect must be assessed in the training task.

Because stretch reflex and tendon-tap reflex involved both neural and musculo-tendinous components that increase the difficulty to determine the exact loci of the adaptations, training effects within the spinal cord are often assessed in humans by testing electrically evoked reflexes, such as the H-reflex. This reflex includes a monosynaptic connection between the group Ia afferents and the alpha motor neurons. Several studies have shown that strength training increases the amplitude of the H-reflex when tested during muscle activation (Aagaard et al., 2002; Scaglioni et al., 2002; Lagerquist et al., 2006; Taube et al., 2007; Holtermann et al., 2007). In addition, adaptation appears to be muscle and contraction type dependent as a 7-week training program of eccentric contractions increased the H-reflex amplitude of the SOL only during eccentric MVC but regardless of the contraction type (isometric, concentric, and eccentric contractions) for the MG (Duclay et al., 2008). Similarly, a program of hopping exercises increased the H-reflex amplitude during functional exercise (Voigt et al., 1998). Furthermore, when an electric stimulus sufficient to evoke a maximal M wave is applied to a motor nerve during an MVC, two reflex responses (V1 and V2) can be elicited. The V1-to-M wave ratio has been used as an index of reflex potentiation, and has been shown

to increase after strength training (Sale et al., 1983), suggesting an increase in the efferent drive to the muscle after strength training (Aagaard et al., 2002). Observations on the operant conditioning of the spinal stretch reflex and the H-reflex in animal suggest that most of the plasticity in these reflexes appears to be located in the spinal cord (Wolpaw et al., 1994) and appears to be attributable to the role of spinal interneurons in integrating the sensory and motor signals that are transmitted to the motor neurons (Nielsen, 2004). In that context, the reduced antagonist coactivation observed after strength training (Carolan & Cafarelli, 1992) maybe the consequence of an increased reciprocal inhibition. Regardless of the exact location of the adaptation that occurs at the spinal circuitry, the absence of adaptation in H-reflex amplitude after ballistic and sensorimotor training, while it increase after strength training, suggests that spinal reflex circuits can adapt specifically to the particular requirements of the task (Taube et al., 2007). From a functional point of view, decreased spinal reflexes may improve movement control in unstable and difficult (eccentric contractions) conditions by preventing reflex-mediated joint oscillations. These neural adjustments may suggest that the control of movement is shifted to supraspinal centers with the goal to control more precisely the activation. In contrast, strength and power training can benefit from high reflex gain, i.e., increase in the input/output relation at the motor neuron pool level, and therefore, any inhibitory mechanisms that diminish the excitatory drive would be counterproductive. Although many investigators have used the H-reflex as an index of motor neuron excitability, the connection between the afferent and the motor neuron is modulated by presynaptic mechanisms, and hence, the amplitude of the H-reflex depends on more than the responsiveness of the motor neuron (Zehr, 2002). In this context, given the observation that acute withdrawal of group Ia feedback reduces discharge rate during a maximal contraction (Macefield et al., 1993), it is likely that training-induced changes in afferent feedback can also influence motor unit discharge rate. In addition, Rempel and colleagues (2001) observed that rats trained to perform a strong contraction during reaching task

exhibited an increase in the density of excitatory, but not inhibitory, synapses onto motor neurons in cervical segments of the spinal cord.

In addition to a possible role of afferent feedback in mediating training adaptations, other evidences also indicate that the properties of the motor neurons themselves can be altered by physical activity (Gardiner, 2006). For example, Beaumont and Gardiner (2003) reported that endurance training in rats changed the biophysical properties of motor neurons, which resulted in a more hyperpolarized resting membrane potential, increased threshold for spike initiation, and faster rise times for antidromic spikes. These adaptations, which likely reflect alterations in ionic conductance of the membrane of motor neuron, can modify the recruitment thresholds and discharge patterns of the neurons. Presumably, strength and power training are likely to cause different adaptations and may explain the greater occurrence of doublet discharges after training with ballistic contractions (Van Cutsem et al., 1998). Such high frequencies, that were observed at the onset of motor neuron discharge in response to fast current injection (Baldissera et al., 1987), maybe related to changes in the intrinsic properties of the motor neuron (Granit et al., 1963). A motor neuron capable of producing a double discharge may undergo a state of increased depolarization (delayed depolarization) that occurs during the falling phase of the action potential (Garland & Griffin, 1999). During this period, the motor neuron is still sensitive to intense synaptic input, and thereby susceptible to reach the threshold of activation and produce a second action potential at a very brief interval.

In conclusion, the neural adaptations that accompany changes in physical training are diverse and distributed along the entire neuraxis (Duchateau & Enoka, 2002). There is some evidence that these adaptations can contribute to improvements in motor performance and are specific to the characteristic of the training task. Because investigation of changes within the nervous system is limited by technical constraints, however, most of the studies report only indirect evidence to support the association between the neural adaptations and changes in motor performance.

Muscular Adaptations

As discussed previously, most of the difference in maximal strength between individuals and its increase following training is due to structural adaptations. In the following sections, we will review different musculo-tendinous factors that can contribute to improve muscle performance (Figure 13.7).

Muscle and Motor Unit Contractile Properties

The amount of the increase in muscle and motor unit force-generating capacity and their associated contractile kinetics can be quantified by recording the mechanical response produced by a maximal tetanic contraction.

Muscle

There are only few studies in the literature on the effects of training on muscle and motor unit contractile properties in human. The main reasons are that maximal tetanic contractions are relatively painful and can present technical limitations in humans *in vivo*. Indeed, the stimulation of some motor nerves can induce the concurrent activation of agonist and antagonist muscles contributing thereby to underestimate the maximal strength of the agonist muscles. In that context, an interesting model to investigate adaptations of the muscle contractile properties following strength training is the adductor pollicis muscle. Among the different muscles of the thumb, this is the only one with, inconsistently, the deep head of the flexor pollicis brevis to be activated when the ulnar nerve is stimulated at the wrist (Merton, 1954). Duchateau and Hainaut (1984) used this technique to compare the effect of two types of training in human volunteers. One group of subjects trained the muscle with maximal isometric contractions (10 contractions of 5 s duration), and the other group performed rapid shortening contractions against a load of 30–40% of maximum during 3 months. The load for the dynamic contractions was chosen because muscle produced maximal power when the contraction force is about 30–40% of maximum in the adductor pollicis (Duchateau & Hainaut, 1984; Baudry & Duchateau, 2007). The study indicated that

voluntary isometric and dynamic exercises have different effects on the muscle maximal force and speed of tetanic contraction. The maximal muscle force increased to a greater extent (20%) after maximal isometric contractions than after dynamic contractions with small load (11%) whereas the RFD was mainly augmented after dynamic training (31% vs. 18%). The difference in the increase in strength is not surprising since it is well known that the gain in force is positively correlated with the intensity of the contraction. However, the greater increase in RFD after dynamic training against light loads indicates that this method is more effective than contractions at maximal intensity to increase the intrinsic contractile kinetics of a muscle. The duration of the training program is also an important parameter to induce changes in the force-generating capacity of the muscle. For example, McDonagh and colleagues (1983) observed no significant changes in tetanic force of the BB after 5 weeks of isometric MVCs of elbow flexors. Similarly, the maximal tetanic force of the adductor pollicis increased moderately (~5%) in the first part of a training program using maximal isometric contractions but more substantially after 40 days of training (Figure 13.12). After 90 days of training, tetanic force increased by 21%.

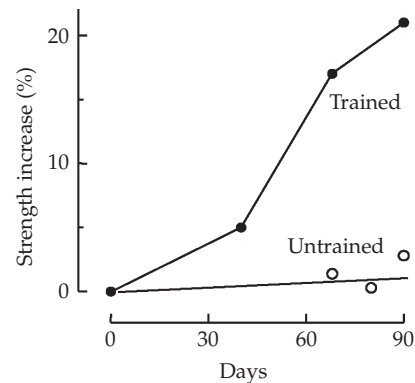


Figure 13.12 Change in maximal tetanic force (100Hz) of the adductor pollicis muscle during the course of a 3-month strength training program. The load was comprised between 60% and 70% of maximum. The increase in tetanic force in the trained muscle was small during the first 40 days (5%) but increased more substantially thereafter, to reach 21% at the end of the training program. No change was observed in the muscle of the contralateral hand (untrained muscle).

Motor Unit

Only one study has recorded the tetanic force of the same motor unit before and after training in humans (Chan et al., 1999). This method consists in the use of percutaneous electrical stimuli to excite single motor axons at a number of sites along the median nerve and to record their contractile properties in thenar muscles. Because the shape and size of motor unit action potentials are relatively reproducible when the electrodes are replaced at a same location in subsequent sessions, the longitudinal tracking of the same motor units is possible. With such experimental approach, Chan and colleagues (1999) reported that motor unit adaptation to a program of high-frequency electrical stimulation differed with their physiological characteristics. While the twitch and maximal tetanic forces of the slower and fatigue-resistant unit increased, surprisingly the force of the faster and more fatigable unit declined. A major drawback of the method for assessing the effects of a training intervention, however, is that the number of motor unit per subjects is very low. For these reasons, some investigators preferred to use another method, called the “spike-triggered averaging” (Stein et al., 1972). Due to its unique design, this method is the only one that can be used to examine the contractile properties of individual motor unit activated voluntarily. Briefly, the action potentials from a single motor unit discharging at low frequency, detected by an intramuscular needle electrode, are used as “spike triggers.” Because the force generated by that particular motor unit is time-locked to the action potential (spike), it can be extracted by averaging of the force signal. When comparing the effect of isometric and dynamic training (see earlier), specific adaptations were observed at motor unit level (Hainaut et al., 1981). After isometric training, all motor units showed a roughly proportional increase in peak force without change in twitch time course. As expected, motor units showed less increase in force after dynamic training but the time-to-peak of the whole population of motor units was reduced. There was no evidence of a change in the “size principle” after either dynamic or isometric training, since a linear relation was

consistently observed between the motor unit force and recruitment threshold (Hainaut et al., 1981). Similar adaptations were found in the TA after dynamic training (Van Cutsem et al., 1998). Once again, these studies indicate that muscle adapts its contractile properties specifically to the type of exercise training.

Relation Between Muscle Size and Strength

Theoretically, the maximal force that a muscle can exert depends mainly on the number of muscle fibers and the number of sarcomeres that are arranged in parallel in each fiber, as well as the angle formed between the fibers and the longitudinal axis of the muscle (angle of pennation). Therefore, the strength of a muscle can be estimated anatomically by measuring its CSA (Narici et al., 1996). This measurement should be made perpendicular to the direction of the muscle fibers (PSCA) but, for technical reasons, it is easier to measure the anatomical CSA, which consists of measuring perpendicular to the long axis of the muscle. In humans, this measurement is usually accomplished by means of imaging techniques (e.g., CT scan, MRI, and ultrasound; see Figure 13.13).

Association Between Muscle Size and Strength

Despite the possible bias of measuring anatomical CSA, a good association was found between the maximal force of the calf muscles during electrically induced contraction and the CSA of a muscle group (Davies et al., 1983). However, there is more variability in the relation when force is assessed during an MVC (Maughan et al., 1983; Schantz et al., 1983). For example, in some studies variation in CSA accounts only for about 50% of the difference in strength between subjects (Jones et al., 1989; Narici et al., 1996). This underscores that other factors than the muscle mass contribute to the force developed by a muscle.

Specific Tension

The maximal force that a muscle or a muscle fiber can exert per unit CSA (N/cm^2) is often referred to as “specific tension.” This parameter is an

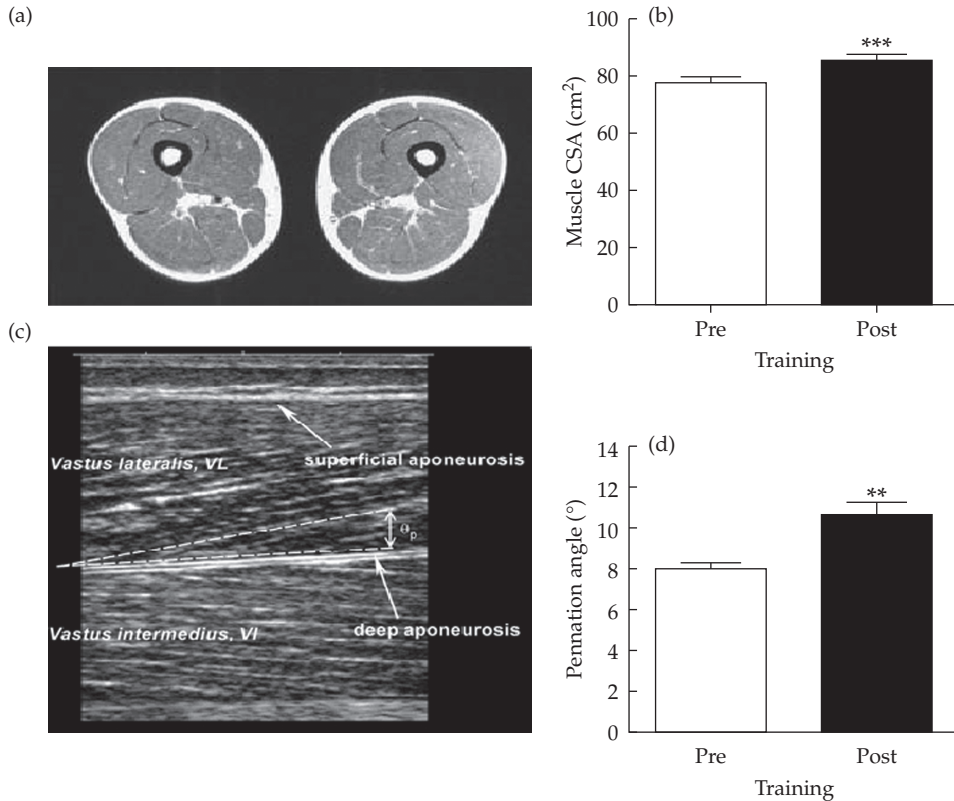


Figure 13.13 Structural and architectural adaptations of the knee extensor muscles after 14 weeks of strength training. CSA of the quadriceps was obtained by axial images of the thigh at mid femur length (a). Anatomical CSA of the quadriceps was increased by 10% (** $P < 0.001$) after the training program (b). In (c), illustration of a sagittal ultrasound image of the quadriceps showing the orientation of muscle fascicles. Pennation angle (θ_p) of the VL was determined between VL muscle fascicles and the deep aponeurosis separating VL and vastus intermedius. In (d), the histograms illustrate the mean value for pennation angles of the VL for all subjects before and after the strength-training program. The pennation angle increased (** $P < 0.01$) by 35% after training.

indication of the intrinsic force-generating capacity of the muscle or muscle fibers. At the whole muscle level, specific tension has been found to be greater (Maughan et al., 1983; Kanehisa et al., 1994) or similar (Kanehisa et al., 1994; Schantz et al., 1983) in trained compared with untrained women, depending in part on the investigated muscle group (Kanehisa et al., 1994). Furthermore, Schantz and colleagues (1983) reported no difference in strength per unit CSA between elite bodybuilders, with extreme muscle hypertrophy, and students in physical education. Nonetheless, the estimate of specific tension from the whole muscle can be

misleading as the amount of connective tissue can differ between individuals. Some of this variability may also be due to the use of anatomical rather than PSCA as an index of muscle size and to a submaximal activation of the muscle group by the volitional drive. These confounding factors can be circumvented by recording specific tension at the muscle-fiber level. In these conditions, it has been shown, for example, that specific tension for type II fibers of the VL is greater than that for type I fibers for young active adults but not for sedentary old adults (Larsson et al., 1996; Harridge et al., 1996; D'Antona et al., 2006) and greater for type II but

not for type I fibers of bodybuilders as compared to sedentary individuals (D'Antona et al., 2006). Furthermore, specific tension was found to increase following a strength-training program (Aagaard et al., 2001; Harber et al., 2004). At the muscle-fiber level, there are at least two mechanisms that can account for variation in specific tension: the density of the myofilaments in the muscle fibers (Claassen et al., 1989) and the efficacy of force transmission from the sarcomere to the skeleton (Patel & Lieber, 1997).

Change in Whole Muscle Mass and Architecture

Due to the high correlation between the maximal force of a muscle and its CSA, an increase in the latter parameter leads to an increase in maximal force (Figure 13.13). Two mechanisms are susceptible to augment muscle mass: an increase in the CSA of individual muscle fibers (hypertrophy) and an increase in the number of muscle fibers (hyperplasia). Most experimental evidence suggests that hypertrophy is the main mechanism for the increase in muscular force (Figure 13.14; Sale et al.,

1987) but hyperplasia may occur in human under some conditions (Kadi & Thornell, 2000).

Muscle Mass

Today, it is well known that the magnitude of the increase in CSA with training depends on several factors, such as the initial strength of the individual (Häkkinen et al., 1987; Alway et al., 1992), the level of the load (McDonagh & Davies, 1984; Sale et al., 1987), the duration of the training program (McDonagh et al., 1983; Duchateau & Hainaut, 1984), and the training method (Enoka, 2008). For example, 6 weeks of isometric training (~80% MVC) in novice individuals increased the CSA of the elbow flexor muscles by approximately 5% (Davies et al., 1988), whereas 8 weeks of similar training of the quadriceps femoris has been shown to increase the CSA by 15% (Garfinkel & Cafarelli, 1992). In contrast, 24 weeks of heavy strength training by highly competitive bodybuilders did not change substantially the CSA of muscle fibers in BB (Alway et al., 1992). Although the mechanisms are still unknown, loads of 60–80% of 1-RM with 6–12 repetitions and 6–10 sets, as used by bodybuilders,

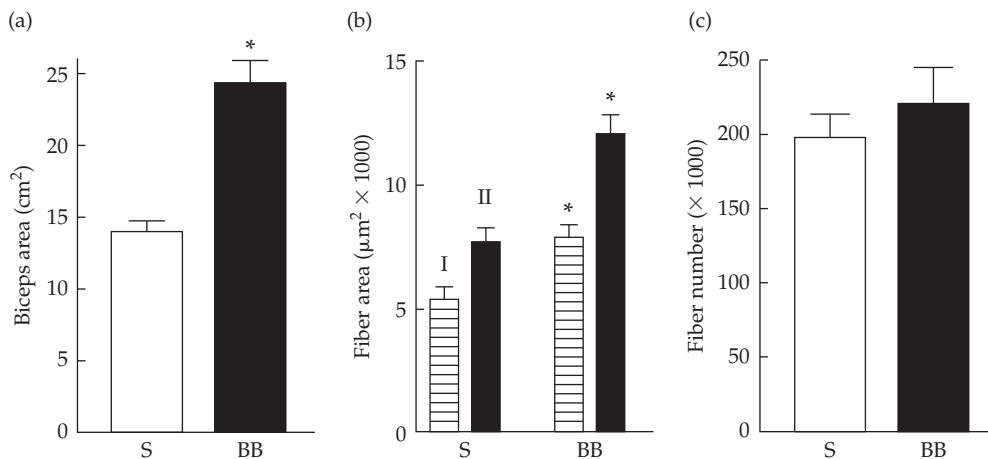


Figure 13.14 Comparison of muscle mass, muscle fiber size, and number of fibers in biceps brachii of sedentary subjects (S) and (BB). The mean CSA for the whole muscle, obtained by computerized tomographic scans of the upper arm (a), and for the two main types of muscles fibers (type I and II), measured from needle muscle biopsies (b), differ between S and BB. In contrast there is no significant difference in the estimated mean fiber number obtained by computing the ratio between the whole muscle CSA and the average fiber CSA in each subject (c). *Denote significant differences (* $P < 0.01$) between the two groups. (Data from Sale et al., 1987.)

seem to be more effective for muscle hypertrophy than heavier loads (>80% of 1-RM) with a reduced number of repetitions (Tesch, 1992). Furthermore, eccentric contractions have been shown to have greater effect on muscle hypertrophy as suggested by the greater increase in CSA after a training program for the knee extensor muscles including concentric and eccentric muscle actions as compared with a training program with only concentric actions (Hather et al., 1991). It has also been reported that strength training can lead to differences in the hypertrophy of the different constituents of a muscle group. For example, after 6 months of strength training of the knee extensor muscles with a load of 80% of maximum, the quadriceps CSA increased by approximately 19% at the distal and proximal regions but only by 13% in the central region of the muscle (Narici et al., 1996).

Muscle Architecture

Although not always observed (Rutherford & Jones, 1992), muscle architecture can be modified by strength training, in addition to changes in CSA (Kawakami et al., 1995; Aagaard et al., 2001). For example, Aagaard and colleagues (2001) measured muscle CSA and volume with MRI, and the pennation angle of muscle fibers in VL by ultrasound (Figure 13a and c). After 14 weeks of training, they observed an increase in MVC force (16%) of the knee extensors, in the volume of the quadriceps (10%; Figure 13.13b), and in the pennation angle of the muscle fascicles of the VL (36%; Figure 13.13d). The greater increase in MVC force compared with muscle volume was attributed to the increase in pennation angle, as it is known to increase the force capacity of the muscle per unit volume. As already evoked, these results have implication in the estimation of specific muscle tension. These results indicate clearly that physiological cross-section is much more accurate than anatomical cross-section to examine the peripheral adaptations to strength training. They nevertheless draw the attention to the fact that in some muscles (e.g., in calf muscles) excessive hypertrophy could increase pennation angle to a level that might be counterproductive for force or speed production.

Muscle Fiber Adaptations

The specific adaptations occurring with the various strength-training methods indicate that muscles are susceptible to undergo changes in their fiber type composition as well as to specific adaptations of their different muscle fiber types. However, despite the variability observed among muscle biopsies, most of the studies reported that strength training does not alter the proportion of type I and II fibers within muscles (Staron et al., 1991; Aagaard et al., 2001; Canepari et al., 2005), even in the case of a 45% increase in MVC force (Alway et al., 1989). However, within the type II, a common observation following a strength-training program is an increase in the proportion of type IIa fibers and decreases of type IIb (IIx) in VL (Staron et al., 1991; Hather et al., 1991; Adams et al., 1993; Hortobagyi et al., 1996; Canepari et al., 2005).

More divergences appear in the magnitude of hypertrophy between muscle fiber types. For example, a 16-week isometric program increased CSA of type I and II fibers by 20% and 27%, respectively, in the SOL whereas the lateral GAST was characterized by a 50% increase of the type II with no change of the type I fibers. Similarly, 14 weeks of training of the knee extensors increased the CSA of type II by 18% with no change in the type I fibers in the VL. Furthermore, programs that involved eccentric contractions appeared to favor the hypertrophy of type II fibers compared with concentric contractions (Hather et al., 1991; Hortobagyi et al., 1996).

Even if the amount of hypertrophy between type I and II fibers did not always differ substantially (MacDougall et al., 1979; Häkkinen et al., 1981), many studies indicate that most of the hypertrophy produced by heavy strength training occurs in type II fibers. Indeed, 6 months of training with heavy load (70–100% of 1-RM) and dynamic contractions (10–60% of 1-RM performed at maximal speed) increased the area of type II fibers by 29% and 13%, respectively (Tesch et al., 1987). A modest increase of approximately 4% in CSA of type I and II fibers was observed. Surprisingly, it has been shown recently that SSC training can induce substantial increase (22–30%) in muscle fiber CSA of the VL (Malisoux et al., 2006). We must, however, keep

in mind that the pretraining status of the subject is a determinant factor in the relative adaptation between the two main fiber types, because already trained athletes do not show such huge effect as observed in novice athletes (Häkkinen et al., 1987; Alway et al., 1992).

Mechanisms for Hypertrophy and Hyperplasia

This section does not intend to evoke in depth the new and important developments of the last few years in the mechanisms of increase in muscle mass. We will only give a brief overview of the mechanisms of hypertrophy and hyperplasia. For further explanations, the reader is referred to more specialized literature (Goldspink & Harridge, 2004; Kadi et al., 2005; Harridge, 2007).

Hypertrophy Mechanisms

The hypertrophy of muscle requires both a change in the net protein synthesis and an increase in the number of myonuclei to manage the increase in the volume of contractile proteins. In presence of chronic stretch-overload of the muscle, the protein synthesis is increased over degradation, resulting in a net contractile protein increase and thereby muscle hypertrophy. New myonuclei are differentiated from the so-called “satellite cells.” Satellite cells are quiescent mononucleated cells, which are located under the basal lamina (Kadi et al., 2005). In response to increased activation, they start proliferating and some fuse with a muscle fiber, thereby adding new nuclei to the existing fibers, whereas others can generate daughter cells that will become quiescent satellite cells (Kadi et al., 1999; Kadi & Thornell, 2000; Eriksson et al., 2005; Kadi et al., 2005). The new myonuclei will produce mRNA and proteins in the same way as the already existing myonuclei. The production of proteins will in turn increase and cause fiber hypertrophy. In the VL muscle, their number seems to be similar in type I and II fibers (Kadi et al., 2005; Thornell et al., 2003).

Currently, relatively little is known about the exact signal that induces the satellite cells to add new nuclei into a muscle fiber. Although it has been shown that satellite cells can be activated by a

single bout of high-intensity exercise, this stimulus is insufficient for cells to undergo terminal differentiation (Crameri et al., 2004; Dreyer et al., 2006). It appears, however, that differentiation of satellite cells into myonuclei requires a significant increase in fiber size. For example, the number of myonuclei is not modified when training increases fiber area by 17%, indicating that the increased fiber size is primarily caused by protein synthesis, whereas larger hypertrophy (>30%) is accompanied with an increased number of myonuclei. This is consistent with the strong association between the number of myonuclei added after strength training and the magnitude of fiber hypertrophy (Petrella et al., 2006). In summary, the primary response to hypertrophy stimuli is a net protein increase due to a change in the balance between protein synthesis and degradation, followed by activation and proliferation of satellite cells. The fusion of satellite cells to muscle fibers is taking place at a later stage of the hypertrophy process, perhaps when further hypertrophy can no longer be obtained by increased protein synthesis alone (Kadi et al., 2005).

There are a number of intracellular signaling pathways that can mediate the increase in protein synthesis and degradation. An important transduction pathway involves protein kinase B (Akt) which activates the mTOR pathway (Bodine, 2006). Akt signaling is itself activated by an isoform of insulin-like growth factor-1 (IGF-1) that is produced mainly by skeletal muscle in response to an exercise stress. This isoform of IGF-1, called “mechano growth factor,” contributes to the upregulation of protein synthesis (Goldspink & Harridge, 2004). After binding to a transmembrane receptor, it initiates a series of interactions that activate Akt, which increases protein synthesis through mTOR pathways and decreases protein degradation by phosphorylating a transcription factor that is expelled from the nuclei and reduces the production of proteins involved in degradation (Tidball, 2005). It seems, however, that other exercise signals besides mechano growth factor are also able to activate the Akt pathway and many of the underlying mechanisms of muscle hypertrophy remain unknown. Furthermore, whereas IGF-1 induces muscle growth, another

factor, myostatin appears to play a role as feedback inhibitor of muscle growth. Interestingly, following strength training, the level of myostatin mRNA is decreased (Roth et al., 2003; Kim et al., 2005; Kvorning et al., 2007) but endurance exercise appears also to reduce the relative myostatin level in absence of muscle fiber hypertrophy (Matsakas et al., 2005). Although mRNA isoform expression was found to be enhanced immediately after a single session of strength training (Caiozzo et al., 1996), it takes 3–6 weeks of training before protein synthesis produced detectable changes in muscle size (Seynnes et al., 2007) and increase in intrinsic muscle strength (see Figure 13.12; McDonagh et al., 1983).

Hyperplasia

Although most experimental evidence suggests that hypertrophy is the main mechanism of the increase in muscle mass, hyperplasia may also play a role in the increase in muscle size. The extent to which fiber hyperplasia might occur in muscles of humans who participate in strength training, however, remains controversial. Indirect evidence based on estimation of fiber numbers per motor unit suggests that some bodybuilders possess more muscle fibers than untrained subjects (Larsson & Tesch, 1986). This is in contrast with the results of Sale et al. (1987) that compared the size of the BB of untrained subjects with elite and intermediate-calibre bodybuilders. Muscle fiber numbers were estimated from the ratio between the measurements of total muscle area computed by tomography scanning and the average fibers area from needle biopsies. The data indicated that the average number of fibers did not differ significantly between groups (Figure 13.14c). Furthermore, in the only longitudinal investigation of hyperplasia in human subjects, McCall et al. (1996) observed no change in fiber numbers in BB of young men, following 12 weeks of intense strength training. More recently, however, it has been hypothesized by Kadi and colleagues (1999, 2005) that in addition to their role in the hypertrophy of existing muscle fibers, satellite cells may fuse to develop new muscle fibers or to repair injured segments of muscle fibers. For example, these authors have shown that,

contrary to untrained subjects, elite power lifter displayed small diameter fibers that expressed embryonic and neonatal myosin heavy chain compositions (MyHC), which are considered as markers for the early stages of muscle fiber development. Despite these interesting observations, the role of hyperplasia appears rather weak in human and therefore fiber hypertrophy remains the main mechanism of the increase in muscle mass following strength training.

Changes in Speed- and Power-Related Properties

In animals, studies have demonstrated that muscle contractile kinetics can adapt specifically to different frequencies of electrical stimulation. For example, a slow muscle that was chronically stimulated at high frequency became faster (Gorza et al., 1988), whereas a fast muscle stimulated at low frequency became slower (Pette & Vrbova, 1992). These results, obtained in rather extreme conditions, suggest that muscle contractile kinetics is susceptible to adapt specifically to the frequency of its activation. In human experiments, it has been reported that training with dynamic contractions (30–40% of maximum) increases the RFD of a maximal tetanic contraction to a greater extent than isometric contractions (31% vs. 18%; Duchateau & Hainaut, 1984). Furthermore, electrically evoked velocity of muscle shortening against no load, increased after dynamic training (21%), but not after isometric exercises.

The effect of training on the load–velocity relation displayed also specific adaptations with training (Duchateau & Hainaut, 1984). The increase in speed of shortening for light load is essentially related to the maximal speed of force development, whereas for heavy loads it is more closely related to the muscle maximal strength (Figure 13.15a and b). Although the moderate load for dynamic contractions training was chosen because it produces maximal power, peak power increase to a greater extent after isometric than after dynamic training (51% vs. 19%; Duchateau & Hainaut, 1984). When loads are expressed relative to the maximal force of the muscle before and after training, however,

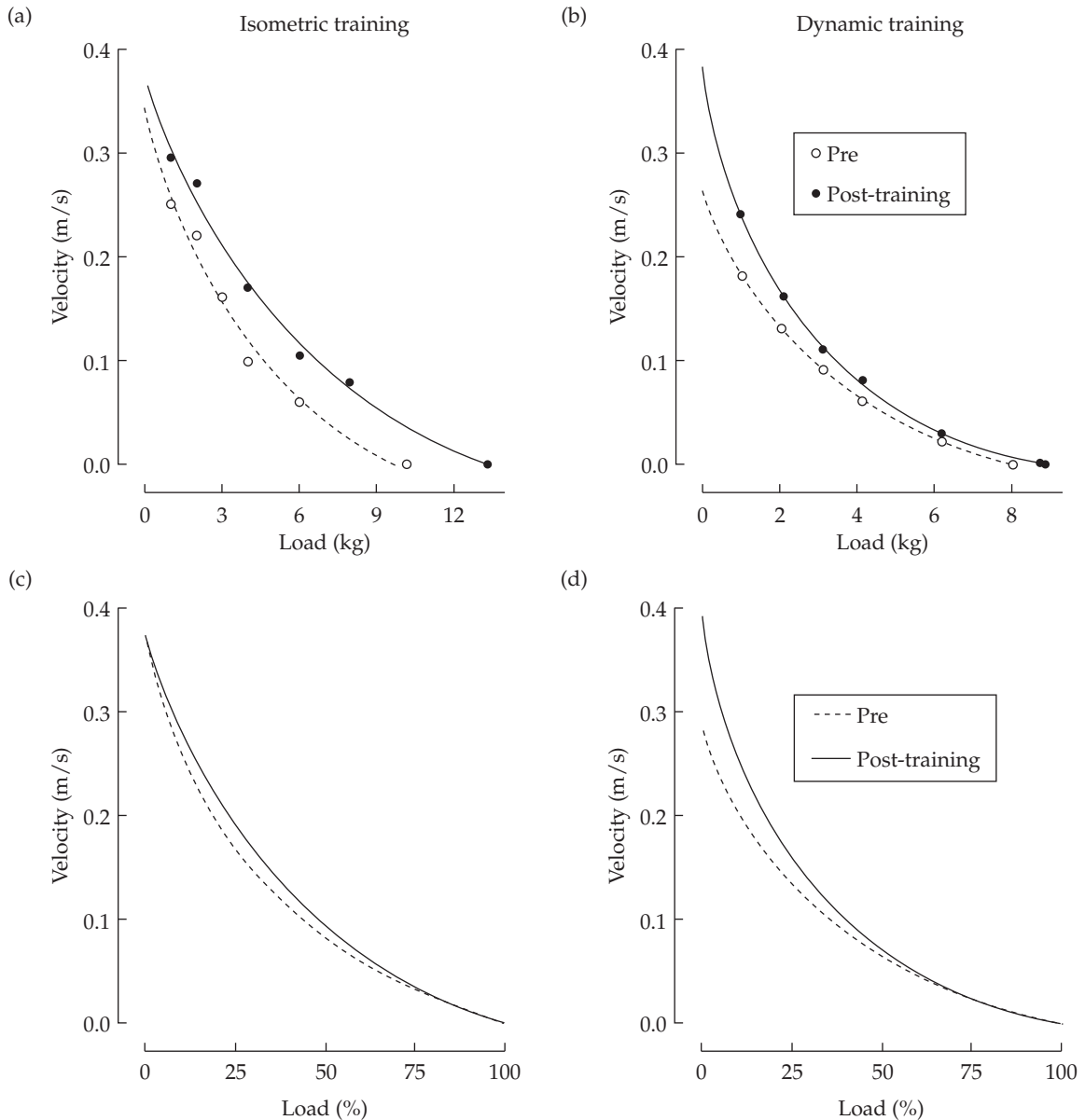


Figure 13.15 Effects of isometric and dynamic training on the load-velocity relation of the adductor pollicis muscle. Subjects trained during 3 months either by performing isometric MVC or dynamic shortening contraction, as fast as possible, against a load of 30–40% of maximum. During testing, muscle contraction against the different loads was induced by tetanic maximal electrical stimulation at 100 Hz frequency and plotted by Hill's equation. Loads are expressed in absolute value (a and b) and as percentage of the maximal tetanic force (c and d). The two training methods induce opposite muscular adaptations. Isometric contractions increase preferentially the velocity of muscle shortening against high loads (a) whereas dynamic contractions increase mainly the speed against light loads (b). As shown in (d), only training with dynamic contractions enhances the intrinsic contractile kinetics of the muscle (see text for explanation). (Data from Duchateau & Hainaut, 1984.)

only dynamic training shifted the peak power toward smaller loads (Duchateau & Hainaut, 2003). This observation is consistent with previous data showing that the contractile kinetics are intrinsically modified by chronic use of dynamic exercises and that the time-to-peak force of single motor unit decreases after such type of training in the adductor pollicis (Hainaut et al., 1981) and TA (Van Cutsem et al., 1998). Interestingly, when the F–V relation is normalized to the muscle maximal force, the curve obtained after training exhibited greater convexity due to higher velocity for loads below 50% of maximum compared with the curve obtained before training, whereas no difference occurs for isometric training (Figure 15c and d). Collectively, these results indicate that dynamic training induces intrinsic adaptation of the muscle contractile kinetics whereas the increase in speed after isometric contraction was due to the increase in maximal strength.

Mechanisms Underlying Changes in Speed- and Power-Related Properties

MyHC Composition

Today, it is possible to measure the mechanical properties of skinned fiber segments obtained by muscle biopsy from human VL muscle and to relate them to their respective MyHC composition (Larsson & Moss, 1993; Bottinelli et al., 1996; Harridge et al., 1996). With this technique, Trappe and colleagues (2000) observed that 12 weeks of strength training in elderly subjects increased the diameter of the MyHC-I and MyHC-IIa fibers (20% and 13%, respectively), and their peak tetanic force (55% and 25%), maximal shortening velocity (75% and 45%), and peak power (129% and 61%). In contrast, Widrick and colleagues (2002) found that the maximal shortening velocity of fiber segments did not change after strength training in young men, whereas there was a 30% increase in CSA for both MyHC-I and MyHC-IIa fibers, as well as 30% and 42% increases in peak power for MyHC-I and MyHC-IIa fibers, respectively. More recently, Malisoux and colleagues (2006) observed quite different results with similar measurements on fiber segments from the VL before and after 8 weeks of

plyometric training (jumping exercises). Training increased 1-RM load for the leg press by 12% and vertical jump height by 13%. MyHC-I and MyHC-IIa fibers displayed increases in CSA (23% and 22%), peak force (19% and 15%), maximal shortening velocity (18% and 29%), and peak power (25% and 34%). These results suggest that the increase in performance after strength or power training maybe related to specific changes in the mechanical contractile properties of the MyHC.

Myosin ATPase Activity

In the absence of a clear shift in the proportion of slow and fast fibers in the muscle after a strength or power training program (Staron et al., 1991; Hather et al., 1991; Adams et al., 1993; Hortobagyi et al., 1996; Canepari et al., 2005), the speed-related adaptation must be located in each muscle fiber. In addition to a change located in the mechanical properties of the MyHC, increase in maximal RFD after dynamic contractions can be related to changes in the chemical properties of the MyHC producing an enhancement of the myosin ATPase activity. It is known that the maximal speed of muscle shortening is closely related to the myosin ATPase activity (Bárány, 1967) and that high-velocity resistance training is associated with an increase of the myofibrillar ATPase activity (Bell et al., 1992). This is consistent with the recent results from Canepari and colleagues (2005) obtained from *in vitro* motility assays. These authors reported that 12 weeks of strength training increased the sliding speed of actin filaments on isolated myosin isoform IIa but not on myosin isoform I.

Calcium-Related Changes

An additional mechanism that could explain the increase in muscle speed-related properties is the enhancement of phasic ionized calcium movements. It has been shown in single barnacle muscle fibers that contraction time course changes are closely controlled by changes in phasic calcium movements (Duchateau & Hainaut, 1986). The finding that only dynamic training reduces twitch contraction time and half-relaxation time suggests that some factors, such as the quantity and/or the quality of SR

might be specifically enhanced by this type of exercise training. Interestingly, it has been reported that the SR can be qualitatively transformed by repetitive electrical activation in animal (Ramirez & Pette, 1974) and that strength training with heavy load (8-RM) increased Ca^{2+} reuptake by the SR and Ca^{2+} -ATPase activity in elderly but not in young women (Hunter et al., 1999). The hypothesis that dynamic training specifically changes calcium movements is consistent with the observation that contraction time maybe more dependent on Ca^{2+} release than on the myosin ATPase (Brody, 1976). Therefore, one could speculate that dynamic training induces larger increase in myosin ATPase activity and phasic calcium movements, compared with isometric exercises. Presumably, these adaptations should have contributed to the increase in RFD of twitch and tetanic contractions observed by Duchateau and Hainaut (1984) after dynamic training.

Changes in Force Transmission

Whereas adaptations in the force-generating capacity of muscle after training are well characterized, adaptation of the passive (tendon) and active (cross-bridges) structures of the SEC, and the cytoskeleton of the muscle that are responsible for force transmission within the contracting muscle are less understood.

Muscle–Tendon and Joint Stiffness

Adaptation of muscle contractile kinetics to exercise training could also be related to changes in the SEC of the MTC. An increase in the stiffness of the SEC maybe suitable for transmitting the force more effectively (Cavagna et al., 1981). Such a change in elastic characteristics will in turn enhance the performance by favoring the release of potential energy during SSC exercises since it shortens the time between the stretching and shortening phase (“coupling time;” Bosco et al., 1981; Ishikawa et al., 2006). In line with this hypothesis, it has been shown in pluri-articular movements, such as DJs, that preactivity of muscles increased after plyometric training. This change may have contributed to increase muscle–tendon stiffness at the time of the impact that likely produced higher stress on the

muscle–tendon (Kyröläinen et al., 1991; Kubo et al., 2006). In addition to changes in muscle activation, Pousson et al. (1990) reported an increased stiffness of the SEC, assessed by means of a quick-release technique, after 6 weeks of eccentric contractions of the elbow flexor muscles. This change was obtained regardless of the force produced by the elbow flexor muscles but was greater at low than at high force level. In contrast, using a sinusoidal perturbation technique, Cornu and colleagues (1997) observed a decrease in the slope of the stiffness–force relation in the plantar flexor muscles after plyometric training. To explain the apparent discrepancy between the two studies, the authors suggested that training can induce opposite changes in the passive structures of the SEC (tendon) and in the active structures (muscles around the joints) so that the change in stiffness of the whole musculo-articular system will depend of their relative adaptations.

Tendon–Aponeurosis Characteristics

Because the quick-release technique does not allow distinguishing between changes within either the contractile or tendon structures, US is now widely used to assess more specifically tendon–aponeurosis complex stiffness and to quantify its change following interventions. For example, it has been reported that tendon stiffness increased after a program of strength training with heavy loads not only in young (Kubo et al., 2001, 2006, 2007) but also in elderly (Reeves et al., 2003) individuals. Similar results have been obtained in young adults after training using eccentric actions (Duclay et al., 2009). The magnitude of tendon adaptation to training appears to be related to the contraction mode. Indeed, data from a same laboratory indicate that isometric training tends to induce a greater effect (58%; Kubo et al., 2001) than dynamic heavy load (30%; Kubo et al., 2006 and 29%; Kubo et al., 2007). In contrast, training with ballistic isometric contractions (Kubo et al., 2001) and plyometric exercises (Kubo et al., 2007) did not change tendon stiffness (Figure 13.16). Because joint stiffness is increased after plyometric training (Kubo et al., 2007), it is suggested that the greatest adaptations are located in the contractile than in the tendon structures. Furthermore,

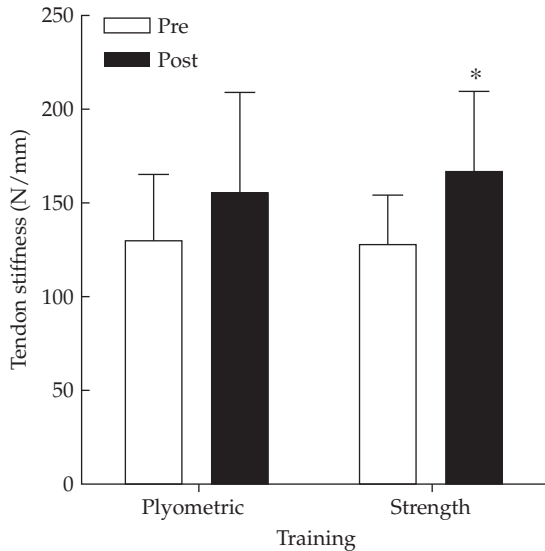


Figure 13.16 Effect of a 12-week plyometric (hopping and DJs) and strength (80% of 1-RM) training programs on tendon stiffness of the plantar flexor muscles. Although both training methods increased tendon stiffness, only values for strength training reached statistical significance (* $P < 0.05$). (Data from Kubo et al., 2007.)

in all these studies, the training protocol did not modify the size of the tendon, it was therefore suggested that training-induced changes in the internal structure of the tendon. This observation contrasts with the greater CSA (22%) of the AT observed in long distance runners compared with nonrunners (Magnusson & Kjaer, 2003). This apparent discrepancy maybe explained by the training mode (endurance vs. strength training) or by the delay of the hypertrophic response that develops only after a much longer period of training.

Lateral Force Transmission

A few years ago, it was hypothesized that training would increase the lateral force transmission to adjacent sarcomeres via intermediate filament system and the extracellular matrix via lateral connections to the endomysial connective tissue, contributing thereby to facilitate force transmission to the skeleton (Patel & Lieber, 1997). Recently, it has been shown that desmin, a cytoskeletal protein connecting myofibrils at their Z lines and to the

sarcolemma through costameres, may play a major role in lateral transmission of force from contracting sarcomeres to the muscle exterior (Bloch & Gonzales-Serratos, 2003; Ervasti, 2003). As an example of the potential for training to change desmin content within human muscles, its content in the VL was found to be enhanced after 14 days of downhill running (Feasson et al., 2002) and after only 4 weeks of an 8-week strength-training program (Woolstenhulme et al., 2006). This increase was not the result of an initial bout of strength training, but was clearly due to repeated bouts of exercise. Interestingly, this adaptation appeared before any muscle fiber hypertrophy could be detected. Therefore, this early adaptation was interpreted as a mechanism that could ensure that an adequate cytoskeletal structure was in place before or in conjunction with increases in the force-generating capacity of the muscle (Woolstenhulme et al., 2005). Furthermore, the increase in desmin appears to plateau after 4 weeks of training which suggests that desmin cytoskeleton adaptations could be mostly completed in the early stages of a strength training program. This early adaptation may result in part from the increase in the force developed by the muscle due to the early changes occurring within the nervous system. Although this study did not permit to differentiate the specific contribution of concentric and eccentric contractions to the increased desmin content during strength training, animal (Barash et al., 2002) and human (Feasson et al., 2002) studies indicated that eccentric contractions is a potent stimulus for the increase in desmin content.

Regardless of the exact mechanisms and locations of adaptation with training and their relative importance, it is clear that the structures associated with force transmission adapt to chronic loading of the MTC and contribute, in addition to muscle and voluntary activation, to increase the rate at which force can be developed.

Conclusions

As evoked in this chapter, a wide range of adaptations occurs in response to a program of strength or power training. Although the major determinant

of muscle strength is the force-generating capacity of the muscle, adaptations located in the nervous system and in the structures related to force transmission also contribute to the improvement in performance (Figure 13.7). This is particularly clear when a high level of power or RFD is needed such as during movements that involve SSC exercises.

Although compelling evidence exists for a significant role of the nervous system in training-induced increases in muscle strength and its rate of development, the specific mechanisms remain largely unknown. This deficit in our knowledge is mainly due to current technical limitations that impede the behavior of the whole motor unit pool of a muscle to be analyzed (Duchateau et al., 2006). However, and although changes in muscle coordination seem to play a major role in the increase in performance during the first sessions of a training program, very few studies have investigated changes in the distribution of muscle activity across multiple muscles with chronic loading. These adaptations and their specificity are particularly relevant for elite athletes who are trying to maximize their performance.

Today, the precise interaction between neural and muscle-tendon adaptations is far to be fully understood. There have been, however, some progress within the last decade on the underlying mechanisms of the increase in muscle mass (Kadi et al., 2005). Two main factors are at least necessary to trigger muscle hypertrophy, a production of IGF-1 and a high mechanical loading. The wide observation that neural adaptations precede muscular changes (Semmler & Enoka, 2000; Sale, 1988; Enoka, 2008) suggests that the greater neural input may contribute to increase the loading and training stimulus to which a muscle is exposed (Komi, 1986). For example, a greater neural activation can increase muscle stiffness thereby inducing a greater adaptation stimulus for the muscle-tendon component during plyometric exercise. Nonetheless, it is still not clear how a change in the pattern of the neural input may maximize further strength and power gains through specific morphological adaptations at the muscle and tendon levels. These questions are particularly relevant for SSC movements but require more focussed investigations.

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Chapter 14

Control and Training of Posture and Balance

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Introduction

Human stance and locomotion are basically related to the ability of balance and the control of posture. It is a great challenge to balance a CoG with a height of approximately 90–110 cm in adults over a small area of support. This can easily be seen when balance is impaired due to vestibular deterioration after accidents or due to selective loss of sensory feedback under pathological conditions. In order to meet the biomechanical requirements of stance and locomotion, it is the major task of the muscles encompassing the ankle, knee, and hip joints to provide adequate forces for appropriate joint stiffness. However, postural control is not only relevant to avoid loss of balance but also to provide a basis for the execution of other movements. Specifically, under dynamic conditions, when rapid movement changes are important and when disturbances from external sources (such as opponents in various sports disciplines) have to be compensated, a sound control of posture is highly relevant to allow the execution of goal-directed actions.

It has been shown that training of balance has a major impact to reduce incidence rates of joint injuries. Especially, sprain injuries at the ankle joint as well as cruciate ligament ruptures at the knee joint can effectively be reduced when subjects perform balance training in parallel or additive to their normal athletic training. Thus, training of balance

control seems to be beneficial to improve motor control in general so that intra- and intermuscular coordination avoids the occurrence of injuries in demanding situations in which the organism is prone to overload.

Postural Control

The term “postural control” describes the ability to balance the body over the base of support as well as providing a situation-specific body position to enable the execution of other tasks. A loss of balance often results from inadequate postural control, which in turn may lead to fall-related injuries. Generally, there are two modes of postural control: the “feedback mode,” which refers to the postural system aiming at reestablishing balance by compensatory reactions and the “anticipation mode” that functions when potential “disturbances” are cognitively anticipated in order to sustain postural control. The “anticipation mode” is controlled by a feed-forward process. It is characterized by its ability to foresee potentially destabilizing moments and sustains itself through self-initiated adequate countermovements. However, it should be noticed that postural control may not be clearly differentiated into either “feedback” or “anticipation” modes during certain movements but may rather be a hybrid from both. Therefore, the present chapter classifies postural control differently and distinguishes between motor control in “nondisturbed” and “externally influenced” postural tasks. The first step in understanding postural control is to identify the systems, which are responsible for

the perception of balance-related sensory information. Subsequently, the neurological centers in which this sensory information is processed are introduced. With this information, the last section of the present Chapter (The Effects of Sensorimotor Training) is composed to point out potential training influences on postural control.

The Organization of Undisturbed Stance

As compared to quadruped animals, humans have a more challenging task of bipedal stance. This is due to our uniquely high-positioned CoG when standing upright, the small stance supporting surface of our feet, and the low joint stiffness at the ankle (Loram & Lakie, 2002; Casadio et al., 2005). Humans are normally not aware of the demands for such a sensorimotor achievement. Only when “uncertainty” during stance is experienced, caused, for example, by an “unstable support surfaces” or a “degraded functional motor system” due to pathological processes, the complexity of postural control is revealed. Mechanically, the major task of the CNS is to avoid falls and thus to keep the body in balance over its supporting surface. The perception of unbalance through either optical (Buchanan & Horak, 1999), proprioceptive (Fitzpatrick & McCloskey, 1994), tactile (Holden et al., 1994; Lackner et al., 1999), or vestibular sensors (Nashner et al., 1989) is essential to secure adequate muscular activation in order to produce countermovements. The information received from various resources of the *analytical neurological system* is not separately processed but rather integrated and processed differently according to the situational needs. An example would be to stand straight maintaining balance in darkness or with the eyes closed. In this situation, the body is unable to utilize visual feedback and must rely on other sensual preceptors to complete the task. The task of standing undisturbed with closed eyes is then mainly organized by the somatic sensory system. This was shown in several experiments (Fitzpatrick & McCloskey, 1994; Winter et al., 1998; Peterka & Benolken, 1995). In one of them, Fitzpatrick et al. (1994) demonstrated that the control of upright stance is even possible after the exclusion of visual, vestibular, and cutaneous sensory information.

Based on these findings, there remain three candidates who maybe selected to achieve proper undisturbed stance conditions: groups Ia and II afferents from muscle spindles, as well as group Ib afferents from GTOs. The importance of these afferents for an organized balanced stance is also supported by findings from patients with polyneuropathy, in whom the degradation of proprioception was related to the extent of their body oscillations (Bergin et al., 1995). Initially, the Ia afferents were thought to be the most significant out of the three feedback modalities for balance control (Weiss & White, 1986; Griffin et al., 1990). Recent studies, however, have emphasized the relevance of the group II afferents (Nardone et al., 2000, 2001; Nardone & Schieppati, 2004). These studies have shown that patients with damaged Ia afferents display no adverse stance stability, whereas patients with stronger developed neuropathy (additional damage to smaller type-II afferents) stand more instable (Nardone et al., 2000; Nardone & Schieppati, 2004). Similarly, hemiparetic patients in whom the type-II afferents are also claimed to be impaired demonstrated reduced balance (Nardone et al., 2001). These observations underscore that stance is regulated and dominated by type-II afferents. However, as external perturbation takes place, other sources may get involved.

Postural Compensation When Stance Is Disturbed

To compensate balance disturbances, the CNS has to activate the muscles appropriately, according to disturbance stimuli and biomechanical conditions. It has been proposed that specific preprogrammed activation patterns would take action depending on the quality and quantity of the perturbation stimuli (Nashner & McCollum, 1985). However, due to the countless types of disturbance stimuli and the multitude of possible different compensation reactions, it is unlikely that such “automatic postural reactions” could be stored in the CNS as detailed movement trajectories (Schmidt & Lee, 1999). Alternatively, it has been proposed that postural actions may consist of constant ongoing feedback reactions (Park et al., 2004). Such an organization seems to be advantageous, because

a set of relatively simple reflex actions can be released and modulated according to the specific situation. In contrast to rigid, preprogrammed responses, the flexibly composed compensation reactions allow for an instantaneous and dynamic interaction between situation-specific needs and the corresponding feedback responses. Specifically, the higher centers of the CNS are responsible for context-specific adjustments of postural reactions (Horak et al., 1989). Depending on the actual situation and the specificity of the disturbance stimulus, information from participating sensory systems will be weighed differently. Due to the close interaction between the different sensory modalities, it is impossible to designate the relative contribution from every system (visual, vestibular, somatosensory). However, disturbance-specific preferences have been identified. For an undisturbed upright stance, the importance of the somatosensory system was already emphasized in the preceding paragraph. This system also exhibits a dominant role for compensating fast direction changes from the supporting surface (Dietz et al., 1988). However, not only the somatosensory system (Horak et al., 1990) but also the vestibular system has the potential to trigger and modulate motor balance reactions utilizing its sensory information (Boyle et al., 1992; Allum et al., 1998). The vestibular system is thereby ascribed to control the slower body oscillations beginning around 1 Hz (Mauritz & Dietz, 1980).

It not only makes sense to differentiate the disturbing stimuli into fast and slow perturbations but also to distinguish between rotational and translational surface displacements. The vestibular-spinal mechanisms in all probability play a major role when the support surface rotates (Allum & Pfaltz, 1985) whereas fast translational disturbances are processed more strongly by the somatosensory system (Dietz et al., 1988). A comparison of patients with different pathological impairments emphasizes the relevance of the somatosensory system compensating translational disturbances. In this situation, people with vestibular deficiencies reveal unaffected postural reactions (Horak et al., 1990), whereas patients with sensory neural pathology demonstrate delayed onset latencies (Inglis et al., 1994).

The organization of postural compensation reactions is not only dependent on the speed and type of perturbation (rotation or translation) but also relies on the supporting surface (Runge et al., 1999). On a solid and stable surface, the ankle strategy is predominantly utilized. In rough approximation, this would mean that the body acts as an inverted single-segmented pendulum. However, when the supporting surface is very small or fails to support, then the "hip strategy" is adopted. Here, the body resembles an inverted pendulum with two segments, which are connected together at the hip. Horak et al. (1990) proposed that an intact somatosensory system is necessary to conduct the "ankle strategy" as they noted a change to the "hip strategy" when the foot and ankle underwent ischemia. On the other hand, patients with vestibular loss were reported to use exclusively the ankle strategy. Thus, it can be assumed that the hip strategy relies on vestibular input (Horak et al., 1990).

The dependency of the type of reflex pattern on the extent of the mechanical perturbation has given rise to assume the existence of "load receptors" (Dietz et al., 1992). It has been shown that the body load influences the amplitude of the reflex responses independently from the stretch velocity. Based on these observations, it was concluded that there is a load-dependent receptor — probably the GTO. This load receptor has been described for the extensor muscles of the spinal cat during fictive locomotion (Conway et al., 1987; Duysens & Pearson, 1980), and it was shown that these receptor signals arise from GTOs before being mediated by Ib afferents to the spinal locomotor generator.

Motor Centers Responsible for Postural Control and Afferent Information Processing

In the preceding sections, the focus was primarily set on stimulus reception and the importance of visual, vestibular, tactile, and somatosensory systems with respect to postural control. In the following part, the processing of this sensory information is discussed. The relevant structures of the CNS with regard to balance control are introduced and it is illustrated how these structures affect motor commands.

Spinal Cord

The quickest and probably the simplest afferent information processing takes place in the spinal cord. Following a fast calf muscle stretch (e.g., slipping on a slick surface), the muscle spindles perceive information about the type of change of muscular length and release action potentials to the spinal cord via Ia afferents. The activation of homonymous alpha motor neurons then leads to a reflex response following around 40–50 ms after the muscle stretch (Gollhofer & Rapp, 1993). Although the stretch reflex is monosynaptic, i.e., only one synapse interposes afferent and efferent pathways, this does not exclude interaction of other parts of the neural system. For instance, during postural demanding situations, reduced reflex amplitudes can be observed, which most likely are caused by an increase in PSI (Taube et al., 2008b; Llewellyn et al., 1990; Hoffman & Kocaja, 1995). PSI is described as an inhibited release of transmitter at the synaptic cleft. In cases where demanding postural balance tasks lead to spinal reflex inhibition, the excitation of the Ia afferents is not fully transmitted to the postsynaptic neuron (the alpha motor neuron). This means that the presynaptic transmitter release is reduced without affecting the postsynaptic side, which is still susceptible to other inputs. During balance control, PSI therefore allows the reduction of spinal reflexes without affecting the input of supraspinal sites to the alpha motor neuron pool. This allows movements to be controlled less by reflexes, but rather by higher centers dictating muscular output. These higher centers are also responsible for modulating the magnitude of the PSI. In this respect, multiple brain structures such as the motor cortex (Meunier & Pierrot-Deseilligny, 1989), basal ganglia (Filloux, 1996), and cerebellum (Dontsova & Shkivskaia, 1980) are capable of modulating context-specific spinal reflexes.

Brain Stem

The spinal cord connects to the brain stem, which is composed of the medulla oblongata, pons, and midbrain. Due to earlier experiments involving animals, it has been deduced that this area of the brain plays an important role in regulating balance.

At the beginning of the 20th century, research showed that mammals were able to compensate postural disturbances, utilizing reflex mechanisms involving the spinal cord and brain stem even if the connections to higher neural centers were dissected (Magnus, 1924; Sherrington, 1906). The reticular formation, which passes through the medulla oblongata, pons, and midbrain, plays an important role in controlling balance. In this area of the brain, information from the vestibular apparatus, the proprioceptive, and the visual systems converge and can be integrated into cortical motor commands. Luccarini et al. (1990), for instance, showed that postural balance adjustments could be suppressed by inhibiting the activity of the brain stem. Preventing the development of neural projections from the brain stem to the spinal cord consequently impairs the development of motor skills and postural control mechanisms (Vinay et al., 2005).

Cerebellum

The importance of the cerebellum for postural control could only first be proven through studies involving patients with cerebral defects. By 1891, Luciani described the following consequences from cerebral lesions: atony (loss of muscular strength), asthenia (muscle exhaustion), astasia (inability to stand), and dysmetria (movement disturbances, from either overshooting or undershooting goal-directed movements) (Luciani, 1891). Later, the description of the symptoms would be expanded and the role of the cerebellum for adjusting agonist/antagonist movements, as well as coordinating ranked movements was discovered to be important for motor control (Diener & Dichgans, 1992). According to issues with balance, the cerebellum seems to be crucial for the selection and memorization of appropriate compensation reactions in the specific disturbance situation. Patients with cerebral lesions are often unable to alter their movement pattern when the situation is changing. Unlike healthy people, they cannot adapt the amplitudes of their reflex responses ("LLR" with a latency of around 120 ms) situation specifically (Nashner, 1976). Morton and Bastian (2007) further inferred by observation that a healthy person's

cerebellum “learns” from previous movement mistakes, thereby developing a different form of postural reaction by adjusting feed-forward control.

Basal Ganglia

Postural impairment is also observable following basal ganglia lesions. A dysfunctional substantia nigra, as for example in Parkinson patients, is commonly associated with insecure stance, numerous falls, and gait disabilities (Bloem et al., 2004; Bloem & Roos, 1995). Visser and Bloem (2005) claim that the basal ganglia facilitate postural control through two means: one being *postural flexibility* and the other *sensorimotor integration*. Postural flexibility is the ability to constantly adapt to environmental changes. Similar to the effects after cerebral lesions, damage of the basal ganglia leads to the inability to adapt muscular responses to specific disturbance stimuli (Bloem & Bathia, 2004). Moreover, during dual tasks incorporating cognitive and motor demands, basal ganglia patients are unable to prioritize the task execution. Instead of placing retention of balance as the highest priority, they attempt to accomplish all tasks simultaneously, increasing the risk of falls (Bloem et al., 2001).

Apart from the loss of postural flexibility — or the ability to flexibly weigh resources — damage to basal ganglia accompanies an impairment to integrate sensorimotor information. When Parkinson patients are asked to close their eyes and then reposition their arm in the same way as their other arm, which has been passively or actively moved, they are often unable to do so (Klockgether et al., 1995; Zia et al., 2000). The dysfunctional capability of integrating sensorimotor information could possibly explain why Parkinson patients fail to realize their own body oscillations, and are often surprised when they see themselves oscillating in front of a mirror (Visser & Bloem, 2005).

Motor Cortex

Initially, the motor cortex was not considered to play a major role in the maintenance of balance. However, recent studies present experimental data which highlight its relevance with respect to

postural reactions. Many of these studies are summarized in two recently published review articles (Jacobs & Horak, 2007; Taube et al., 2008a). In rabbits and cats, the role of the motor cortex in postural regulation was demonstrated by a correlation of cortical and muscular activity during balance reactions while directly recording electrical physiological currents from their cortical neurons (Beloozerova et al., 2003, 2005). With humans, evidence of cortical involvement during balance reactions was provided using noninvasive electrophysiological techniques such as TMS, positron emission topography (PET), or electroencephalography (EEG) (Ouchi et al., 1999; Jacobs & Horak, 2007). For instance, during walking, the CNS is responsible for balancing numerous dynamic body segments with a high CoG over a small base of support. During such tasks, TMS made it possible to demonstrate that despite highly automated movements, control was still relying on by cortical centers (Nielsen, 2003).

However, cortical involvement is not confined to walking. Cortical control was also apparent following rapid perturbations during upright bipedal stance resulting in so-called “transcortical long-loop reflexes” of the lower extremity muscles with latencies around 85–100 ms (Taube et al., 2006). When the surface underneath a person is taken away, such as when abrupt accelerations or decelerations are experienced in buses or trams, these events are perceived by the muscle spindles of the calf muscles. Under lab conditions, these events are investigated by quick and unpredicted fast forward or backward displacements while the subjects are in upright stance. When the length change of the muscle is sufficiently fast, a short-latency stretch reflex (occurring after 40–50 ms) is evident. The perception of stretch does not only trigger the SLR but is also transmitted to higher centers where it meets the sensory cortex following approximately 50 ms. After a short central processing (~10 ms), a motor command from the motor cortex to the muscles is sent and there arrives an additional 30–35 ms later (Taube et al., 2006; Petersen et al., 1998). The cortical compensation response following a balance disturbance appears later (around 85–100 ms) as the response generated at the spinal level (40–50 ms;

SLR). However, the cortical responses are better adaptable to the specific situation.

It appears plausible that when people find themselves in situations where they may potentially lose their balance, cortical structures are conceptually producing counteractive measures. However, in recent studies, it was further shown that the motor cortex is also active in stable situations like during undisturbed two-legged stance (Tokuno et al., 2009; Soto et al., 2006). In this situation, no correlation between the modulation of motor cortex excitability and the observed body oscillations could be demonstrated (Tokuno et al., 2009). This could mean that the motor cortex is either shifted to a readiness position in order to quickly intervene in case of lost balance or that other noncortical areas of the brain (i.e., cerebellum) are dependent on (tonic) motor cortex output to become self-active.

The Effects of Sensorimotor Training (Balance Training)

The ability to maintain balance is subject to strong fluctuations during life span. Small children need approximately 1 year in order to stand upright or to independently learn how to walk. Children then require at least 7 years until they have developed similar balance strategies as adults. Throughout the course of maturation, postural control continuously improves before it starts to get impaired with increased age. Age-related balancing ability is, however, not solely influenced by age but is strongly affected by the activity level of an individual. Studies demonstrated that balance is trainable in nearly every period of life (Granacher et al., 2006). In the following, training interventions to improve balance will be presented and the corresponding neural adaptations will be discussed. This chapter concentrates on the adaptations following sensorimotor training (SMT, balance training) that are related to improvements in strength and power as well as in jump abilities.

SMT Sequences

Training content and exercises can strongly vary from SMT to SMT. Currently, there exist no

guidelines about the optimal duration and intensity of these exercises. In contrast to the highly differentiated protocols for strength and endurance training, the influence of intensity or load- or volume-dependent adaptations have not been investigated yet. Generally, the majority of training interventions are based upon one- or two-legged balancing on varying undergrounds. A typical training session is characterized by several sets of 20 s one-legged balance, both executed with eyes open and with eyes closed. The sets are usually separated by 40 s pause. In most of the intervention studies, the training requirements are weekly adapted and the complexity of the balance task or the stabilization criteria are stepwise enhanced. The equipment consists of wobble/tilting boards, spinning tops, mini trampoline, soft mats, and cushions as well as other unstable undergrounds. Figure 14.1 illustrates a standardized training program, which was used in several studies. Most studies evaluating neural physiological adaptations in response to SMT are based on such a training program.

In the literature, the effects of SMT on the incidence rate of ankle injuries have been intensively investigated. Several reports found remarkable reductions in ankle injuries after application of balance training in parallel to normal athletic exercises. In a recent research work, Hupperets et al. (2009) investigated the self-reported incidence rates on the basis of a controlled intervention trial with a 1-year follow-up. They specifically found a significant lower risk in the intervention group in those athletes in whom the sprain was not treated properly.

Sensorimotor training has also been applied to specifically address the knee joint stability. Gruber et al. (2006) investigated the effects of an SMT performed either barefooted or with a ski boot to fix the ankle joint complex. In their experiments, the knee joint stability was investigated before and after 6 weeks of SMT. They found that in both training groups the improvements in knee stability, determined as the displacement of the tibia in relation to the patella following a standardized anterior drawer, were associated with training-induced improvements of the hamstring reflex activation. In those experimental situations in which slow






Instruction	"Stand as quiet as possible. Avoid falling off the training device."	
Standardized training position	Exercises are performed with the knee slightly bent (~ 30°), barefoot, eyes opened, and with hands akimbo.	
Exercises	 ①	Each exercise (1–4) consists of four sets which where performed for 20s with a 40s rest between each set. A longer rest of 3 min. is allowed between different devices in order to avoid fatigue.
	Soft mat	
	 ②	
	Ankle disk	
	 ③	
	Balance board	
	 ④	
	Air cushion	

Figure 14.1 Exemplary balance training protocol. Exercises are performed in one-legged stance on different training devices. The total duration of each training session is 60 min incorporating a 10 min warm-up and a 10 min cool-down. (Modified from Gruber et al., 2007a).

mechanical displacements were applied, the tibial displacement was reduced about 30% and the stiffness of the knee joint was enhanced about 56% in the ski boot group. At the same time, the activity of the hamstrings following SMT increased about 37%. No changes were found for the groups that trained barefooted.

Neural Adaptations to SMT

The current published training studies show that adaptations after SMT are very context specific:

reflex responses as well as descending volleys from the motor cortex (MEPs) following SMT are not always facilitated or not always inhibited. This is strongly dependent upon the situation in which the reflexes/MEPs are provoked. The organism seems to learn when responses are functionally relevant and if so these responses are facilitated. On the contrary, when the responses are counterproductive, the CNS will learn to inhibit them.

Spinal Adaptations Associated With SMT

The CNS is capable of regulating the reflex activity situation specifically. Generally, spinal reflexes are strongly and distinctively inhibited when the demands of the postural condition are enhanced. This was shown from Llewellyn and colleagues (1990) who conducted reflex measurements from subjects who either walked on a balance beam or on the floor. On the beam, the electrically evoked reflexes (H-reflexes) were inhibited. Similarly, reflex actions were inhibited when subjects were prompted to close their eyes during upright stance (Hoffman & Kocaja, 1995; Earles et al., 2000). It is important to note that reflexes are not only inhibited as postural complexity increases, but they are also facilitated once postural demands become less challenging, for instance by additional mechanical support (Tokuno et al., 2009; Katz et al., 1988) or augmented visual feedback (Taube et al., 2008b).

Apart from these instantaneous reflex adaptations, reflexes can adapt in response to long(er) lasting training. After several weeks of SMT, a reduction of spinal reflex function was observed in several studies (Gruber et al., 2007b; Taube et al., 2007a,b). Functionally, inhibited spinal reflexes following SMT (during postural demanding situations) may serve to counteract reflex-induced joint oscillations (Aagaard et al., 2002b; Llewellyn et al., 1990). Furthermore, the suppression of spinal reflexes may transfer the control of muscular activation from primary spinal to more supraspinal centers. This could be beneficial in terms of an improved movement control (Solopova et al., 2003).

As previously mentioned, not all reflex contributions are reduced following SMT. In cases where the reflex response plays a functional relevant role,

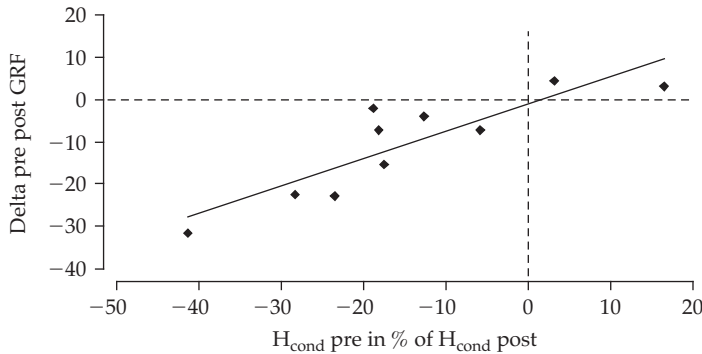


Figure 14.2 Balance training–related differences of pre- and postmeasurement of the conditioned H-reflex (H_{cond} ; indicating cortical excitability) were illustrated on the abscissa while changes in the vertical peak GRF were displayed on the ordinate. The regression line clearly demonstrates that greater improvements in stance stability (reduced GRFs) were accompanied by greater reductions in the facilitation of H_{cond} (greater reduction of cortical excitability). (Adapted from Taube et al., 2007a).

training can enhance this contribution. Granacher et al. (2006) conducted a 13-week SMT with elderly people and analyzed balance reactions from the participants following progressive gait perturbations. After the training, the seniors showed an increase in compensatory reflex contributions in line with an improved postural control.

Supraspinal Adaptations to SMT

Horak and colleagues (1989) demonstrated that previous experience and knowledge of the postural task influenced the compensatory reaction. When subjects expected a greater perturbation as actually took place, they overresponded. The opposite was true when subjects underestimated the perturbation. Based on this observation, it was concluded that both peripheral and central mechanisms are participating during compensatory reactions and that their relative contribution can be modulated by previous experience. Two decades later, electrophysiological methods provided evidence that there exists a correlation between improved balance control and cortical plasticity (Beck et al., 2007; Schubert et al., 2008; Taube et al., 2007a). The subjects in all three studies exhibited a reduced cortical activity after SMT when measured during a postural task. Furthermore, subjects who reduced their cortical contribution the most showed the greatest improvement in balance ability (Taube et al., 2007a) (Figure 14.2).

All studies assessing supraspinal adaptations following SMT have shown a reduction in cortical excitability. At the same time, the spinal

contributions were either reduced (Taube et al., 2007a) or unchanged (Beck et al., 2007; Schubert et al., 2008) while overall muscular activity remained the same. Therefore, it was assumed that potentially subcortical centers increased their participation of movement control (see Figure 14.3) (Taube et al., 2008a). This might be seen as a mechanism to be more independent from voluntary control and to make motor control more automatic. As balance reactions become more automated, it was proposed that the cerebellum and basal ganglia play progressively a more important function in movement control, similar to observations regarding the acquirement of fine motor tasks (Puttemans et al., 2005). The importance of the cerebellum and basal ganglia for balance organization, and also their impact on the choice of the adequate compensation reaction, was already described.

SMT, Strength, and Power

Recent studies have shown that balance exercises can improve strength, power, and even jumping abilities (Gruber & Gollhofer, 2004; Granacher et al., 2006; Taube et al., 2007b; Bruhn et al., 2004; Heitkamp et al., 2001; Gruber et al., 2007a). Growing interest has been brought up in the literature about the functional importance of quick and efficient activation of the lower leg muscles. Theories have been established favoring that in injury-related situations, a fast “access” to the muscles maybe beneficial in order to stiffen joint complexes properly before mechanical perturbation (Konradsen et al., 1998; Alt et al., 1999;

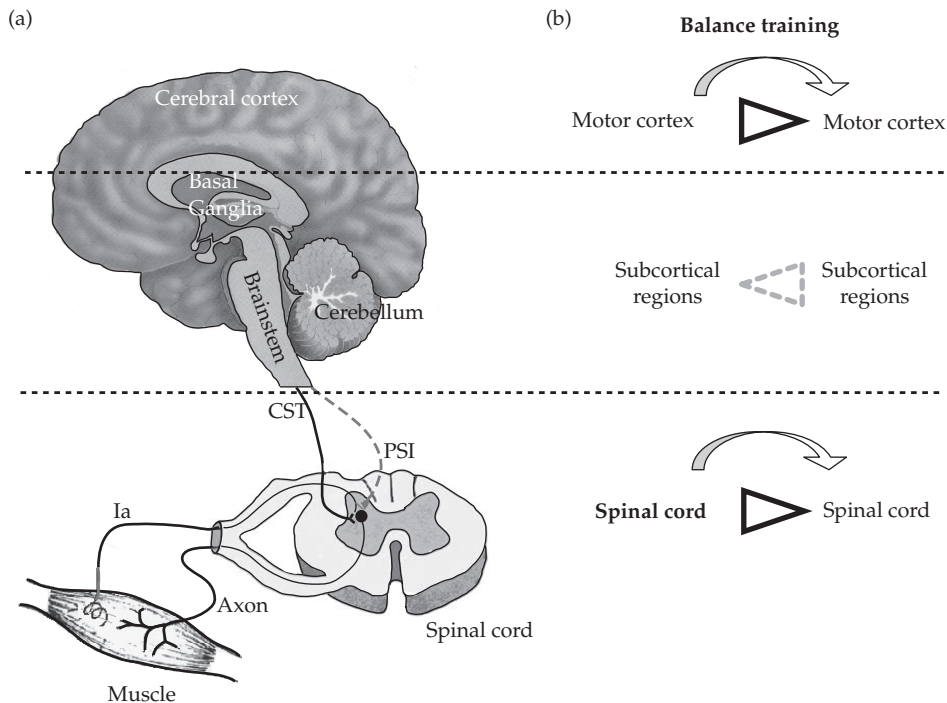


Figure 14.3 Simplified depiction of balance training-induced adaptations assessed during postural tasks. (a) Structures of the nervous system, which are considered to play an important role in the maintenance and recovery of balance. Sensory information from visual, vestibular, cutaneous, and proprioceptive sources is integrated into postural control. With respect to the somatosensory system, changes in muscle length after perturbation are signaled by Ia and II (not illustrated) afferents originating from the muscle spindle. This information is transmitted to the spinal cord and supraspinal centers (the latter connection is not displayed). The early part of the compensatory response (the SLR and the MLR) is processed at the spinal level. After approximately 90–100 ms, there is sufficient time for supraspinal sources to control muscular output (the LLR), e.g., via the corticospinal tract (CST) emanating from the motor cortex. (b) Neither the spinal nor the supraspinal structures generate stereotypical compensatory reactions after postural disturbances. Prior experience (e.g., balance training) or anticipation of the destabilizing stimulus as well as the context-specific intention can lead to changes in the postural response. Balance training is thought to reduce spinal reflex excitability by increasing the supraspinal-induced presynaptic inhibition (PSI). This reduction (\blacktriangleright) in spinal cord contribution after balance training is schematically illustrated in the bottom line of Figure 14.5. Another well-documented adaptation after balance training is the reduction in cortical involvement (first line of Figure 14.5). It is therefore assumed that training of balance skills and improved postural control after balance training strongly relies on subcortical structures (this indirect evidence of enhanced importance is indicated as a gray dotted triangle (\triangleleft) in the middle line of Figure 14.5). (Adapted from Taube et al., 2008a).

Bloem et al., 2000). From a functional point of view, it has been argued that the vast reductions in ankle and knee joint injuries following SMT might be explained by two mechanisms: (1) improved neuromuscular coordination as described in the previous chapter; (2) enhanced ability to activate joint muscles in order to achieve a quick joint stiffness in situations when unexpected perturbations are needed to be compensated.

Strength and Power Adaptations to SMT

It has been shown that an increase in force development is closely related to improvements in neural drive of the trained muscles, especially in a dynamic, explosive type of strength training (Hakkinen et al., 1985). More recently, Van Cutsem et al. (1998) have demonstrated that neural adaptations caused by an explosive type of training

are primarily responsible for an increased RFD. By analyzing single motor unit recordings, the authors were able to demonstrate the preservation of the orderly motor unit recruitment pattern. However, after training, motor units were activated earlier and showed increased firing frequencies. From intramuscular EMG recordings, the authors support the idea that explosive type of training is associated with high-frequency discharges ("doublets") occurring at the onset of muscular action. This might also be the case after SMT (Gruber & Gollhofer, 2004).

Improvements in strength capacity can either be achieved by enhanced muscle protein mass (Staron et al., 1990, 1991; Narici et al., 1996) or by improvements in the neuromuscular control of the muscle (Moritani, 1993; Narici et al., 1996; Aagaard et al., 2002a). While maximum voluntary strength clearly correlates with the CSA of the muscle, the RFD is largely dependent on the discharge rate of the muscle units recruited (Van Cutsem et al., 1998), to the modulations of the recruitment characteristics (Kukulka & Clamann, 1981) or to a combination of both (Duchateau & Hainaut, 2003).

Following a 4-week SMT, an improvement of 33% of the RFD was achieved in an isometric leg press exercise (Gruber et al., 2007a). These observations are remarkable as during SMT the muscles are activated with only submaximal intensities to secure balance. In their conclusion, the authors claimed that afferent contributions might play an important role for the observed adaptations in explosive strength. Examining the recruitment thresholds and the firing rates of single Motor Units (MUs), Grande and Cafarelli (2003) reported that MUs of m. VL were recruited earlier but with lower firing rates when activated via the Ia afferent pathway compared to a voluntary activation. This clearly indicates that recruitment thresholds and firing rates of MUs depend on the amount of afferent input.

In the study of Gruber et al. (2007a), the adaptive responses in the RFD after an SMT and after ballistic strength training were compared. Following a 4-week training, both training modalities caused increased RFD without drastically changed maximum voluntary forces. Concurrently, they found that the mean amplitude of the superficial EMG

was more increased in the ballistic type of training as compared to the SMT (Figure 14.4a). However, analyzing the median frequency of the GAST and SOL EMG they found that especially after SMT both muscles showed increased median frequencies, whereas after ballistic voluntary training only GAST frequency was increased. Based on the observation that the mean activation amplitudes were significantly higher in the ballistic training group, the authors suggested that different mechanisms of activation are likely to be associated with the training type, which might at least in part explain the observed differences in RFD.

From a practical point of view, these adaptations can be taken to suggest that balance training could be used complementarily to ballistic strength training. Especially for athletes who need contractile explosive strength properties together with precise movement control, this type of combination might be beneficial.

Reactive Jump Abilities and SMT

In few papers, the effects of SMT on individual jump abilities were investigated (Bruhn et al., 2006; Taube et al., 2007b). Both in normal as well as in highly trained athletes, the jump performance was enhanced after a 6-week SMT. In both studies, the training effects have been compared to classical strength training. Both types of training produced similar improvements in the maximum jump height and SJ, CMJ, or DJ conditions (Figure 14.4b). However, analyzing the neuromuscular activation differences between both training regimens could be observed. In the paper published by Taube et al. (2007b), the neuromuscular activation was verified by a thorough investigation of the spinal excitability by peripheral nerve stimulation. They stated that the improvement observed after classical strength training relies on the ability to more intensively activate the motor neurons pool as the background EMG activity was enhanced after training. After SMT, a reduction of the spinal excitability was observed. The authors concluded that the diminished spinal excitability maybe important to prevent reflex-mediated joint oscillations. However, the authors also admitted that these observations

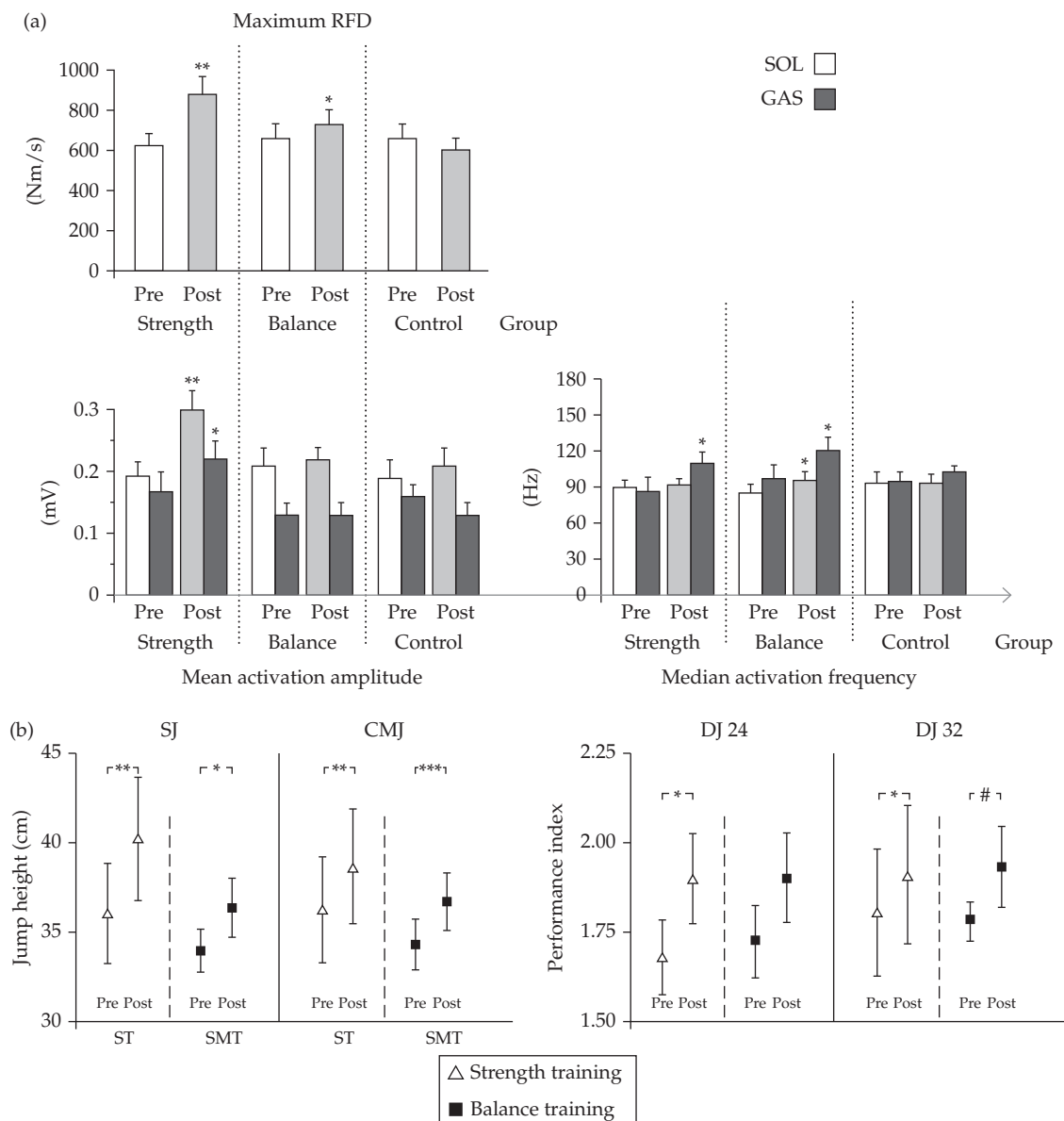


Figure 14.4 (a) Maximum rate of force development(RFD), mean activation amplitude, and median activation frequency before and after a 4-week training intervention of either strength or balance training. Values of the controls are also given. Mean activation amplitude and median activation frequency were determined within the time phase of -30 to 170 ms with respect to the onset of the isometric contraction. (* $P < 0.05$; ** $P < 0.01$). (Adapted from Gruber et al., 2007a). (b) Mean (\pm SD) jump height in squat (SJ) and countermovement (CMJ) before and after a 6-week strength or balance training (left) and mean (\pm SD) performance index observed in both drop jump (DJ) conditions (24 cm [DJ 24] and 32 cm [DJ 32] dropping height). Performance index was calculated as $PI = \text{Jump height} \times (\text{contact time})^{-1}$. (* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$). SOL, soleus; GAS, gastrocnemius. (Adapted from Taube et al., 2007b).

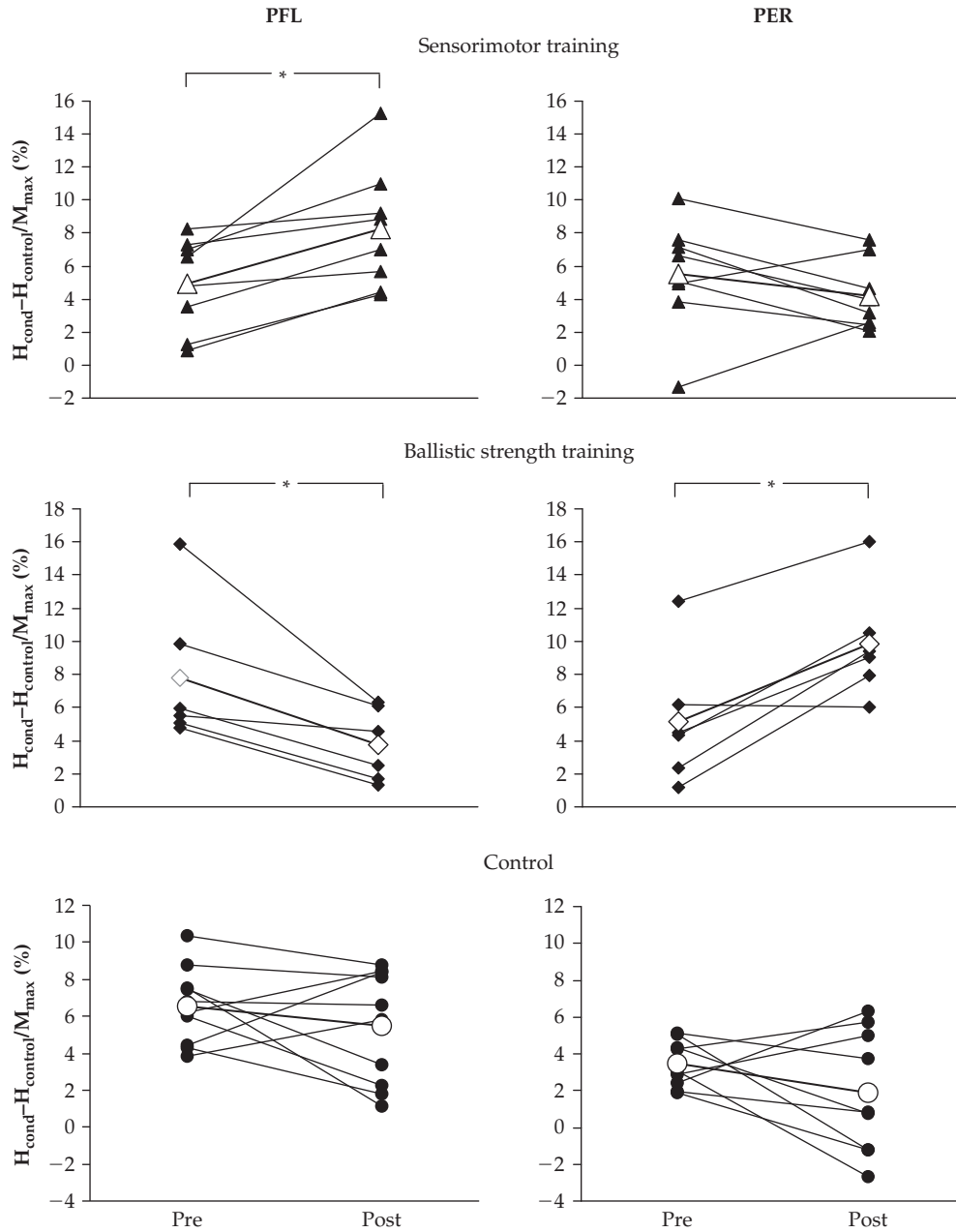


Figure 14.5 Effect of training on the short-latency facilitation (“cortical contribution”) during a voluntary task of plantar flexion (PFL; sitting condition, left) and a postural perturbation (PER; standing on treadmill, right). The “cortical contribution” was compared before and after an SMT (top) or a ballistic strength training (middle) or no training (bottom, control) (* $P < 0.05$, Wilcoxon signed-rank test). Adapted from Schubert et al. (2008).

can hardly explain the training-induced jump performances.

Specificity on Neuromuscular Adaptation as a Function of the Training Stimuli

The neuromuscular adaptations responsible for this enhanced strength and jumping abilities after balance training are not entirely understood. Most of the above-mentioned studies did not demonstrate significant changes in the surface EMG. Only in one study, increased EMG activity of the agonist was observed during explosive isometric contractions (Gruber & Gollhofer, 2004), while in another study solely the median frequencies of the EMG power spectrum were increased (Gruber et al., 2007a). Both of these studies could not clarify whether the modified neural drive was derived from spinal or supraspinal centers. Recent results suggest that primarily supraspinal centers contributed more strongly to explosive force production following balance training (Schubert et al., 2008). Interestingly, SMT-trained people showed increased cortical excitability when tested during strength/power exercises. However, when tested during a postural task, they exhibited reduced cortical excitability (Schubert et al., 2008) (Figure 14.5).

The enhancement of explosive strength following SMT might therefore be traced to an improved cortical activation of the target muscle(s). It is speculated that balance training strengthens synaptic neural connections responsible for the innervation of the muscles encompassing the ankle joint and this might contribute to an increased explosive strength capability of these muscles (Taube et al., 2008a).

Conclusion

Maintenance of balance is a very complex skill which requires integration of sensory information from many different sources as well as processing on all levels of the CNS. Training of postural control can improve motor performance with respect to balance, strength, and jumping abilities. This is not only relevant for elderly adults and patients but also for athletes. The underlying neural adaptations were shown to occur at different sites of the CNS. Thereby, spinal, corticospinal, and cortical plasticity proved to be highly task specific. So far, little is known about the plasticity of cerebellar and subcortical areas with regard to balance training due to the difficult accessibility of these structures.

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Chapter 15

Sport Performance in Master Athletes: Age-Associated Changes and Underlying Neuromuscular Factors

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Introduction

Aging is characterized by decreased capacity of the neuromuscular system to produce maximal strength and power. The age-related deterioration in performance can be attributed to both the aging processes per se and the lifestyle factors such as declines in the amount and intensity of physical activity. However, the changes in physical activity among the aging population in the developed world are not unequivocal. At the same time, as the proportion of inactive people is increasing, so too are the numbers of highly active individuals who continue training and participating in competitive sports well into old age.

Master athletes are competitors who exceed the minimum age specific to their sport and who participate in international, national, or local competitions for “masters,” “veterans,” or “seniors.” The minimum age is typically 35 or 40 in sports such as track and field, cycling, orienteering, cross-country skiing, and weight lifting, but it is often lesser (30 or only 25) in sports such as tennis and swimming, where top performance can be achieved already in teenage (Spirduso et al., 2005). In general, masters’

competitions are divided into 5-year age categories, with the oldest athletes often being more than 90 years old.

Conducting masters’ competitions is a relatively new phenomenon in the world of athletes. In some sport events, such as orienteering and cross-country skiing in Finland, older age categories were included in local competitions and military sports as early as in the 1930s; although the organizing of national and international championships specifically for masters did not begin until the 1960s and 1970s (Olson, 2001). For example, the first Annual US Masters Track and Field Championships were held in 1968, while the first official Finnish veterans championships in cross-country skiing and in track and field were held in 1972 and 1977, respectively. The World Masters Track and Field Championships have been organized biennially since 1975 and the corresponding European Championships since 1978. During the last 20 years, thousands of competitors from different countries have participated in these games. Currently, most sports including both outdoor and indoor events and a variety of ball games have their own national and international masters’ competitions.

For researchers and scientists in the field of exercise physiology, biomechanics, and gerontology, master athletes provide an interesting population to study. Although elite athletes—females in particular—continue to represent a small proportion of their cohort, individuals with long-term

devotion to physical training offer an economical means of investigating the role of exercise in the prevention of age-related decrements in physiological capacities and function. Highly motivated athletes provide official and controlled physical performance data, and they offer a barometer of what is possible in physical aging (Harridge & Suominen, 2003; Spirduso et al., 2005). Ideally, master athletes with optimized living habits could provide a human research model of "primary" or "inherent" aging, where age-related physiological changes are less influenced by confounding factors such as sedentary lifestyle and chronic diseases (Maharam et al., 1999; Hawkins et al., 2003; Tanaka & Seals, 2003; Rittweger et al., 2004; Lazarus & Harridge, 2007; Korhonen, 2009).

Regardless of training, athletic performance and associated physiological functions decline with aging. This is shown by cross-sectional and longitudinal studies, the majority of which have been conducted among endurance athletes (Wilson & Tanaka, 2000; Tanaka & Seals, 2008; Reaburn & Dascombe, 2008). Endurance-trained athletes maintain an excellent maximal oxygen uptake and muscle oxidative capacity, but the slope of the age-related decline in aerobic capacity and in muscle mass and performance resembles that in untrained persons (Klitgaard et al., 1990; Harridge et al., 1997; Tanaka & Seals, 2008). However, this decline cannot be solely attributed to aging, as these athletes also reduce their training intensity and volume as they get older (Trappe, 2001; Tanaka & Seals, 2008). On the other hand, the age-related decline of aerobic capacity in controls maybe biased in that the subjects tested in the oldest age groups probably represent individuals with better health and fitness than the average sedentary population. It is also noteworthy that the relative difference in aerobic capacity between endurance athletes and nonathletes is actually greater with aging, where the slopes of the decline are similar. There is less literature on the age-related decline in performance or the factors that contribute to this decline among strength- and speed-trained master athletes, but it is possible that the critical age-related changes could be further diminished by continuing high-intensity resistance or sprint training (Korhonen, 2009; Reaburn & Dascombe, 2009).

The purpose of this chapter is to describe the age-associated changes in sports performance and underlying neuromuscular factors in master athletes. Special attention is paid to track and field events where strength, speed, and power are essential elements of performance.

Record Performances

Since Moore (1975), an increasing number of journal articles and book chapters have highlighted the value of athletics records in providing an insight into aging and performance (Stones & Kozma, 1981; Conzelmann, 1997; Maharam et al., 1999; Harridge & Suominen, 2003; Korhonen et al., 2003; Spirduso et al., 2005; Wright & Perricelli, 2008; Korhonen, 2009; Reaburn & Dascombe, 2009). Official record performances in organized masters' sports such as track and field and weight lifting provide a valuable database for describing maximal physical performance throughout the life span and comparing age-related changes in athletic events imposing different demands on the neuromuscular system.

The more highly trained competitors there are in masters' athletic events, the greater should be the degree to which records approach the upper limits of performance in the different age groups. So far, track athletic events, such as the 100 m sprint, provide perhaps the best examples of masters' sports with a need of high power output, where a great many athletes regularly compete at a high international level. The current world records in the 100 m sprint (Figure 15.1) show that running time increases in an almost linear manner until approximately 80 years of age in men and 75 years of age in women (0.87% and 1.24% per year, respectively), whereupon the exponential decline in performance is more evident. In terms of running speed (m/s), the corresponding figures are smaller (0.58% and 0.74% per year), but nevertheless indicate that even elite athletes show distinct deterioration of maximal performance with increasing age. However, it is clear that the older champions represent a cohort that has never performed as well as their present-day young counterparts (Lazarus & Harridge, 2007). In fact, longitudinal data, such as the individual-best 100 m performance times of the present

world record holder Merlene Ottey, show a smaller decline in running time (0.56% per year) and speed (0.53% per year) between age 36 and 46 and no decline at all when using 20 years of age as the baseline. Similarly, the individual records of Jorma Manninen, the Finnish men's record holder in the

age categories of 55–59, 60–64, and 65–69 years and world champion in 2007 and 2009 (see Figure 15.2), show a minor decrement in performance over the years (0.38% per year in running time and 0.28% per year in running speed). As more elite competitors continue to participate in masters' athletics in

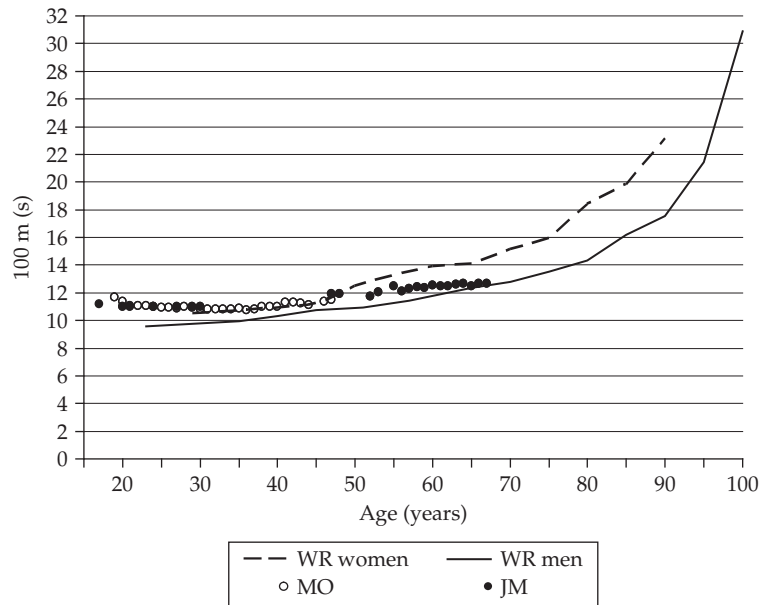


Figure 15.1 Official world records for the 100 m track event in different age categories for men and women (World Masters Athletics, 2009). The figure also shows the individual developmental processes for a female world record holder (MO) and for a male Finnish record holder (JM).



Figure 15.2 Jorma Manninen, number one master sprinter in Finland and world champion in the 100 m sprint in the age category of 65–69 years in 2007 and 2009. (Photo: Jaakko Avikainen.)

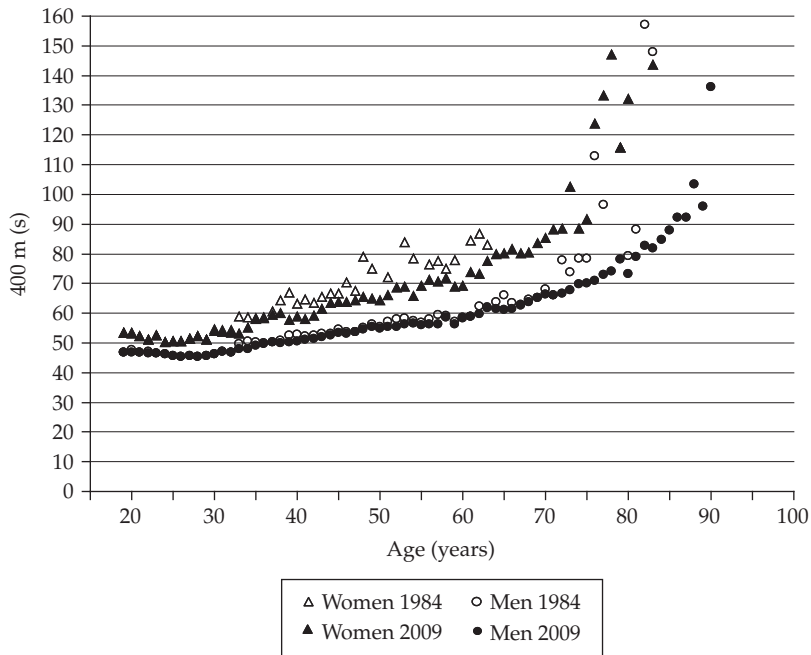


Figure 15.3 Development of the best Finnish performance times over 25 years for the 400m track event in annual age categories for men and women. (Data from Dunkel, 2009.)

the older age groups, it is likely that the current records, even in this highly competitive event, will further improve.

The improvement in record performances in the older age groups over the last 25 years can be clearly seen in the Finnish best times in the 400m track race (Figure 15.3). While the younger adults show hardly any changes, the increased number of active master competitors has markedly improved running times in the oldest men and women. Although age-related deterioration in performance is inevitable, athletes continue to maintain superior levels of performance throughout their lives in this type of athletic event where, in addition to strength and power, the capacity for anaerobic energy metabolism obviously plays an important role (Korhonen et al., 2005). For comparison, 80% of the male population at 85+ cannot even walk 500m without difficulties (Aromaa & Koskinen, 2004), not to mention running 400m in a minute and a half.

Somewhat different curves can be obtained for athletic events such as the high jump (Figure 15.4). Although the world records are again excellent in comparison with what can be presumed to

be the performance levels in the general population, the age-related decline in performance looks steeper in middle age (0.9% per year) and more linear throughout the age range than that reported for running speed in the previous examples. This may, in part, be due to the more complex mixture of strength, power, flexibility, and technical skill needed in the high jump than in the events such as sprint running. In addition to the aforementioned differences in competitive status and in training volume and intensity, the use of different jumping techniques in the younger compared to older athletes further emphasizes the role of cohort differences in cross-sectional comparisons. In the absence of any major changes in training or untoward injuries occurred, longitudinal data should thus indicate a much smaller age-related decline. This is supported by the results of jumper HS in the figure, who has retained a similar jumping technique and training volume over the years. In the case of a relatively low training status and performance level in adulthood, the plasticity of individual developmental processes makes it possible, at least over a limited period of time, to postpone the age-related decline

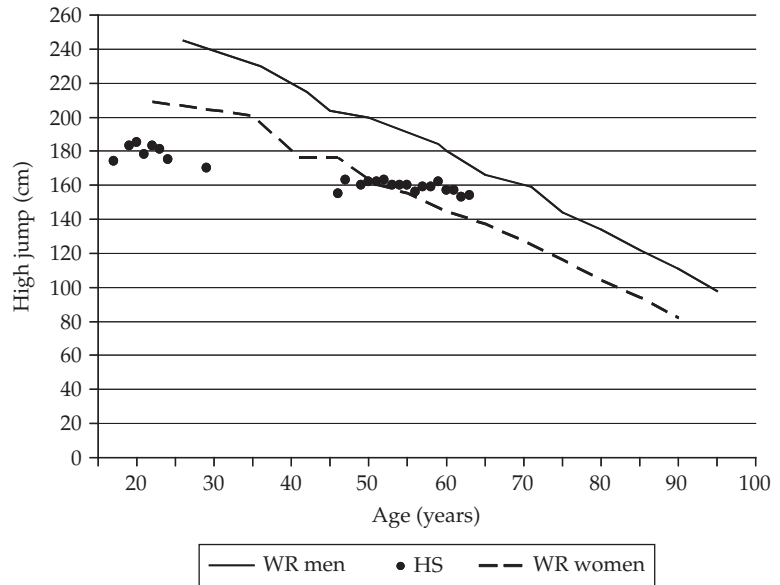


Figure 15.4 Official world records for high jump in different age categories for men and women (World Masters Athletics, 2009). The figure also shows the individual developmental process for a Finnish jumper (HS).

in performance or even to improve it (Conzelmann, 1997). In addition, an increase in the number of elite competitors with modern jumping techniques in the older age groups is likely to considerably improve the records.

Hurdling is a combination of a running race and a field event, where technique and style rule perhaps more than in any other event. It is a plyometric and ballistic event that demands high speed along with a highly refined technique on the part of the athlete. Consequently, an event-specific performance would be very difficult for the older age groups unless appropriate modifications to the event were made. In the masters' sprint hurdles, this is accomplished by shortening the running distance and the space between the hurdles, and lowering the height of the hurdles along with the increasing age of the athletes (Figure 15.5). The event remains demanding, but the best hurdlers can quite easily get through the event with normal hurdle clearance and the three-step pattern between the hurdles. Competitors may be motivated by anticipating success in the future age categories such as 50 and 70 years, where hurdling is made easier, thus enabling them to complete the race in about the same time as earlier.

Factors Affecting Performance

Multiple factors influence athletic ability and age-related decline in performance. As suggested earlier, these include the volume and intensity of physical training and the properties of the neuromuscular system, particularly those required in strength, speed, and power events. In the following, muscle strength and power, muscle mass and composition, fiber contractility, and neural activation ability are considered as primary outcomes of aging and training when discussing the determinants of performance in master athletes.

Muscle Strength and Power

Most skeletal muscle functions are impaired by the aging process. In normally active people, the reduction in muscle strength becomes evident in the sixth decade and occurs at an increasing rate of up to 2.5% per year thereafter (Larsson et al., 1979; Frontera et al., 2000; Suominen, 2007; Reaburn & Dascombe, 2009). Performance in maximal concentric and SSC muscle actions is usually lost more than performance in isometric and eccentric actions (Bosco & Komi, 1980; Vandervoort, 2002; Spirduso et al., 2005), particularly in the lower extremities,

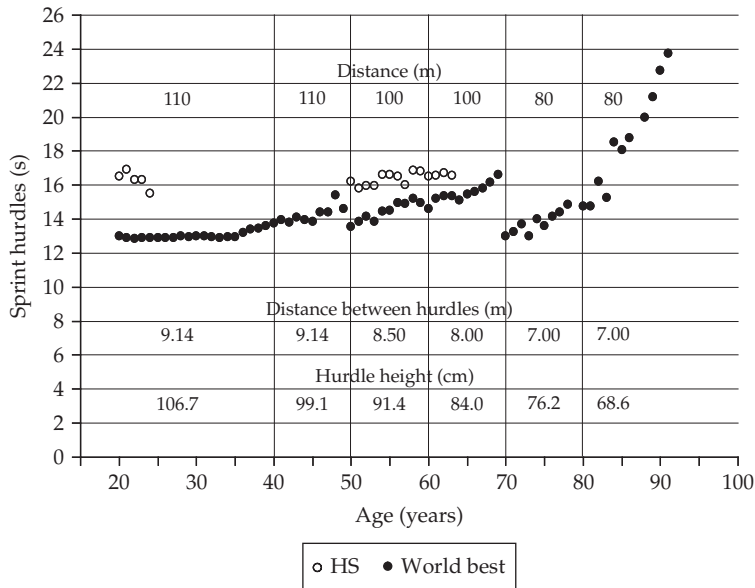


Figure 15.5 World all-time best results for sprint hurdles in the annual age categories for men. The figure also shows the individual developmental process for a Finnish hurdler (HS). (Data from World Masters Athletics, 2009; Gasselsberger, 2009; Larsson, 2009.)

which tend to lose more strength than the muscles of the upper extremities (Lynch et al., 1999).

Explosive strength and muscle power, the product of force of contraction and speed of movement, are particularly vulnerable to age-related changes. This is seen in performances where elderly persons are faced with a resistance or inertia, such as body mass in the vertical jump. The forces required to overcome this load represent a greater proportion of a weaker older muscle's maximum force. The nature of the F-V relation of skeletal muscle means that weaker older muscles have to contract more slowly to produce these forces. This results in a less optimum speed for power generation (Harridge & Suominen, 2003).

Master track and field athletes, such as sprinters, jumpers, and throwers, have superior strength levels well into old age (Klitgaard et al., 1990; Sipilä et al., 1991; Ojanen et al., 2007; Korhonen et al., 2009). However, the age-related decline in muscle strength appears to be similar or only slightly smaller in athletes than controls. The rates of decline estimated from cross-sectional data up to age 75–80 have been in the order of 0.8% per year in maximal bilateral isometric knee extension force in both sprinters (Korhonen et al., 2009) and throwers (Ojanen et al.,

2007), and 0.9% per year in dynamic strength (concentric half-squat 1-RM) in sprinters (Korhonen et al., 2009; see Figure 15.6). Nevertheless, estimated specific force, i.e. knee extension torque/muscle thickness, did not show typical age-related differences. The general decline in muscle strength in sprinters may, in part, be associated with the reduced volume of resistance training observed in older age groups (Korhonen et al., 2006). This is supported by the finding that incorporating weight-training exercises into the overall training resulted in improvements in maximal strength, even in a selected group of elite master sprinters (Cristea et al., 2008).

In view of the record performances of master athletes in sprinting, jumping, and throwing events, it is not surprising that their capacity in explosive strength and power tests, such as the rate of isometric force development and vertical CMJ, is far beyond the levels of sedentary persons (Sipilä et al., 1991; Ojanen et al., 2007; Aagaard et al., 2007; Michaelis et al., 2008; Korhonen et al., 2009). While a significant age-related decline is evident among these athletes as well, the data in sprint-trained athletes suggest that deterioration in the RFD and vertical jumping capacity proceeds only slightly faster than that in maximal strength (Korhonen

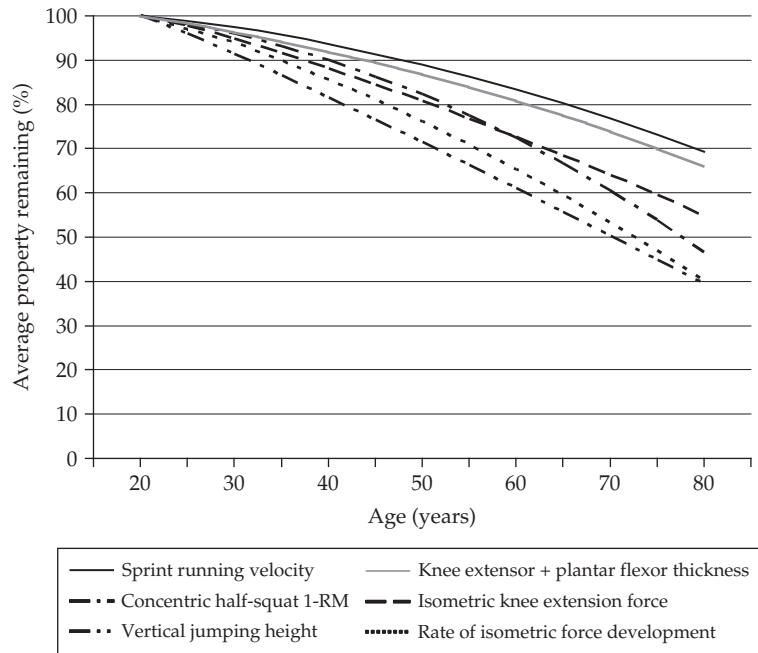


Figure 15.6 Average age differences in maximal sprint running velocity and associated neuromuscular factors from age 20 in male sprinters. (Adapted from Korhonen et al., 2009.)

et al., 2009, see Figure 15.6). Nevertheless, the sprinters show greater declines in the conventional muscle strength and power tests than in maximal sprint running velocity, which is likely to impose high requirements on force production and neuromuscular coordination. This may be associated with high training specificity and neuromuscular adaptation among these athletes, i.e., better maintenance of exercise stimuli for sprint running compared to many other, even simpler, tasks. In fact, when sprinters participated in a periodized strength training program, in which heavy-resistance exercises were combined with explosive types of weight training and plyometric exercises, their explosive force production and power characteristics improved significantly (Cristea et al., 2008).

Muscle Mass, Composition, and Contractility

The age-related decline in muscle strength and power is associated with a reduction in physiologic muscle CSA. As with muscle strength, the decrease in muscle CSA accelerates after about 50 years of age, resulting in a total loss of approximately

40% by age 80 (Lexell et al., 1988). In whole muscle studies, the decrease in the contractile material may even be underestimated, since with increasing age skeletal muscle is encroached with fat and connective tissue (Macaluso et al., 2002; Harridge & Suominen, 2003).

Age-related loss of muscle mass involves a marked reduction (up to 50% by age 80) in the number of muscle fibers and motor units, a decrease in fast type II fiber area, and a relative increase in “hybrid” fibers, i.e., fibers that express more than one of the MyHC isoforms (Lexell et al., 1988; Andersen et al., 1999; Faulkner et al., 2008). These changes may be associated with ongoing transformation processes, suggesting that the larger fast-twitch motor units are preferentially lost in older age (Harridge & Suominen, 2003). Single-fiber studies have shown that fibers that express the MyHC-I isoform are slower to shorten and generate less force per unit area and power than the MyHC-IIa and MyHC-IIx fibers (Larsson & Moss, 1993; Bottinelli et al., 1996; Harridge et al., 1996), and that there is an aging-related slowing of contractile speed and decrease in specific tension in

fibers expressing the MyHC-I and MyHC-IIa isoforms, which are the prime isoforms in elderly persons (Larsson et al., 1997).

Muscle atrophy may also accompany changes in the architectural arrangement of the fibers such as reduced fascicle length and pennation angle (Narici et al., 2003). Together with the changes in the tendon/connective tissue structures, this will further influence mechanical stiffness and force-generating capacity in various explosive sporting movements. Increased tendon-aponeurosis stiffness is beneficial for the transfer of muscular force and an association has been observed between the stiffness of the force-transmitting tissue and dynamic actions such as vertical jump performance and its force- and velocity-related determinants (Bojsen-Moller et al., 2005). On the other hand, tendon stiffness in knee extensors has been shown to be inversely related to augmentation in jump height from SJ to CMJ (pre-stretch augmentation), suggesting that too stiff tendon structures may have negative influence on the use of elastic energy in jump performance (Kubo et al., 1999). In old age, increased passive resistance of the connective tissue structures, particularly when occurring in the antagonist muscles, may also act against rapid elongation and joint movements. Experiments in old rats have suggested that increased flexor muscle stiffness during extension depends chiefly on age-related overgrowth of nonelastic connective tissue replacing degenerated active muscle fibers (Wolfarth et al., 1997).

As expected, master athletes, with long-term strength training history and superior muscle strength and power, show excellent values for muscle mass that are similar to those in much younger untrained persons (Klitgaard et al., 1990). However, muscle thickness in trained agonist muscles, such as TB in male throwers (Ojanen et al., 2007) and knee extensors and plantar flexors in sprinters (Korhonen et al., 2009), is reduced with ageing, as shown in Figure 15.6. This may, at least in cross-sectional comparisons, be partly associated with the lower volume of strength training and smaller body size of the oldest athletes.

Consistent with the whole muscle thickness and CSA, significant differences between master athletes and untrained people occur in muscle fiber

size. Older strength-trained athletes have larger CSA, especially of type IIa and IIb/IIx fibers in the VL muscle, than age-matched endurance-trained or sedentary men (Klitgaard et al., 1990; Aagaard et al., 2007). When considering their training background and competition history, elite master sprinters should provide even better athletic model for studying the potential of intensive training for counteracting the age-related atrophy of fast-twitch muscle fibers. However, sprinters also show a typical age-related reduction in type II fiber size and a shift toward a slower MyHC isoform profile, even though they can maintain fiber size above the normal values well into old age and preserve the qualitative aspects of muscle contraction, such as single fiber shortening velocity and specific force, virtually unchanged with age (Korhonen et al., 2006; Korhonen, 2009; Korhonen et al., 2009). When the sprinters participated in the aforementioned strength training program, the CSA of type IIa fibers was increased, but the single fiber-specific force and shortening velocity remained unchanged, suggesting that intensified training does not lead to further adaptations on the qualitative contraction mechanisms in these athletes (Cristea et al., 2008).

The early studies by Sipilä and Suominen (1991, 1993) showed that older male and female athletes also maintain better muscle architecture and quality in terms of better-defined fasciae and connective tissue septa and less fat than the controls. The results suggested that even endurance-type training may counteract the replacement of contractile tissue by other tissues such as fat. In a recent study of sprinters, pennation angle was shown to decline with aging in the VL, but not in the GAST muscle. There were no significant age-related differences in fascicle length in any of the muscles studied (Korhonen et al., 2009). To what extent the suggested better muscle quality and stiffer connective tissue structures along with greater tendon width (Kallinen & Suominen, 1994) in master athletes help to preserve fast force-transmitting capacity, and how this compares with the utilization of elastic energy in sport performances, needs to be studied further. In sprinters, the relative increase in vertical jump height from the static jump to the CMJ did not differ with age (Korhonen, 2009),

indicating that both force transmission and elastic energy storage and release are adapted to long-term training in older athletes, possibly in the way suggested by Bojsen-Moller et al. (2005), i.e., that proximal leg extensors mainly exhibit force transmission and that plantar flexors with a large tendon-to-muscle fiber length ratio contribute to the utilization of elastic energy.

The influence of tendon structures and the use of elastic energy in movement may, however, vary with muscle group and the type and intensity of performance. For example, studies in young competitive runners have indicated that greater elongation of the tendon structures of the VL is favorable for 100m sprint times, whereas no such relationship exists between the GAM and sprint performance (Stafilidis & Arampatzis, 2007). A compliant tendon of the triceps surae muscle might not match with the force production characteristics (reactive foot contact) in sprint running. It has been reported that the normalized stiffness of the triceps surae tendon and aponeuroses is higher in sprinters than in endurance runners and nonsport-active adults (Arampatzis et al., 2007), and that the load imposed by endurance running is not sufficient to counteract the effect of aging on tendon stiffness (Karamanidis & Arampatzis, 2006).

Neural Activation

Aging may also impair the effectiveness of neural mechanisms. For older untrained people, the most important concern could be the central activation and neural drive required to activate all the motor units of the agonist muscles (Stevens et al., 2003). However, it is not generally known to what extent the age-related impairment is related to the recruitment of a smaller number of motor units, a lower rate of motor unit recruitment or impaired motor unit synchronization. It has recently been suggested that the selective cell death of larger motor neurons, which have higher discharge rates than those of surviving motor neurons serving type I motor units, can impair the neural drive required to activate the agonist muscles (Lehman & Thompson, 2009). The resulting strength decrement may also be explained by increased coactivation of

the antagonist muscles in different isometric and dynamic actions (Izquierdo et al., 1999). In daily tasks, such as stair walking, the antagonist coactivation attempts to compensate for the reduced activation capacity of the agonists and to maintain joint stability and apply a safer neuromuscular strategy (Macaluso et al., 2002; Larsen et al., 2008). Recent evidence on nonathletes further suggests an age-related deficit in neural activation patterns during higher impact stretch-shortening actions. Using DJ exercise, Hoffren et al. (2007) observed that compared to young subjects, elderly subjects had lower relative agonist activation with higher antagonist coactivation in calf muscles in the braking phase. The lower activation in the braking phase with age may imply an inability to stiffen the fascicles and ankle joint leading to lower TT/MTU stretch ratio and less efficient utilization of tendon elasticity.

In comparison with muscle structure, little is known about the neural component of muscle force-generating capacity in master athletes. Pearson et al. (2002) found an age-related decrease in the maximal EMG amplitude of the agonist VL muscle during isometric knee extension in elite level male weight lifters. However, the quantity of maximal EMG was significantly higher in weight lifters than in untrained controls in all age groups. The study by Leong et al. (1999) indicated that the mechanism for higher neural activation in trained master weight lifters compared to untrained older people could be a higher discharge rate in the former.

Recent evidence also suggests that the rate of force production during fast voluntary contractions is compromised with age by decreases in the rapid neural activation of the motor units. For example, master throwers (Ojanen et al., 2007) and sprinters (Korhonen, 2009) showed an age-related decrease in relative agonist EMG activation during the first 100ms compared to maximum activation (normalized iEMG) during isometric bilateral leg extension (Figure 15.7). In sprinters, coactivation of antagonist muscles did not, however, significantly increase with aging, either in the early or late maximal force phase of rapid isometric action, thus differing from the previous findings on sedentary older people. Although surface EMG studies

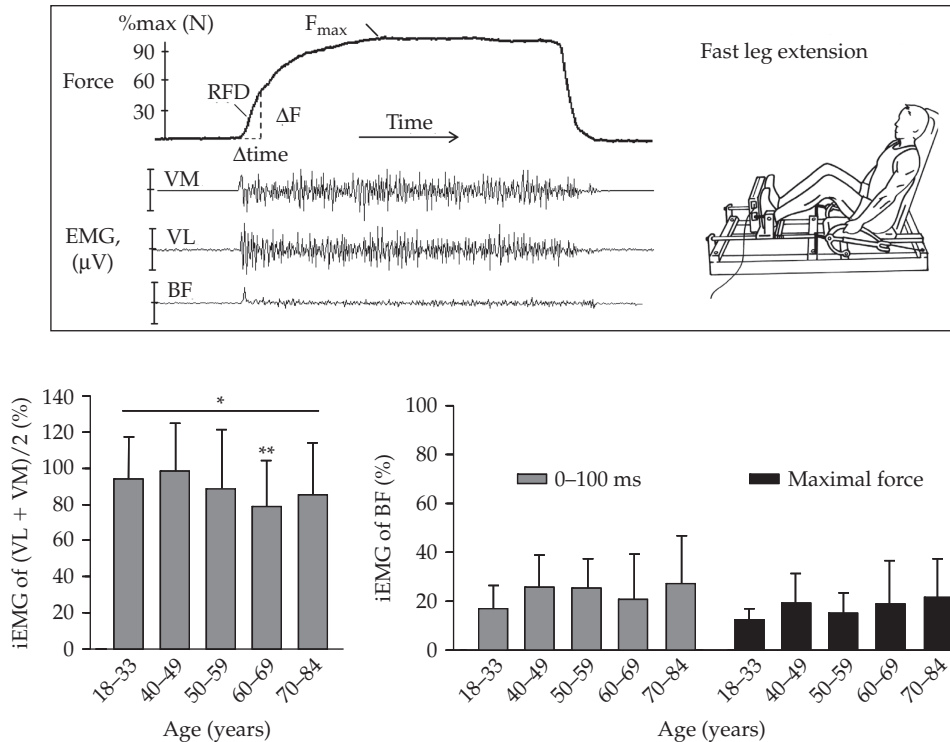


Figure 15.7 Electromyography (EMG) of the agonist (vastus lateralis, VL; vastus medialis, VM) and the antagonist (biceps femoris, BF) muscles during fast bilateral leg extension. The values for the different age groups of male sprinters represent relative agonist activation during 0–100 ms compared to maximum activation (lower left), and antagonist activation during 0–100 ms and the maximal force phase in relation to maximum integrated EMG (iEMG) during knee flexion (lower right). * $P < 0.05$, ** $P < 0.01$ significantly different from the maximal iEMG value; # $P < 0.05$ significant age effect. (Adapted from Korhonen, 2009.)

suggest that decreased efferent neural drive plays an important role in functional loss in maximal and explosive strength in aging athletes, no conclusive evidence has been obtained due to lack of information about the effect of aging on the specific neural mechanisms. Moreover, an important question that needs to be addressed in future athlete studies is whether age-related activation deficits also exist in the high-intensity SSC actions that characterize many sporting movements. Information about the degree to which specific systematic training with aging maintains neural control mechanism during SSC action (agonist/antagonist pre- and eccentric activation and reflex amplitude) would be valuable for developing effective exercise programs to limit the age-related decline in neural activation

and related loss of performance in natural dynamic actions.

Determinants of Maximal Running Speed with Aging

The last part of this chapter will summarize the age-related decline in athletic ability and the underlying factors discussed earlier by illustrating how selected neuromuscular characteristics of performance may work as determinants of maximal sprint running velocity with aging.

Accordingly, Figure 15.8 shows a schematic diagram of the association between the age-related decline of maximal running velocity and changes in stride cycle parameters, GRFs, and muscle strength,

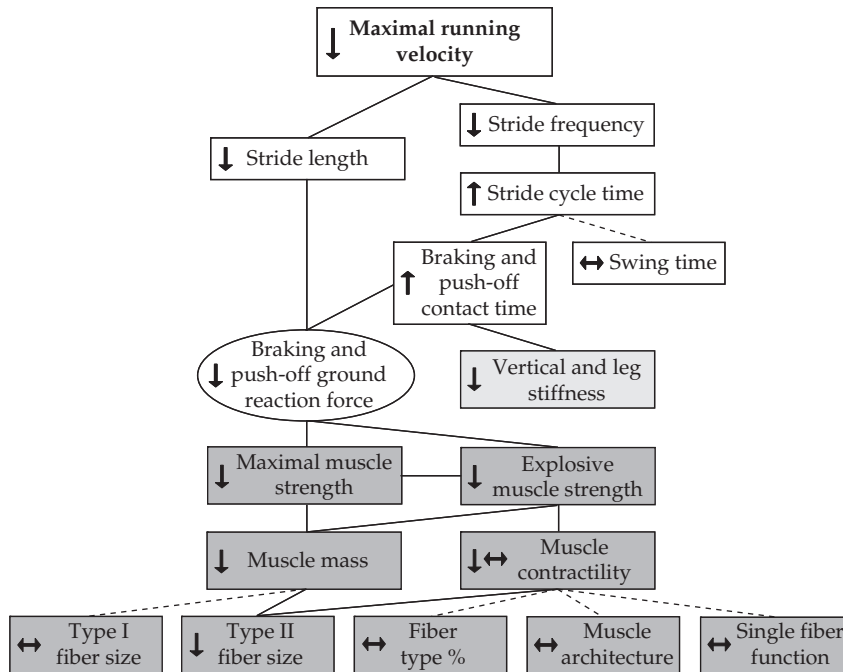


Figure 15.8 Schematic diagram of the biomechanical and neuromuscular factors that may determine age-related decline in maximal sprint running velocity in master sprinters. Arrows denote significant decrease (↓), increase (↑), or no changes (↔). (Adapted from Korhonen, 2009 and Korhonen et al., 2009, with permission by Wolters Kluwer Health, Lippincott Williams & Wilkins).

mass, composition, and contractility. It seems that the critical factors that impair running speed are decreased stride length, reduced ability to produce efficient GRFs, and decreased lower extremity stiffness to tolerate the higher impact forces involved in the fast transition from the braking to push-off phases (Korhonen, 2009).

The age-related changes in maximal and explosive strength required for generating running velocity and producing the above described GRFs can be attributed to the loss of muscle mass and the decline in fast-twitch fiber area rather than to the reduction in neural activation (Korhonen, 2009; Korhonen et al., 2009). The importance of hypertrophic adaptations is further supported by an experimental training study in master sprinters, where the improvement in maximal running velocity was accompanied by an increase in stride length, rate of propulsive force development, leg stiffness, isometric and dynamic strength, jumping

tests, and type IIa fiber area, while the changes in neural activation and the qualitative mechanisms of contraction were less evident (Cristea et al., 2008).

Conclusions

Master athletes with long-term devotion to strength and power-type physical training challenge the critical age-related changes in the neuromuscular system. Track and field records and sport-specific test results show that athletic performance may be preserved at an extraordinary high level well into old age. In line with this, the values for maximal and explosive muscle strength and power, as well as muscle mass, composition, and fiber characteristics are far above the age norms.

However, a significant age-related decline is nevertheless observed in most neuromuscular functions. This may, in part, be associated with a

reduced volume and intensity of strength training and consequent loss of muscle mass in the older age groups. In track athletes such as sprint runners, combining sprint training with heavy and explosive strength exercises may induce further improvement in maximal, explosive and sport-specific force production, and hypertrophy of fast-twitch muscle fibers.

Modified power types of exercises combined with heavy-resistance training could also be

recommended as part of overall physical training for less active middle-aged and elderly persons to prevent fast-twitch fiber atrophy and loss of explosive strength and power. If, before the onset of mobility impairments, elderly persons could switch their physical activity toward the type of training practiced by master athletes, they would have considerable potential to improving their neuromuscular function and, thereby, reducing the risk of mobility impairments with aging.

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Chapter 16

Rehabilitation of Overuse Tendon Injuries and Ligament Failures

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Introduction

This chapter focuses on the implementation of recently generated biomechanical knowledge into functional treatment of injured tendons and ligaments. Besides muscle strains, tendon and ligament injuries are most common in athletes, and the structures of the lower extremity are most frequently affected. Especially, the large tendons in the lower extremity are subjected to overload as a result of chronic loading (repetitive microtrauma). Ankle and knee ligaments are most frequently strained when the joint mobility exceeds its anatomical constraints due to an acute injury.

Incomplete structural healing and insufficient functional rehabilitation of tendon and ligament injuries maybe the first step into an injury cascade, ultimately leading to poor sports performance. Profound knowledge of the injury mechanisms, differential diagnostic, treatment, rehabilitation, and preventive strategies therefore are major concerns aiming to minimize the absence of athletes from training and competition.

Achilles Tendon

AT Tears

Epidemiology

Achilles tendon tears are frequent. About 11.3 AT ruptures occur per 100,000 people per year in a general population (Clayton & Court-Brown, 2008). Over the past decades, the number of AT ruptures seems to increase as shown within a Danish population. In 1984, 18.2/10,000 inhabitants sustained an AT rupture, and that frequency has increased to 37.3/10,000 inhabitants in 1996. In that population, males more frequently than females (3:1) sustained that injury (Houshian et al., 1998). It is to expect that this epidemiologic change is even true for the Olympic athlete. The reasons for this increase remain unknown.

The majority of the AT ruptures (74.2%) that occurred were sport related. Ball and racket games seem to bear a heightened risk causing 89% of these ruptures. Sport-related ruptures (74%) occurred predominantly in the age group of 30–49 years while ruptures not related to sport were found most frequently between 50 and 59 years (Houshian et al., 1998). Thus, it seems that athletic activity is a risk factor within a certain age group to sustain an AT rupture.

Etiology

It seems most probable that AT tears are the result of multifactorial influences. In a histological study, completely healthy tendon structures could only be found in the control group (152 out of 222 previously healthy individuals), while all 397 ruptured ATs revealed preexisting pathological changes (Kannus & Jozsa, 1991). Recent biomechanical research suggests that load is nonuniformly distributed across the different areas within the AT. Sport-related maneuvers like cutting, jumping, or landing on uneven grounds may induce peak loads in specific AT fibers presumably resulting in microtears. When the tendon load increases additional stress overloads the remaining fibers. This further damage may most probably lead to a complete tendon rupture.

Literature suggests that other agents serve as predisposing cofactors for AT tears. Pharmacotherapy (fluoroquinolones, corticosteroids), inflammation, genetic profile (male, ABO blood group), and altered lipid metabolism are intrinsic factors affecting the AT's quality. Training errors ("too much too early"), weather conditions (cold and moist), poor equipment, and inadequate training terrain are regarded as extrinsic factors of risk for AT structural damage (Cook & Khan, 2007). Currently, however, the threshold for each single risk and combinations of risk factors acting as real pathologic catalysts for a specific individual in a specific situation is difficult to define.

Rehabilitational Concepts

Currently, there are three different concepts available for initial treatment of acute AT ruptures. Traditionally, open surgery has been the treatment of choice. Using long paraachillar approaches, end-to-end repairs were performed by different suturing techniques or augmented reconstructions. Besides this, minimal invasive percutaneous suturing has increasingly become popular during the past decade. The third option is conservative treatment.

Postoperative and conservative rehabilitation traditionally follows a 6- to 8-weeks plaster immobilization in an equinus foot position. In an animal model on ligament healing, superior results following

early functional rehabilitation protocols and deleterious effects following prolonged immobilization were achieved (Woo et al., 1987). Consequently, functional rehabilitation by orthotic posttreatment is meanwhile established as the golden standard. Summarizing and analyzing the results from literature yield that "open operative treatment of acute Achilles tendon ruptures significantly reduces the risk of re-rupture compared to non-operative treatment, but produces a significantly higher risk of other complications, including wound infections. The latter may be reduced by performing surgery percutaneously. Post-operative splintage in a functional brace appears to reduce hospital stay, time off from work and sports, and may lower the overall complication rate" (Khan et al., 2004).

Based on personal experience with top-level athletes, it can be summarized that an AT tear consistently results in reduced athletic performance regardless of performed initial and postsurgical treatment. However, following expert opinions, there is a tendency to open surgery for this specific group of patients as this treatment offers a reduced risk for re-rupture compared to the conservative option (Khan et al., 2004).

Principles of an Orthotic Device for Posttreatment of AT Tears

Orthoses used for rehabilitation of AT tears are applied to limit dangerous joint motions and therefore protect the healing structure from overload. Medical and clinical experiences as well as functional analyses leave only little doubts for the superiority of early functional treatment following an AT rupture (Khan et al., 2004). Theoretically, progressive load should be applied to the healing AT during the course of the rehabilitation. This means that the healing AT must be specifically protected in the early phase (about 6–8 weeks), whereas during the following 4–6 weeks, a stepwise reduction of external functional fixation is demanded.

For functional conservative and postoperative treatment, several ankle foot orthoses and walking boots have been introduced. One example trying to fulfill the demands for functional AT rehabilitation is the Vario-Stabil boot (Orthotech GmbH,

Gauting, Germany). The individually adjusted device is composed of specific AT load reducing elements arranged modularly (Figure 16.1). A rigid anterior nylon support tongue restricts ankle dorsiflexion. This thermoplastic element is adapted to the desired equinus position by individual thermomodulation. A variable heel lift supports the foot's equinus position. Rigid medial and lateral nylon lower leg stabilizers prevent the foot from pronounced varus or valgus movement. During the course of the AT rehabilitation, the heel lift is reduced stepwise and the thermoplastic elements are adapted. With that, the treating physician intends to put progressive load to the healing AT according to its actual capacity as judged clinically.

The question how much load should be applied to the healing AT in a specific phase of the rehabilitation of an individual patient cannot be actually answered without the knowledge of directly measured tendon forces. In order to establish objective data for the amount of AT load depending on different rehabilitational devices and during different structural conditions of each of these devices in vivo, experimental investigations have been performed (Froberg et al., 2009; Lohrer et al., 2003).



Figure 16.1 Modular construction of the Vario-Stabil boot. Most important elements are rigid anterior nylon tongue, variable heel lift, and rigid medial and lateral nylon lower leg stabilizers.

AT Tensile Stress During Rehabilitation

The effect of two different ankle foot orthoses on AT load has been studied in subjects with uninjured AT (Froberg et al., 2009; Lohrer et al., 2003). In both studies, functional constructive elements of these devices implemented to reduce the load on the AT were tested using the OF technology (Komi et al., 1996) which enabled a direct in vivo measurement of the AT load under the protection of these rehabilitational ankle foot orthoses. Additionally, surface EMGs of different muscles acting on the ankle were recorded. To enable the implantation of the OF, the ATs were uncovered from surrounding shoe material (Figure 16.2).

One study tested the subjects during single-leg stance and while walking on an even surface. Results show that the completely equipped Vario-

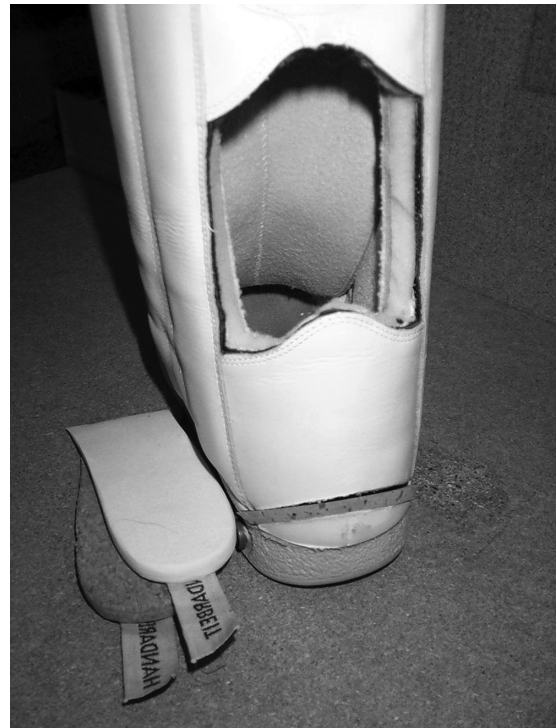


Figure 16.2 The Vario-Stabil boot has been prepared for the OF measurements. Two posterior windows are cut to enable OF implantation and modular heel lifting with the boot in place.

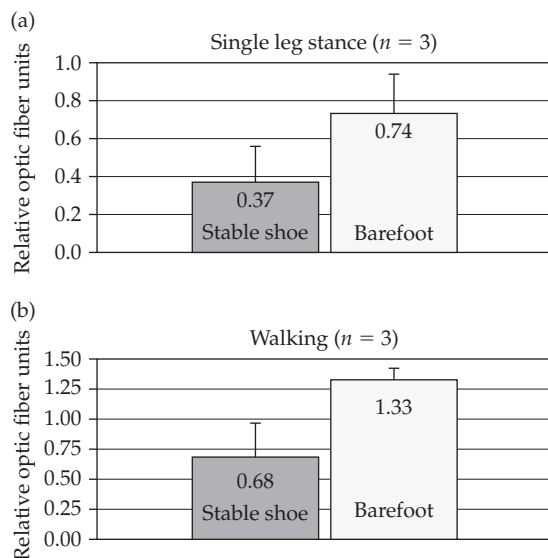


Figure 16.3 Maximum OF values during single-leg stance (a) and during walking (b) in barefoot (control) and boot (test) condition. Maximum values registered during stance phase. Mean values and SDs calculated from nine load phases of each subject. 1 = OF amplitude during isometric maximum voluntary contraction.

Stabil boot with the foot fixed in an equinus position reduced the AT load compared to the barefoot (control) condition. However, there was no complete reduction of the AT load observable (Figure 16.3a and b). Reduction of the heel height induced a considerable increase in tensile load. The highest values were observed in the barefoot condition. Even with the fully equipped stable boot, the AT load was reduced by only about 50% compared to the barefoot condition. This means that a relevant load is applied to the healing AT, providing the desired functional stimulation to the healing tendon tissue. Another interesting result from that study is an approximately parallel course of the EMG coinciding with OF signal excursion. Considering an EMG delay, which was already demonstrated earlier (Komi et al., 1996), these curves present a similar pattern (Figure 16.4).

In a different experimental approach (Froberg et al., 2009) the lower leg was fixed in an ankle foot orthosis featuring adjustable ankle angles that allow for controlled range of motion (short leg walker with articulating hinge, DeRoyal Europe Inc, Ireland). In that study, maximum ATF was highest

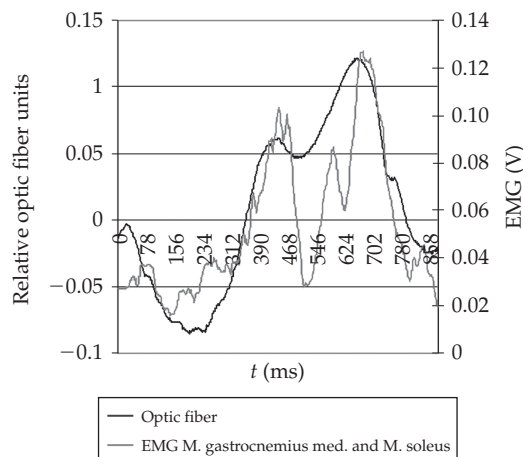


Figure 16.4 Example for similar forms of the OF and smoothed EMG curves obtained from one subject during walking. There is a high correlation ($r = 0.821$). Mean values and SDs calculated from three load phases of each subject. 1 = OF amplitude during isometric maximum voluntary contraction.

when dorsiflexion was limited to 20° equinus (3.1 times BW). AT load progressively decreased when dorsiflexion was limited to 10° equinus and 10° dorsiflexion. Surprisingly, only 2.1 times BW AT load was measured during barefoot walking ($P < 0.01$). In contrast to this, but in line with the results of the aforementioned study, the SOL EMG activity was clearly reduced when dorsiflexion was progressively restricted and was highest during barefoot walking. As the ankle foot orthosis seems to put more load to the AT, one could conclude to propagate barefoot walking to reduce the tendon load in the initial phase of rehabilitation following AT tears or surgery.

Thus, the available information is conflicting with regard to the lower leg EMG activity and AT loading as measured with the OF method. The differences maybe explained by different functional demands provided by the orthotic devices used in the respective tests. Unprotected rehabilitation (barefoot) following AT tear has never been reported in literature and therefore should not be implemented in rehabilitational concepts before further investigation has clarified this issue. The clinician is responsible to protect the healing tendon tissue from too much load especially in the initial

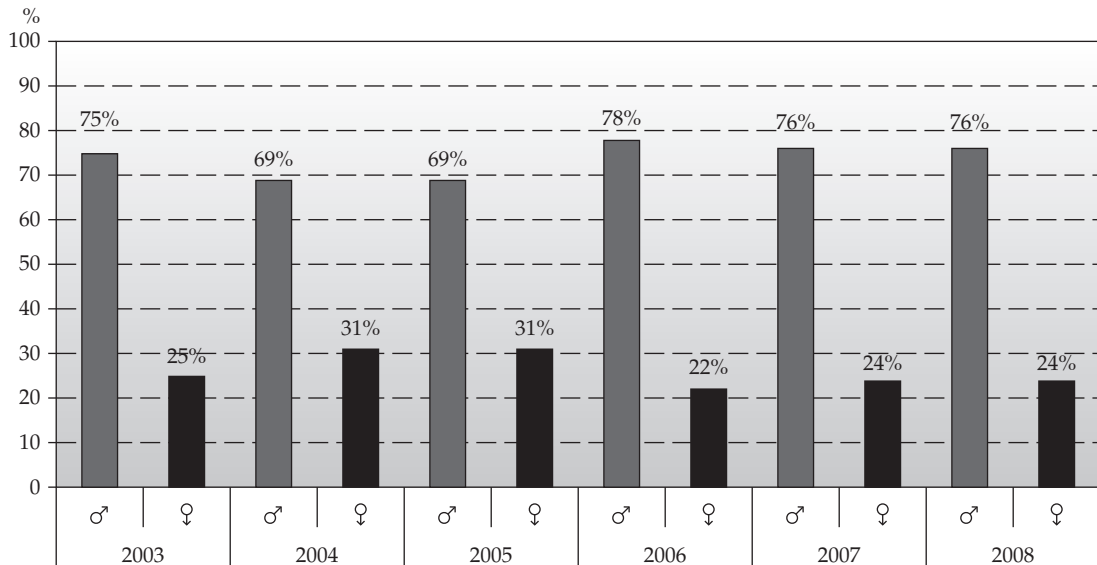


Figure 16.5 Gender difference in patients suffering from Achilles tendinopathy. Five years evaluation, Institute for Sports Medicine Frankfurt am Main.

phase of the rehabilitation. The AT load should thus increase over time. Especially in the early phase of the rehabilitation, no single barefoot loading is allowed as this would clearly overload the sensitive scar tissue leading to tendon elongation and functional insufficiency leading at least to impaired performance or even inability in sport.

AT Overuse Injuries and Haglund's Disease

Definitions

The terminology for overuse injuries of the AT and the surrounding area (posterior heel pain) is traditionally confusing. For reasons of different etiology and treatment, Achilles tendinopathy, Haglund's disease, and AT insertional lesions should be differentiated. The diagnosis of these overuse injuries relies on clinical examination. Pain on palpation over the respective area and load-induced pain especially during warming up and mainly triggered by sports activities are mandatory findings. Tendon or paratenon swelling is frequent but not necessarily present. *Achilles tendinopathy* is defined as an overuse induced lesion of the main body of the AT and/or its paratenon located between the myotendinous junction and about 2cm proximal to the calcaneal

insertion. *Haglund's disease* is defined as a chronic retrocalcaneal bursitis induced by an impingement between a prominent posterior calcaneal tuberosity (Haglund's prominence) and the anterior AT border. *Achilles tendon insertional tendinopathy* is a chronic pathology affecting directly the junction from the distal AT into the calcaneus. It is frequently associated with radiographically detectable posterior heel spurs representing intratendinous calcifications and with systemic diseases like diabetes, hyperuricemia, seronegative spondyloarthropathies, Cushing syndrome, hyperlipidemia, and rheumatic disorders. Therefore, AT insertional tendinopathy should not only be regarded as a typical overload sport induced injury but also as an enthesopathy.

Epidemiology

During the 2000 Olympic Games in Sydney, 30% of the members of the German track and field team were restricted in their activity due to Achilles tendinopathy or Haglund's disease. An incidence of 0.01/1000km is reported in runners (Knobloch et al., 2008). Achilles tendinopathy involves mainly males (Figure 16.5) and according to our experience, it is expected to occur after 12 ± 8 years of

training. Apparently, the appearance of this overuse injury depends on various factors, especially on the intensity and volume of training or competition. Haglund's disease also predominantly involves runners. Compared to Achilles tendinopathy, it is more rare (3:2) and has been found to occur after 13 ± 4 years of athletic training in our patients.

The association between AT injuries and risk factors like body height, malalignment of the foot and lower extremity, and muscular imbalance remains unclear and the respective research findings are inconsistent (van Mechelen, 1992).

Recently, we developed a novel method to measure the individual anatomic position of the subtalar joint axis in vivo. A large ($n = 495$) field study showed that the incidence of chronic AT problems seems to be related to the spatial orientation of the individual subtalar joint axis. Further research in this field should be directed to understand the underlying biomechanical reasons.

Rehabilitational Concepts

In a systematic literature review, treatment options for tendinopathy were evaluated (Andres & Murrell, 2008). The authors conclude that NSAIDs (non-steroidal anti-inflammatory drugs) and corticosteroids appear to provide short-term pain relief, but the long-term effectiveness has not been demonstrated yet. Shockwave and physical therapy modalities (ultrasound, iontophoresis, and low-level laser therapy) provided inconsistent results. Eccentric strengthening, sclerotherapy, and nitric oxide patches are recommended. Even if surgery is reported to result in "inconsistent outcomes," it is recommended as "the last option" in the treatment chain. This knowledge leads to the conclusion that "the ideal treatment for tendinopathy remains unclear." Nevertheless, about 50% of Haglund's patients have been resistant to conservative treatment and therefore have to be regarded as candidates for operative treatment (Nicholson et al., 2007).

Eccentric training is a treatment option which has become increasingly popular in Achilles tendinopathy in the past 10 years. A recent randomized and controlled study compared eccentric AT training performed daily for 12 weeks with three sessions

low-energy shockwave therapy applied in weekly intervals. A third group served as control ("wait and see") but advices regarding training modifications, stretching exercises, and ergonomic aspects were given to these patient. Additionally, paracetamol or naproxen was allowed as pain medication in that group. Results support the effectiveness of eccentric strengthening and low-energy shockwave therapy for Achilles tendinopathy. "Complete recovery" or "much improvement" was observed at 4-month follow-up in 15/25 of the eccentric, 13/25 of the shockwave, but only in 6/25 of the "wait and see" group, respectively (Rompe et al., 2007).

Historically, it has been generally agreed that pain-provoking activity should be restricted during Achilles tendinopathy treatment. Athletes, however, wish to continue with their specific training. In this context, another level 1 investigation is interesting especially for the athletic population. It was questioned if continued AT loading activities exerted during the treatment phase were relevant for the outcome. In that study both groups were treated for Achilles tendinopathy with an identical daily performed exercise program including eccentric activities. The exercises were continued until a patient reported to be free of symptoms. One group ($n = 19$) was allowed to continue AT loading activities like running and jumping, if the resulting pain level was tolerable. Participants of the second group ($n = 19$) had to stop those activities during the initial 6 weeks. After 12 months, both groups were equal with respect to the VISA-A questionnaire and the pain level during tendon loading (Silbernagel et al., 2007).

It is hypothesized that Achilles tendinopathy is connected with pronounced subtalar motion. For treatment it, therefore, seems logical to stabilize the subtalar joint. This can be effectively done by orthotics.

Patellar Tendinopathy (Jumper's Knee)

Definition

Jumper's knee represents an overuse injury of the patellar tendon featured by a localized tenderness at the inferior pole of the patella. Imaging

(MRI, ultrasound) may reveal degenerative tissue and neovascularization concentrated in the posterior region of the proximal patellar tendon but pathologic MRI and ultrasound findings can be found in asymptomatic tendons and even tendons with inconspicuous imaging can be symptomatic (Cook & Khan, 2001).

Epidemiology

In elite-level athletes from different sport disciplines, the overall prevalence of jumper's knee was 14.2%. Volleyballers and basketballers are particularly at risk. Patellar tendinopathy is frequently progressive and is a long-lasting disorder (Kettunen et al., 2002). The prognosis for patients suffering from this injury is limited with respect to a successful career in the specific sport. In a prospective case control study, 53% (9/17) of the athletes suffering from patellar tendinopathy at baseline but only 7% (1/14) in the control group discontinued their career as a consequence of their knee problems (Kettunen et al., 2002). Jumper's knee predominantly involves males (Figure 16.6).

Etiology

Achilles tendon and patellar tendon both lack a tendon sheath. Instead, a paratenon surrounds these tendons. Contrasting, however, patellar tendinopathy is located at the enthesis between the patellar ligament and the apex of the patella and not in the tendons' midportion. It is generally agreed that chronic overload produced by intensive jumping and landing activities in sport is a considerable major cause of patellar tendinopathy. So, degeneration induced by the tendon's incapacity to adequately repair microtears and not inflammation is the predominant histologic finding (Cook & Khan, 2007). Impingement between the inferior patellar pole and the patellar tendon is additionally discussed as a relevant etiologic agent.

Rehabilitational Concepts

Treatment of jumper's knee is not based on high evidence levels and is therefore empirically and polypragmatically handled. Rest, exercise, stretching, ultrasound, electrotherapy, heat, cryotherapy, frictions, and local or systemically applied

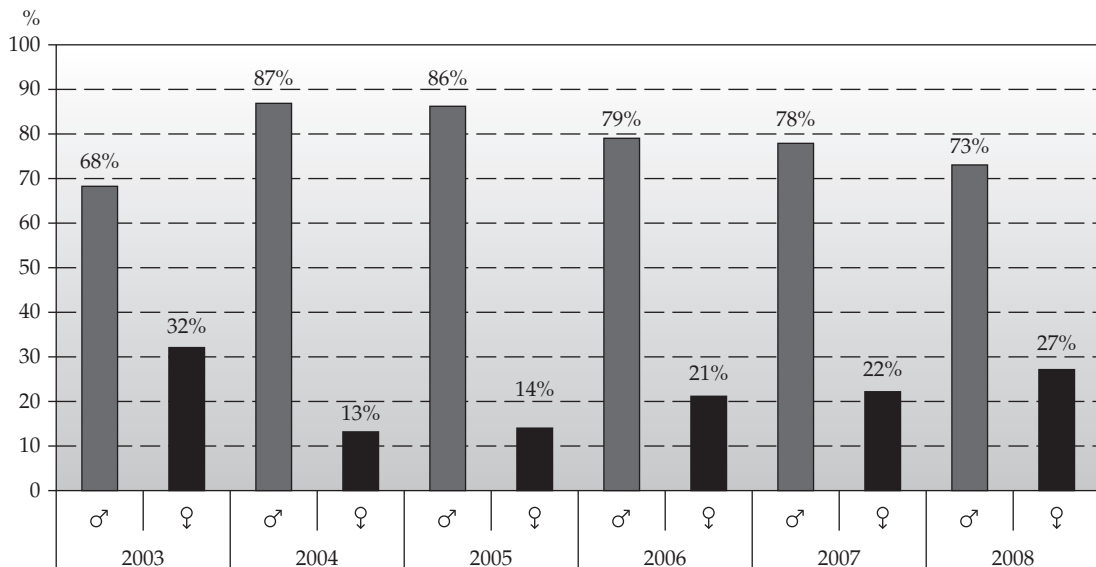


Figure 16.6 Gender difference in patients suffering from jumper's knee. Five years evaluation, Institute for Sports Medicine Frankfurt/Main.

drugs (corticosteroids, nonsteroidal antirheumatic [NSAR]) have been popular conservative modalities, but no specific treatment including surgery could be recommended from literature analysis (Cook & Khan, 2001). Meanwhile, eccentric training has increased in popularity. In a recent literature analysis, seven randomized controlled trials (162 patients) published later than 2000 were found. It was concluded that “eccentric training may have a positive effect” (Visnes & Bahr, 2007). Peritendinous corticosteroid injections, eccentric decline squat training, and heavy slow resistance training have been compared in another randomized controlled single-blind trial (Kongsgaard et al., 2009). Results show that corticosteroid injections have good short-term effects but results deteriorated between the 12 weeks and 6-month follow-up. Resistance training with high loads and slow contraction dynamics revealed good effects at both follow-up investigations, and additionally increased collagen turnover. Sclerosing injections also had favorable effects in an evidence level 1 study (Hoksrud et al., 2006). Actually, platelet-rich plasma was implemented for clinical use in patellar tendinopathy patients but its usefulness requires further investigation and the interference with antidoping regulations has to be cleared.

Operative resection of the involved tendon tissue is performed in recalcitrant cases and can be performed open or by arthroscopy. However, controlled investigations addressing the effects of operative treatment are completely missing. According to our experience, it has to be emphasized that postoperative rehabilitation needs up to 1 year.

In Vivo Evaluation of Load Distribution in the Proximal Patellar Tendon

In vivo measurements were performed in seven healthy persons, inserting an OF transversely into the anterior and posterior column of the proximal patellar tendon, respectively. Compared to the anterior located OF, the posterior fiber perceived more load during all the investigated exercises. Jump and squat exercises induced the greatest differential signal output, indicating greater forces in

the posterior region of the patellar ligament origin (Dillon et al., 2008).

These investigations demonstrate that the patellar tendon is likely asymmetrically loaded during knee activity. The posterior column of the proximal patellar tendon transmits the greatest loads. This is especially true for higher flexion angles in the knee. Preventive training therefore should aim to optimize taking-off and landing techniques for athletes at risk.

Ligament

Ankle Ligaments

General Considerations

Deltoid ligament lesions and ankle fractures or even dislocations maybe initiated by excessive pronation combined with external rotation. Medial ankle instability is a rare condition (Ferran et al., 2009). Consequently, the following section focuses on lateral ankle ligament injuries.

Current evidences indicate that ankle sprains resulting in partial or complete ligament tears are multicausal events based on different extrinsic and intrinsic risk factors. Two types of acute injuries play an important role especially in team sports like soccer, basketball, handball, or volleyball: contact injuries involve other athlete(s) while noncontact injuries involve no other athlete(s). The main stream in prevention research is focussed on non-contact injuries and the occurrence is expected to be influenced by preventive interventions. Therefore, these kinds of injuries will exclusively be considered in this section.

Epidemiology

Lateral ankle injuries are the most common injuries across a wide variety of sports. The risk to sustain ankle sprains is especially increased in sports like basketball, soccer, and volleyball. Numerous epidemiologic studies have shown that about 10–30% of all sports injuries are ankle sprains and as much as 73% of competitive and recreational athletes are considered to have a history of recurrent ankle

sprains (Dizon & Reyes, 2009). It has been established that the rate of injury is higher during game or competition than in practice. The most important intrinsic risk factor is a previous injury. In a systematic review including 24 high-quality trials, full recovery was reported by 36–85% of the analyzed patients within a period of 3 years. However, 5–33% of the players suffered from ankle pain after 1 year. Intensive training (>3 times a week) and being male were recognized as prognostic factors for residual symptoms. Three to 34% of the athletes had a re-sprain between 2 weeks and 96 months after the initial injury (van Rijn et al., 2008).

Injury Mechanisms

It is commonly accepted that lateral ankle noncontact sprains are the result of a combined plantar flexion and inversion (supination). Video analyses of 26 live inversion injuries in football revealed that a laterally directed force is induced by an opponent's foot on the medial aspect of the lower leg or ankle just before or at foot strike causing the ankle to land in an inverted position (Figure 16.7). A second mechanism consists of forced plantar flexion of the player's foot during shooting or clearing the ball while striking an opponent's foot. This mechanism is supposed to cause "footballer's ankle" by a

repetitive traction strain to the anterior capsule of the ankle (Andersen et al., 2004).

Recently, an ankle inversion injury was accidentally observed in a dynamic biomechanical (kinematic and kinetic) study during cutting motion trials. Compared with the preceding trials (uninjured), it could be shown that between 11 and 20 ms after ground contact the ankle explosively entered a combined 48° of inversion, 10° of internal rotation position, and especially noticeable 18° of dorsiflexion position leading to a grade I sprain (Fong et al., 2009).

Jumping and landing are skills frequently performed by basketball players. Accordingly, 45% of all ankle injuries were induced during landing, and almost another one-third (30%) of ankle injuries occurred during a sharp twist or turn (McKay et al., 2001). A clear understanding of the injury mechanism requires not only the analysis of movements resulting in a specific injury but also the knowledge of mechanical properties (e.g., failure loads, stress-strain behavior) of the lateral ligaments. In that field, only few cadaver studies have been performed. From these we calculated a mean ultimate tensile strength of 177 N for the ATFL. Direct stress measurements have been performed using different types of implantable force transducers in the ligaments while selected movements were studied. Both the small length (<20 mm) of the ATFL and its anatomic variations seem to be critical points in these investigations.

We used an *in vivo* approach based on the paper of Komi et al. (1996) to investigate load in the ATFL during static and dynamic load. The OF technology has been introduced in 1999 to measure stress in the ATFL. In uninjured subjects it could be shown that the signal of the measurement system increased during unloaded inversion movements and during manually performed anterior talar drawer and talar tilt test. Compared to the unloaded situation, loading with BW increased the signal obtained from the ATFL only slightly. This finding corresponds well with previous research indirectly indicating that incremental loading of mechanically unstable ankles on sloped surfaces was able to increasingly reduce the talar tilt radiographic

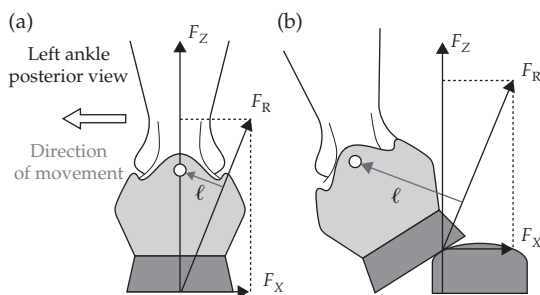


Figure 16.7 Simplified schematic drawing of a left ankle viewed from posterior. Vectors of resulting forces (F) during lateral ankle sprains: F_x horizontal GRF, F_z vertical GRF, F_R resulting force, l lever arm of F_R with respect to the estimated axis of the subtalar joint. (a) Landing or cutting with the left ankle on even surface and (b) landing on uneven surface.

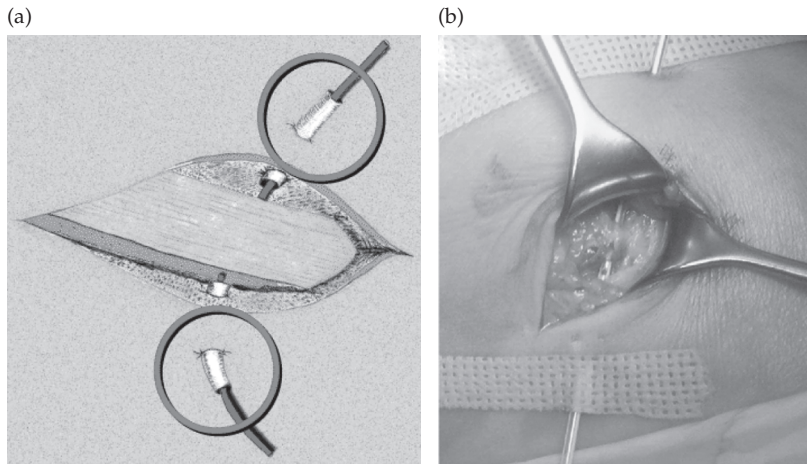


Figure 16.8 Drawing (a) and corresponding photograph (b) of the implanted OF in the ATFL in vivo. Note: the circles within (a) and the arrows within (b) indicate the catheters which were used as a tube to prevent the OF from mechanical interaction with surrounding soft tissue before entering the ligament under consideration (ATFL). Using this configuration, it could be guaranteed that the fiber was only in contact with the tissue of interest.

instability (Gollhofer et al., 2000). Bending of the OF (as a stress transducer) is the most critical problem; therefore, in this investigation, the fiber has been prevented from interactions with the surrounding soft tissue by catheters inserted into the skin and the subcutaneous fat (Figure 16.8). Nevertheless, the data should be carefully interpreted as calibration was not possible and the experiments are not repeated by other authors so far.

Rehabilitational Concepts

Patients as well as surgeons tend to neglect “minor ankle injuries.” In this context, the term “ankle sprain” only describes an injury mechanism without clear diagnostic evidence. Therefore, the resulting ligament and possible accompanying joint lesions must be further graded and differentiated, finally leading to adequate treatment (Figure 16.9).

In the literature, a large variety of studies have been presented dealing with the management of ankle ligament injuries, and numerous systematic reviews and meta-analyses of ankle ligament injuries have been conducted. Derived from these analyses, functional treatment of acute lateral ankle ligament lesions has been proposed as the method of choice (Kerkhoffs et al., 2007). However, as

conservative treatment results in a significantly higher incidence of objective instability, it is also suggested to surgically repair acute ligament tears in athletes (Ferran et al., 2009).

Even if the evidence obtained from in-depth studies is limited, functional treatment is considered to be superior compared with immobilization of acute ankle sprains (Kerkhoffs et al., 2002a). A certain amount of external support is necessary for a successful rehabilitation following lateral ankle ligament injuries and initially, a short period of immobilization maybe useful. The patients included in a recent randomized and controlled study, designed to evaluate that issue, were unable to bear weight on the injured leg in the first 2–3 days after injury indicating a severe sprain or additional joint lesions. Documented by a 3-month follow-up investigation, patients in a below-the-knee cast for only 10 days or an ankle brace had a more rapid recovery than those provided only with a tubular compression. Nine months following the injury, differences between the devices disappeared (Lamb et al., 2009). The question which kind of functional external ankle stabilizers is most appropriate for treatment of ankle sprains is currently unsolved. Nevertheless, an elastic bandage appears to be insufficient and produces more instability and

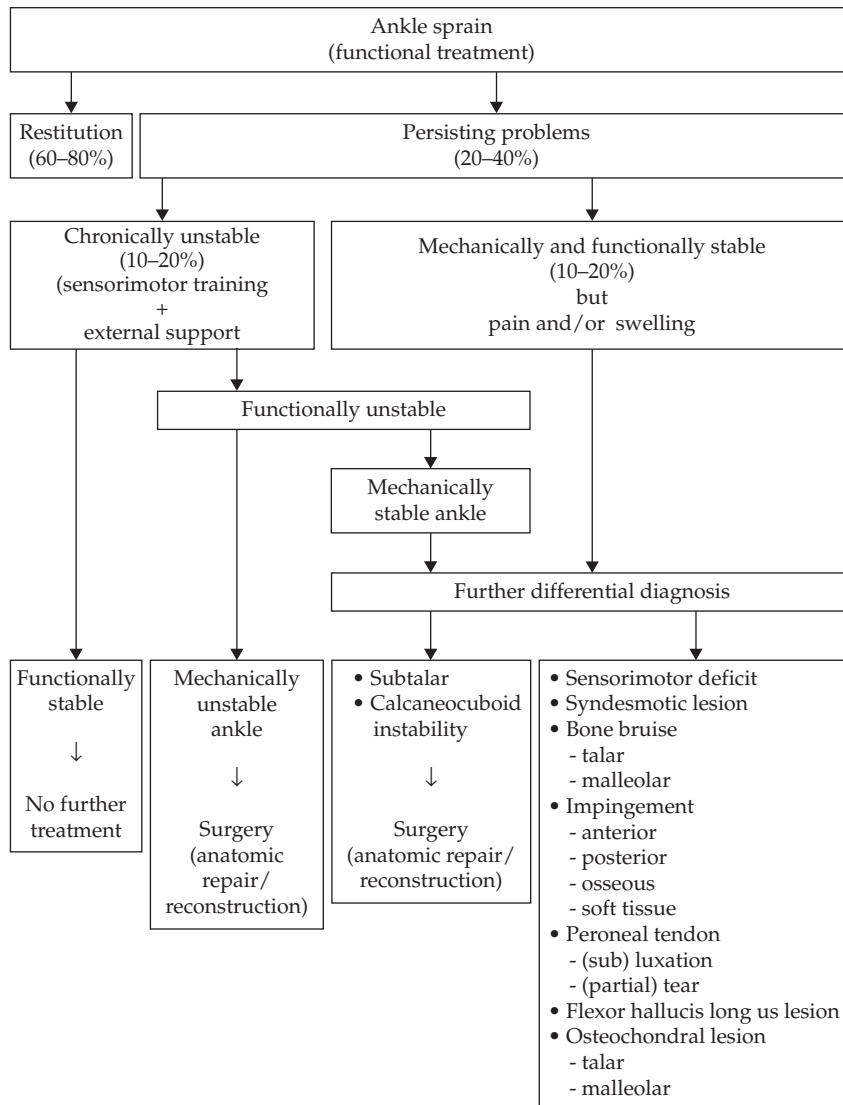


Figure 16.9 Flow chart demonstrating the different diagnostic pathways and the resulting treatment following an “ankle sprain”. (Revised with permission: Lohrer & Nauck, 2006 *Dtsch Z Sportmed* 57: 271–276.)

a slower return to work and sport compared with a semirigid ankle support (Kerkhoffs et al., 2002b).

Contrary to these findings, elite athletes took a higher risk of ankle reinjury when they suffered from a low-grade (and not from a high grade) acute lateral ankle sprain (Malliaropoulos et al., 2009). Ankle stability and frequency of reinjury seem to be significantly influenced by the activity level of

an athlete. Higher activity levels led to more instability and higher reinjury rates in 1-year follow-up study (Haraguchi et al., 2009).

Athletes require a rapid return to sports. This interest, however, is conflicting with the time needed to reestablish the ligaments' strength during the healing course. Currently, it is impossible to determine the actual capacity of a ligament to resist

load occurring during sport. The healing time of the lateral ankle ligaments after an acute ankle sprain was estimated to require at least 6 weeks to 3 months. Nevertheless, in numerous patients, objective mechanical laxity and subjective ankle instability persist following treatment of an acute ankle sprain (van Rijn et al., 2008).

Chronic lateral ankle instability frequently develops after an initial ankle sprain. To date, there is no sufficient evidence to recommend an operative or conservative procedure. Therefore, initial conservative treatment should be implemented and only cases suffering from persisting instability or pain should be regarded for surgical interventions (Ferran et al., 2009). These operations have to be planned carefully, as reasons different from mechanical ankle instability may produce persisting symptoms (Figure 16.9). Despite having a stable ankle, some patients present with giving-way produced by a mechanical instability originating in a different anatomic location likely the calcaneocuboid or the subtalar joint (Lohrer & Nauck, 2006). Furthermore, ankle pain may indicate intra-articular pathologies like osteochondral lesions or bone spurs. Freeman introduced the term “functional ankle instability” meaning a condition characterized by giving way despite a mechanically stable ankle (Freeman, 1965). Concluding from the concept presented earlier (Figure 16.9), it seems questionable whether a merely “functional” or “stable” instability really exists. It is at least possible that these conditions are unrecognized, when articular lesions do not originate from the ankle.

Regaining mechanical stability is the aim of surgery in chronically unstable ankles and methods for anatomic reconstructions are currently recommended (Ferran et al., 2009). Additionally, rebuilding of the previously reduced sensorimotor abilities can be expected as a result of anatomic reconstruction.

Preventive Measures

Braces and Tape

The review of older as well as current literature reveals that prophylactic ankle bracing or taping has consistently been shown to be effective in

reducing the incidence of ankle sprains (Dizon & Reyes, 2009). In an interesting study, a numbers-needed-to-treat analysis was applied to previously published studies. The authors found that protective effects are greater when prophylactic ankle taping or bracing was performed by athletes with a history of ankle sprains, compared with those having no history of previous sprains (Olmsted et al., 2004). Compared with elastic bandage, fewer patients reported instability at short-term follow-up when treated with a semi-rigid support (Relative risk 8.00) (Kerkhoffs et al., 2002b). Until now, there is no evidence that the prophylactic use of ankle braces or tape could cause muscle atrophy or any other negative side effects to the ankle.

Sensorimotor Training

Several studies show that specific balance training or multimodal training interventions including warm-up procedures and special instructions could reduce the incidence of ankle sprains.

The complex structure of the mechanisms of sports-related injuries has been addressed in several studies. Extrinsic and intrinsic factors have been identified to determine the actual cause of sports injuries. It has been shown that the adaptations of the neuromuscular system following SMT are highly specific and closely associated with alterations in motor control of the lower limb muscles. Recent studies provide evidence that SMT interventions are highly effective resulting in reduced injury incidences (Verhagen et al., 2004).

From a biomechanical point of view, balance is characterized by changes of the center of pressure with respect to the actual projection of the CoM to the supporting area during a distinct motor task. The ability of the nervous system to detect joint positions, movement directions, and force applications is mainly processed by proprioceptive afferents. In addition, precise information is necessary to balance gravitational forces. From postural control and balance, it is known that reflexes largely contribute to keep the center of pressure within the constraints of the supporting area.

Knee Ligaments

General Considerations

Knee ligaments are especially prone to injury in athletes younger than 30 years. A knee ligament injury is often a serious break in an athlete's career. Beside a long-lasting rehabilitation, recurrent instability, functional limitation, pain, and osteoarthritic development maybe initiated by such an event, and may eventually result in reduced sports performance and could lead to a sport career's end.

During the last 30 years, the knee ligament injuries have become a main concern among orthopedic surgeons. During this period, a bulk of literature has been produced dealing with biomechanics, rehabilitation, and surgical implications. Nevertheless, there exists a considerable lack of knowledge regarding the adequate management of an individual case.

Epidemiology

The ACL is the most frequently injured knee ligament. Interestingly, isolated ACL ruptures are comparatively rare. About 50% of 44 ACL ruptures have been associated with MCL ruptures (Fayad et al., 2003). For both genders, high-risk sports are soccer, football, handball, and alpine skiing. "ACL injury continues to be the largest single problem in orthopaedic sports medicine, with the incidence of non contact ACL tears being much higher in female athletes in team sports such as basketball and handball than in male athletes" (Renstrom et al., 2008).

In a prospective study, data were collected over a 14-year period from a total of 2989 athletes (1624 males and 1365 females) aged <50 years (Iwamoto et al., 2008). The most common injuries involved the ACL (14.3%) followed by non-ACL-related knee pain (13.7%), and by ankle sprains (9.4%). Among these types of sports injuries, a significant higher proportion of females engaged in basketball (24.4% vs. 10.5%), volleyball (20.5% vs. 4.5%), or skiing (41.4% vs. 26.5%) sustained an ACL injury, compared with their male counterparts.

Female athletes are at increased risk for certain sport-related injuries, particularly those involving the knee. Almost each epidemiologic study has found that females possess higher rates of noncontact ACL injuries compared to males.

Injury Mechanisms

Possible components of the ACL injury mechanism include combined anterior translation, dynamic valgus position, and knee extension. Low knee flexion in this respect is probably due to increased quadriceps and GAST muscle activity. These aspects can be examined when most or all of the acting force is placed on a single leg with the foot displaced away from the body's CoM, and when additionally trunk motion is increased (Renstrom et al., 2008).

Rehabilitational Concepts

Common practice and relevant research have agreed that both conservative and functional managements are important in treatment of isolated MCL tears (Edson, 2006).

A mail survey to the American orthopedic surgeons revealed surprisingly significant differences regarding whether ACL-deficient patients can participate in all recreational sports activities. The surgeons' opinions additionally varied considerably in their opinion that ACL reconstruction reduces the rate of knee arthrosis, and whether or not a post-operative brace should be used (Marx et al., 2003). In line with that study, the review of literature does not provide evidence that ACL reconstructions effectively reduce long-term symptoms, and even a decrease in the rate of knee osteoarthritis is questioned (Lohmander et al., 2007). Although it is not underlined by high level evidence, it is generally agreed that athletes suffering from chronic instability (recurrent giving way) are considered as candidates for ACL reconstruction aimed at restoring normal knee mechanics. A recently performed large cohort study, based on the Norwegian National Knee Ligament Registry (3475 patients with primary ACL reconstruction), supported this notion

and revealed that the odds of cartilage and meniscus lesions increased by nearly 1% and 0.5% per each month of delayed surgery, respectively. This clearly indicates that early surgery maybe associated with fewer cartilage and meniscal lesions (Granan et al., 2009).

Historically, ACL reconstructions were performed using the central third of the patellar tendon as a transplant. In the last decade, semitendinosus autografts became more popular and numerous fixation techniques have been developed. In a randomized controlled trial, subjective and objective outcomes were similar when comparing bone-tendon-bone patellar ligament and triple/quadruple semitendinosus autografts 7 years after ACL reconstruction. Additionally, no differences in donor-site morbidity could be found (Liden et al., 2007).

With respect to associated MCL tears present at early ACL reconstructions, the results were equally good, independent of operative MCL reconstruction or not (Halinen et al., 2006). This randomized controlled trial supports the previous practice suggesting that an isolated ACL reconstruction is a sufficient treatment to combat anteromedial complex knee instability.

Until today, different forms of exercises are generally implemented in rehabilitation of knee ligament injuries despite if surgery was performed or not. In a systematic review, however, there is no study available comparing the effect of exercise versus no exercise in knee rehabilitation. Comparing different exercises following surgery or during conservative treatment provided insufficient evidence of superior efficacy of one intervention over the other (Trees et al., 2007).

Summarizing those findings, there is no doubt that only refixing of a torn ACL is not sufficient to restore its function. Especially, preventive strategies for the uninjured knee and following knee ligament injuries have to be addressed in the future.

Preventive Measures

Braces

Ankle bracing is generally accepted. In contrast to this, the effectiveness of prophylactic knee bracing to prevent knee injuries is controversially

discussed. Especially in football, there is no evidence proving the efficacy of prophylactic knee bracing to reduce knee injuries (Pietrosimone et al., 2008).

Sensorimotor Training

A meta-analysis gives evidence that neuromuscular training decreases the risk of ACL injury (Hewett et al., 2005). Three out of the six training interventions demonstrated significant positive effects on ACL injury rates. From these data, it can be concluded that neuromuscular training maybe effective to reduce ACL injuries in female athletes if the training program contains plyometrics, balance, and strengthening exercises, and if the training sessions are performed more than one time per week for a minimum of 6 weeks. Those authors speculated that the plyometric training components of the three effective interventions influence the connective tissue, train the muscles, and the nervous system. So, proper technique and body mechanics during maneuvers dangerous to the knee joint like landing or cutting could be improved. A randomized controlled trial underlines that serious knee injuries could be prevented by neuromuscular training or warm-up programs (Pasanen et al., 2009).

A detailed description of SMT and its effects is given in chapter Control and Training of Posture and Balance, Chapter 14.

Conclusion

Tendons and ligaments are composed of the same connective tissue. Therefore, it is not astonishing that the principles of rehabilitation are similar. It is most important for postoperative and for conservative treatment that functional rehabilitation is superior to immobilization for a period of more than several days. Putting a progressive load to the healing structure according to its actual capacity seems to be the adequate stimulus for optimal tendon and ligament healing. To achieve that demand, functional orthotic treatment is generally accepted and its different product-specific features are a matter of actual and ongoing discussion in clinical and biomechanical research.

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Index

- Achilles tendinopathy, 287, 288
- Achilles tendon (AT), 66, 283
 - overuse injuries and Haglund's disease
 - definitions, 287
 - epidemiology, 287–8
 - rehabilitational concepts, 288
 - tears
 - epidemiology, 283
 - etiology, 284
 - extrinsic factors affecting AT, 284
 - intrinsic factors affecting AT, 284
 - posttreatment, orthotic device for, 284–5
 - rehabilitational concepts, 284
 - tensile stress, during rehabilitation, 285–7
- Achilles tendon force (ATF), 66, 140
 - in different sport activities, 180
 - in heel-toe running, 177, 178
 - hopping and jumping, 141
 - in normal locomotion and exercise, 179
 - passively induced stretch reflexes on, 26
 - peak ATFs
 - during cycling, 142, 143
 - during maximal counter movement (CMJ) and squatting (SJ) jumps, 141, 142
 - potentiation, during reflex-induced mechanical response, 29
 - response to stretch, 28
 - during walking at different speeds, 139
- Achilles tendon insertional tendinopathy, 287
- aerodynamic forces, 91, 92, 99
- aging and underlying neuromuscular factors, in sport performance, 270
 - factors affecting performance, 274
 - maximal running speed with aging, determinants of, 279–80
 - muscle mass, composition, and contractility, 276–8
 - muscle strength and power, 274–6
 - neural activation, 278–9
 - record performances, 271–4
- agonist muscles activation, change in, 227
 - motor unit level, 228–31
 - whole-muscle level, 227–8
- alpha motor neuron, 2, 4, 257
- alpine skiing, 78
 - carving techniques, biomechanics of, 81–6
 - dynamics, 78
 - forces while skiing a turn, 80–81
 - gliding across slope, 79–80
 - straight gliding down the fall line, 78–9
 - recreational skiing, muscle functions and muscle fatigue in, 86–9
- ankle bracing, 294, 296
- ankle extensors, 38, 63
- ankle inversion injury, 291
- ankle ligaments
 - chronic lateral ankle instability, 294
 - epidemiology, 290–91
 - general considerations, 290
 - injury mechanisms, 291–2
 - preventive measures
 - braces and tape, 294
 - sensorimotor training, 294
 - rehabilitational concepts, 292–4
- ankle sprain, 176, 290, 292, 293, 294
- ankle strategy, 256
- ankle taping, 294
- antagonist coactivation, changes in, 231–2
- anterior cruciate ligament (ACL), 167
 - displacement and strain measurements, 174
 - injury, 295
- anterior talofibular ligament (ATFL), 167, 291
- aponeuroses, 172
- apparent efficiency, 103
- “Armstrong's model”, 204
- average power, 217
- B-mode US, 168
- background EMG activity (BGA), 122
- balance disturbances, compensating, 255–6

- balance training, 254, 259
 - neural adaptations to SMT, 260–61
 - SMT Sequences, 259–60
 - strength and power, 261–6
- basal ganglia, for postural control, 258
- basic biphasic pattern, of SSC fatigue, 192–4
- biological tissues, neuromechanical loading of, *see* neuro-mechanical loading, of biological tissues
- biphasic SSC recovery, 206–7
- body mass index (BMI), 91
- brain stem, in postural control, 257
- braking phase
 - EMG activity during, 158
 - of muscle action, 105
 - of running, 17, 161
 - in SSC action, 107, 109
- buckle transducer, 136–8, 166–7
 - advantages and disadvantages, 142–3
 - in different locomotor activities, 138
- C-reactive protein (CRP), 204, 205
- carving techniques, biomechanics of, 81–6
- cerebellum, in postural control, 257–8
- cervicomedullary motor-evoked potential (CMEP), 123–4
- cervicomedullary stimulation (CMS), 123–4
- challenged populations, cycling in, 73–4
- chronic lateral ankle instability, 294
- circular coil, 117, 118
- coactivation, definition of, 231
- collagen fibers, 169, 171, 172
- concentric contraction, 218, 219, 221
- concentric muscle action, 6, 7, 16
 - versus eccentric muscle actions, 33, 104
 - electromyographic (EMG) force relationship in, 13
 - F–V relationship in, 10
 - models, 7
- connective tissues, 164, 169, 173, 185, 277
- constant load, 219
- “contractile mechanism”, 183
- contraction and loading technique, 218–19
- corticomotoneuronal system, anatomy of, 118
- corticosteroids, 288, 290
- counter movement jump (CMJ), 22, 141, 142, 144, 146
- coupling time, 17, 18, 19, 244
- cross-country skiing, 32, 49–50, 188, 189, 233, 270
 - SSC in, 33
 - EMG and GRF evidence, 36–40
 - kinematic evidence, 33–6
 - mechanical and metabolic power implications, 42–5
 - ski tester to simulate SSCs, 45–8
 - ultrasound measurements on muscle fascicles as evidence, 40–41
- cycling task, neuromechanics of, 52
 - ATF action, 142, 143
 - in challenged populations, 73–4
- kinematics, 53–5
- mechanical work and power, 59–60
- muscle mechanics
 - MTU and muscle fascicle length and velocity changes, 64–9
 - muscle activity patterns, 60–64
 - muscle moments, 58–9
 - neural control, 69–73
 - pedal reaction forces, 55–8
- cyclists with a transfemoral amputation (CTFA), 73–4
- cyclists with transtibial amputation (CTA), 73
- “damage–inflammation–repair” pathway, 204
- delayed onset muscle soreness (DOMS), 45, 184, 185, 186, 193–4, 207
- delta efficiency, 103
- differential variable reluctance transducers (DVRTs), 166, 167, 168
- direct force transducers, 166
- direct variables, of neuromechanical loading, 165
- DOMS, *see* delayed onset muscle soreness (DOMS)
- downhill skiing, *see* alpine skiing
- drag coefficient, 79
- drop jump (DJ), 19, 20, 109, 156, 157
 - breaking point in, 21
 - drop and rebound intensity-specific muscle behavior during, 159
 - fascicle–tendon interaction in, 158
 - short-latency reflex (SLR) response, 29
 - in SSC demonstration, 22
- dynamic movements, 154, 177, 178
- dynamometer arm, 185
- eccentric/concentric cycle (ECC), 33, 186
- eccentric contraction, 218, 219, 221, 239
- eccentric muscle action, 6, 16
 - and concentric muscle action
 - comparison between, 104
 - functional differences between, 33
 - electromyographic (EMG) force relationship in, 13
- eccentric training, 186, 288, 290
- “effectiveness”, definition of, 103
- electroencephalography (EEG), 258
- electromyographic (EMG) activity, 8, 136
 - in SSC prediction improvement, 36–40
- elite athletes, 270, 271
- EMG–force relationship, 104
- EMG patterns during propulsion, 61–3
- energy expenditure before, during, and after
 - marathon, 112
- excitatory postsynaptic potential (EPSP), 118, 128
- “extensor bottom transition muscle”, 72
- fascicle–tendinous behavior, in isolated muscle
 - actions, 152–3
- fascicle–tendon interaction, 203

- with aging and training, 161
- effects of push-off intensity on, 158
- and SSC task intensity, 156–8
- fast, fatigable (FF) type unit, 2
- fatigue responses
 - during exercise, 190–92
 - intermediate maximal and submaximal testing tasks, 192, 193
- fatigue-resistant motor unit, 2, 3, 222
- fibroblasts, 169, 170
- figure-of-eight coil, 117–18
- “final common pathway of the motor system”, 2
- Finnish veterans championships, 270
- FIS (Fédération Internationale de Ski), 91
- flattened tendons, 172
- foil strain gauge transducer, 166
- foot and ankle
 - of ligaments, 176–7
 - of muscles and tendons, 177–9
- “footballer’s ankle”, 291
- force, 216
- force–frequency relationship, 5
- force–length (F–L) relationship, 8–10, 174
- force potentiation, 15, 16
- force–time (F–T) characteristics, 7–8
- force–time (F–T) relationships, 5
- force transmission, changes in, 244
 - lateral force transmission, 245
 - muscle–tendon and joint stiffness, 244
 - tendon–aponeurosis characteristics, 244–5
- force–velocity (F–V) relationship, 10–13, 174, 219
 - classical, 23
 - instantaneous, 23, 24
- functional ankle instability, 294
- functional imaging (fMRI), 233
- functional rehabilitation, 284
- fusimotor-muscle spindle function, specific
 - alteration in, 206
- gamma-aminobutyric acid (GABA), 118, 125
- gastrocnemius (GAST) muscle, 9, 24, 64–70, 74
- gliding across slope, 79–80
- gross efficiency, 103
- ground reaction force (GRF), 29, 108
 - and knee angles of parallel (skidded) turns, 82
 - of skis and poles, 36, 37
- Haglund’s disease, 287, 288
- Hall effect strain transducers (HESTs), 166, 167, 175
- Hall sensors, 166
- Hill curve, 12, 23
- Hill’s classical model, 152
- hip strategy, 256
- Hoffmann reflex (H-reflex), 122, 124, 125, 233, 234
 - conditioning by subthreshold TMS, 128–9
 - excitability, 30
 - stimulations, 122, 124, 126
- hopping and jumping, ATFs in, 141, 142
- hopping training, 20
- “hybrid” fibers, 276
- hyperplasia, 241
- hypertrophy mechanisms, of muscles, 240–41
- implantable force transducers, 166, 167
- in-vivo human tendon force measurements, 135
 - clinical relevance and future challenges, 147, 148
 - methodological development, 136
 - buckle transducer, 136–8, 142–3
 - cycling, 142
 - hopping and jumping, 141
 - long jump, 141–2
 - walking and running, 138–40
 - during natural human locomotion, 138
- OF Technique, 143
 - forces, examples of, 144–6
 - pros and cons, 146–7
- indirect force transducers, 166
- injury mechanisms
 - of ankle ligaments, 291–2
 - of knee ligaments, 295
- instantaneous F–V curves, 145, 146, 174
- insulin-like growth factor-1 (IGF-1), 240
- interleukin 6 (IL-6), 204, 205
- intermediate maximal and submaximal testing
 - tasks, 192, 193
- internal tendons, *see* aponeuroses
- intracortical facilitation (ICF), 125–6
- invasive percutaneous suturing, 284
- ipsilateral TMS, 127–8
- isometric and concentric contraction, 219
- isometric contraction, 216, 218
- isometric muscle action, 6–7, 16
 - models, 7
- joint forces, moments of, 22
- joint ligaments, 169, 170
- joint muscle powers, calculating, 59, 60
- joint stiffness, 244
- jumper’s knee, *see* patellar tendinopathy
- kinematic arm, 110, 111
- knee joint
 - ligaments of, 174–6
 - muscles and tendons of, 177, 178
- knee ligaments, 295
 - epidemiology, 295
 - general considerations, 295
 - injury mechanisms, 295
 - preventive measures
 - braces, 296
 - sensorimotor training, 296
 - rehabilitational concepts, 295–6

- Lachman test, 175, 176
 lateral force transmission, 245
 leukocyte concentration, 205
 ligament talofibulare anterius (LTFA), 176
 ligaments, 174
 - ankle ligaments, 290
 - chronic lateral ankle instability, 294
 - epidemiology, 290–91
 - general considerations, 290
 - injury mechanisms, 291–2
 - preventive measures, 294
 - rehabilitational concepts, 292–4
 - direct insertion, 169
 - foot and ankle, 176–7
 - indirect insertions, 169–70
 - knee joint, 174–6
 - knee ligaments, 295–6
 - mechanical properties, 170–71
 - morphology and function, 169–70
- liquid metal gauge transducers, 166
- loading technique, 219
- locomotion and exercise, neuromechanical tissue
 - loading in, 174
 - ligaments, 174
 - foot and ankle, 176–7
 - knee joint, 174–6
 - muscles and tendons
 - foot and ankle, 177–9
 - knee joint, 177
- long intracortical inhibition (LICI), 125
- long jump, ATFs in, 141–2
- long-lasting SSC performances, 188–9
- magnetic stimulation, 116
- marathon, mechanical efficiency during, 112
- marathon run model, 191
- master athletes, sport performance in, 270
 - factors affecting performance, 274
 - maximal running speed with aging, determinants of, 279–80
 - muscle mass, composition, and contractility, 276–8
 - muscle strength and power, 274–6
 - neural activation, 278–9
 - record performances, 271–4
- masters' competitions, 270
- maximal and submaximal SSC testing conditions, 196–8
- maximal force, 216, 224, 225
 - and muscle activation, 194–6
- maximal RFD, 224–6
- maximal running speed with aging, determinants of, 279–80
- maximal versus endurance-type isometric tests, 200–201
- maximal versus submaximal SSC dynamic contractions, 201–2
- maximal voluntary contraction (MVC) force, 194, 195, 216, 221, 228, 229, 239
- maximal voluntary isometric contractions, 110
- mechanical efficiency (ME) of SSC exercise, 13, 103
 - EMG activity patterns, 110
 - human movements
 - background studies, 104
 - Jyväskylä studies, 104–7
 - during marathon, 112
 - total SSC exercise, 107–10
 - training adaptation, 111–12
- mechanical stress concept, 185
- “mechano growth factor”, 240
- medial collateral ligament (MCL), 171
- medial gastrocnemius (MG) muscle
 - behavior during running and walking, 155
 - fascicles, 26, 155, 156, 157, 158, 160
 - stiffness regulation, 160–61
 - ultrasonographic images of, 152
- moderate-volume training, 218
- motor cortex
 - in muscle function, 123
 - cervicomedullary stimulation, 123–4
 - H-reflex, 124
 - ipsilateral TMS, 127–8
 - SICI and ICF, 125–7
 - transcranial electrical stimulation, 123
 - for postural control, 258–9
 - sensory input at, 207–9
- motor unit
 - adaptation, training effects on, 235–6
 - functional significance, 2–4
 - impulses, synchronization of, 230
 - levels, 228–31
 - recruitment and rate coding, 222–4
 - synchronization, 229–30
- motor-evoked potentials (MEPs), 115, 118–20
 - comparison with other evoked potentials
 - cervicomedullary stimulation, 123–4
 - H-reflex conditioning by subthreshold TMS, 128–9
 - H-reflex stimulation, 124
 - SICI and ICF, 125–6
 - transcranial electrical stimulation (TES), 123
 - muscle activity effect on, 122
- mTOR pathway, 240
- muscle, contractile properties of, 235–6
- muscle action, types of, 6–7
- muscle activation, maximal capacity for, 224
 - maximal force, 224, 225
 - maximal RFD, 224–6
- muscle activity changes, during 3h skiing session, 88
- muscle activity patterns, during pedaling cycle, 60–64

- EMG patterns, 61–3
 - SOL and GAST activity, 63–4
- muscle architecture modification, by strength training, 239
- muscle atrophy, 277
- muscle control by the nervous system, 1
- muscle coordination, change in, 232
- muscle fascicle–tendon model, 153
- muscle fiber adaptations, 239–40
- muscle mass, 238–9
 - age-related loss of, 276–8
- muscle mechanics
 - force–length relationship, 8–10
 - force–time characteristics, 7–8
 - force–velocity relationship, 10–13
 - muscle action, types of, 6–7
- muscle moments, during pedaling cycle, 58–9
- muscle power, 216–17
 - in aging, 274–6
- muscle size and strength, relation between, 236–8
 - association between, 236
 - specific tension, 236–8
- muscle stiffness, 25, 174
- muscle strength, 216
 - in aging, 274–6
- muscle–tendon complex (MTC), 23, 146
- muscle–tendon mechanics, 4–6
- muscle–tendon stiffness, 244
- muscle–tendon unit (MTU), 6
 - length change pattern, 64–9
- muscle tissue, 169
- “muscle wisdom”, 205
- muscles
 - mechanical properties, 173–4
 - morphology and function, 173
- muscles and tendons
 - foot and ankle, 177–9
 - knee joint, 177
- muscular adaptations, 235
 - muscle and motor unit contractile properties, 235–6
 - muscle fiber adaptations, 239–40
 - muscle size and strength, relation between, 236–8
 - whole muscle mass and architecture, change in, 238–9
- muscular efficiency, 103
- MVO₂, 44
- MyHC isoforms, 276, 277
- MyHC-IIa fibers, 276
- MyHC-IIx fibers, 276
- naproxen, 288
- “negative work”, 42, 45, 106, 107
- net efficiency, 103
- neural and mechanical recoveries, parallelism
 - between, 194
- neural activation
 - in master athletes, 278–9
 - reflex adjustments of, 206–7
- neural adaptations, 226
 - agonist muscles activation, change in, 227
 - motor unit level, 228–31
 - whole-muscle level, 227–8
 - antagonist coactivation, changes in, 231–2
 - coordination between muscles, changes in, 232
 - potential locations for, 232
 - spinal level, 233–4
 - supraspinal level, 232–3
 - to sensorimotor training, 260
 - spinal adaptations, 260–61
 - supraspinal adaptations, 261
- neural mechanisms, in SSC-type fatiguing
 - exercises, 205
- fusimotor-muscle spindle function, specific
 - alteration in, 206
- motor cortex level, influence of sensory input at, 207–9
- neural activation, reflex adjustments of, 206–7
- neuromechanical loading, of biological tissues, 164
 - and mechanical properties
 - morphology, 169–74
 - normal locomotion and exercise, neuromechanical tissue loading in, 174–80
 - methods, 165–9
 - variables of, 165
- neuromuscular adaptation, 235, 266
- neuromuscular fatigue, 112
 - during isolated eccentric actions, 184–6
- NSAIDs (nonsteroidal anti-inflammatory drugs), 288
- OF transducer, 136, 167
- Ohm’s law, 222
- one- and two-joint muscles, motor neuron pools of, 71
- one-legged cycling, 72
- 1-repetition maximum (1-RM), 220
- onset of blood lactate accumulation (OBLA), 45
- open surgical intervention, 284
- optic fiber (OF) technique, 136, 143, 177
 - forces, examples of, 144–6
 - pros and cons, 146–7
- optical dimension analysis, 166
- overload principle, 217
- overuse tendon injuries and ligament failures,
 - rehabilitation of, 283
- Achilles tendon (AT), 283
 - AT tears, 283–7
 - Haglund’s disease, 287–8
- ankle ligaments, 290–94
- knee ligaments, 295–6
- patellar tendinopathy, 288–90
- oxygen consumption (VO₂)–power relationship, 104

- paracetamol, 288
- passive reflex testing conditions, 198
- passive tension, 2, 174
- patella tendon force (PTF), 144, 145, 146
- patellar tendinopathy, 288
 - definition, 288–9
 - epidemiology, 289
 - etiology, 289
 - proximal patellar tendon, in vivo evaluation of load distribution in, 290
 - rehabilitational concepts, 289–90
- peak force reduction (PFR), 197
- peak power, 12, 13, 217
- pedal reaction forces, 55–8
- pedaling cycle, 55, 65, 73
 - angular displacement of the hip and knee for, 54
 - joint muscle powers during, 59–60
 - major skeletal muscles and ATF measurement during, 143
 - mean joint angles for ankle, 55
 - muscle moments during, 58–9
- performance deterioration and recovery, with exhaustive SSC exercise, 203
 - neural changes, potential sources of, 205
 - fusimotor-muscle spindle function, specific alteration in, 206
 - motor cortex level, influence of sensory input at, 207–9
 - neural activation, reflex adjustments of, 206–7
 - structural changes and subsequent inflammation/remodeling process, 204–5
- performance determination in master athletes, factors affecting, 274
 - maximal running speed with aging, determinants of, 279–80
 - muscle mass, composition, and contractility, 276–8
 - muscle strength and power, 274–6
 - neural activation, 278–9
- performance potentiation, in SSC, 17–21
- phase-contrast magnetic resonance imaging (PC-MRI), 168, 177
- plyometrics, 219, 220, 245
- position sense test, 203
- positive work
 - efficiency of, 42
 - mechanical efficiency of, 106–7
- positron emission topography (PET), 258
- POST testing, 197
- posterior cruciate ligament (PCL), 167, 168
- postoperative rehabilitation, 284
- postural control, 254
 - and information processing, 256
 - basal ganglia, 258
 - brain stem, 257
 - cerebellum, 257–8
 - motor cortex, 258–9
 - spinal cord, 257
 - anticipation mode, 254
 - balance disturbances, compensating, 255–6
 - feedback mode, 254
 - undisturbed stance, organization of, 255
- postural flexibility, 258
- power-related properties, changes in, 241–3
 - calcium-related changes, 243–4
 - MyHC composition, 243
 - myosin ATPase activity, 243
- presynaptic inhibition (PSI), 210, 257
- progressive overload, *see* progressive resistance exercise
- progressive resistance exercise, 217
- prostaglandin E2 (PG E2), 204
- protein kinase B, 240
- proximal patellar tendon, 290
- push-off intensity
 - effects on fascicle–tendon interaction, 158
 - effects of variation of, 158
- push-off phase, 105
 - of muscle action, 105
 - in running, 17
- rate of force development (RFD), 217, 263, 264
 - and speed of movement, 219
- reactive jump abilities, and SMT, 263–6
- record performances, 271–4
- recreational skiing, 86–9
- rehabilitational concepts
 - of ankle ligaments, 292–4
 - of AT tears, 284
 - of Haglund's disease, 288
 - of knee ligaments, 295–6
 - of patellar tendinopathy, 289–90
- repeated SSC loading, fatigue definition applied to, 186–7
- resistance training, 232
- resting motor threshold (rMT), 116, 120
- resting tension, 2
- RFD, and speed of movement, 219
- running
 - braking phase of, 17
 - push-off phase of, 17
 - SSC of triceps surae muscle in, 16
- running, in vivo tensile measurements in, 138–40
- satellite cells, 240
- seat height, 53–4, 66
- seated cycling, 66
- sense of position and velocity, delayed influence on, 202–3
- sensorimotor integration, 258
- sensorimotor training (SMT), 259, 294, 296

- neural adaptations, 260
 - spinal adaptations, 260–61
 - supraspinal adaptations, 261
- sequences, 259–60
- strength and power, 261
 - neuromuscular adaptation, specificity on, 266
 - reactive jump abilities, 263–6
 - strength and power adaptations, 262–3
- serum creatine kinase (S-CK) activity, 204, 205
- Sharpey's fibers, 172
- short intracortical inhibition (SICI), 125–6
- short- and long-term SSC fatiguing protocols, 188–90
- short-latency stretch reflex (SLR), 23, 26, 28, 160
- short-range elastic stiffness (SRES), 140
- "short-range stiffness", 16
- short-term SSC exercise protocols, 189–90
- silent period (SP), 122–3
- simulated takeoffs, 98
- "size principle", 222
- skeletal muscle, 2, 4, 6
 - force and power potential of, 15
 - mechanical properties, 169
 - morphology and function, 173
- ski jumping, kinetics and muscular function in, 91
 - angular momentum, 93–4, 95, 98–9
 - BMI rule, effect of, 91
 - optimal aerodynamic position after takeoff, 95
 - simulated takeoffs, 98
 - take-off technique
 - duration, 99
 - kinematic characteristics of, 92–5
 - kinetics, 95–101
 - wind tunnel experiments, 99–101
- ski tester, to simulate SSCs, 45–9
- skidding angle β , 84, 85
- sledge jump, 108–9
- soft connective tissue, mechanical properties of, 170–71
- soleus (SOL) muscles, 23
 - mean length, velocity, and EMG for, 67
 - muscle stiffness versus force in, 25
 - rectified and averaged EMG pattern of, 29
- sonomicrometer, 168–9
- "specific tension", 236–8
- speed-related properties, changes in, 241–3
 - calcium-related changes, 243–4
 - MyHC composition, 243
 - myosin ATPase activity, 243
- "spike-triggered averaging", 236
- "spike triggers", 236
- spinal adaptations, associated with SMT, 260–61
- spinal cord, 257, 262
- spinal-level adaptation, 233–4
- spinal stretch reflex, 233, 234
- squatting jump (SJ), 22, 141, 142
- stationary bicycles, 52, 175
- strain gauge transducers, 166
- strength and power training
 - main parameters for, 218
 - contraction and loading technique, type of, 218–19
 - magnitude, of load, 218
 - RFD and speed of movement, 219
 - training volume, 218
 - muscle power, 216–17
 - muscle strength, 216
 - overload principle, 217
 - rate of force development (RFD), 217
 - training effects, specificity of, 219–22
- stretch- and H-reflex responses, 198–200
- stretch reflex activity, during normal dynamic actions, 160–61
- stretch reflexes, in SSC, 23
 - functional significance, 24, 27–8
 - intervention, 23–4
 - during treadmill running, 24
- stretch-shortening cycle (SSC), 6, 7, 13, 154, 156
 - braking phase, 17
 - in cross-country skiing, 32–3, 49–50
 - EMG and GRF evidence, 36–40
 - kinematic evidence, 33–6
 - mechanical and metabolic power implications, 42–5
 - ski tester to simulate SSCs, 45–9
 - ultrasound measurements on muscle fascicles as evidence, 40–41
- definition, 16
- demonstration, 21–2
- effective SSC action, 23
- exercise, mechanical efficiency of, 103
 - human movements, 104–7
 - total SSC exercise, 107–10
 - training adaptation, 111–12
- and fascicle–tendon interaction, 156–8
- instantaneous force–velocity relationship during, 23, 24
- in locomotion, 17
- performance potentiation in, 17–21
- push-off phase, 17, 158
- stretch reflexes, 23–4, 24–8
- task-dependent modulation of reflex gain, 28–9
- stretch-shortening cycle (SSC) fatigue, 183
 - experimental settings
 - short- and long-term protocols, 188–90
 - testing problematics, 190–92
 - functional influences
 - basic biphasic pattern, 192–4
 - contralateral influence, 202
 - fascicle–tendon interaction, 203
 - sense of position and velocity, delayed influence on, 202–3

- stretch-shortening cycle (SSC) fatigue (*continued*)
 “testing-task” influence on activation pattern, 200–202
 “time-dependent” effect, 194–200
 neuromuscular fatigue, during isolated eccentric actions, 184–6
 performance deterioration and recovery, with
 exhaustive SSC exercise, 203
 neural changes, potential sources of, 205–9
 structural changes and subsequent
 inflammation/remodeling process, 204–5
 repeated SSC loading, fatigue definition applied to, 186–7
 striated muscle, 169
 submaximal running, 16–17
 supraspinal adaptations, to SMT, 261
 supraspinal level adaptation, 232–3
 “suprathreshold stimulation”, 120
 take-off technique, of ski jumpers, 91
 duration, 99
 kinematic characteristics of, 92–5
 kinetics, 95–101
 wind tunnel experiments, 99–101
- tendinous tissues (TTs), 10, 146, 147
 tendon–aponeurosis stiffness, 244–5
 tendon length, definition of, 153
 tendon stiffness, 244, 277
 tendons
 mechanical properties of, 172–3
 morphology and function of, 171–2
 tensile stress
 in normal locomotion and exercise, 179
 “testing-task” influence, on activation pattern, 200
 maximal versus endurance-type isometric tests, 200–201
 maximal versus submaximal SSC dynamic contractions, 201–2
 “time-dependent” effect, 194
 maximal and submaximal SSC testing conditions, 196–8
 maximal force production and muscle activation, 194–6
 stretch- and H-reflex responses, 198–200
 “top transition-extensor muscle”, 72
 torque, 216
 training adaptation, of neuromuscular system, 216
 motor unit recruitment and rate coding, 222–4
 muscle activation, maximal capacity for, 224–6
 muscular adaptations, 235
 force transmission, changes in, 244–5
 hyperplasia, 241
 hypertrophy mechanisms, 240–41
 muscle and motor unit contractile properties, 235–6
 muscle fiber adaptations, 239–40
 muscle size and strength, relation between, 236–8
 speed- and power-related properties, changes in, 241–4
 whole muscle mass and architecture, change in, 238–9
 neural adaptations, 226
 agonist muscles activation, change in, 227–31
 antagonist coactivation and coordination between muscles, changes in, 231–2
 potential locations for, 232–4
 strength and power training
 main parameters for, 218–19
 muscle power, 216–17
 muscle strength, 216
 overload principle, 217
 rate of force development (RFD), 217
 training effects, specificity of, 219–22
 transcortical longloop reflexes, 258
 transcranial electrical stimulation (TES), 115, 123
 transcranial magnetic stimulation (TMS), 115, 258
 during movement, 129–30
 physiological background, 118
 corticomotoneuronal system, anatomy of, 118
 effect of muscle activity on MEP, 122
 input–output relationship, 120–22
 motor cortex role in muscle function, evaluation of, 123–9
 motor-evoked potential, 118–20
 silent period (SP), 122–3
 technical background for, 116–18
 trunk extensors (ES), 38
 turn radius, 81
 two-legged cycling, 72
 type I collagen, 170, 172
- ultramarathon run, 188
 ultrasound (US), 150
 dynamic movements including SSC actions, 154
 fascicle–tendinous behavior during isolated muscle actions, 152–3
 fascicle–tendon interaction
 with aging and training, 161
 effects of push-off intensity on, 158
 and SSC task intensity, 156–8
 historical development, 150–51
 impact and push-off intensities, effects of
 variation of, 158
 measurements, 40–41
 movement specificity, 154–6

- muscle specificity, 158–60
- musculoskeletal US, 151
- stretch reflex activity during normal dynamic actions, 160–61
- undisturbed stance, organization of, 255
- variable load training, 219
- Vario-Stabil boot, 285
- vertical ski forces, 188
- walking, 22, 138, 139, 155–6
- whole muscle mass, change in, 238–9
- whole-muscle level, 227–8
- work efficiency, 103
- work–velocity relationships, 18, 19
- World Masters Track and Field Championships, 270
- Young’s modulus, of tendon, 172, 173