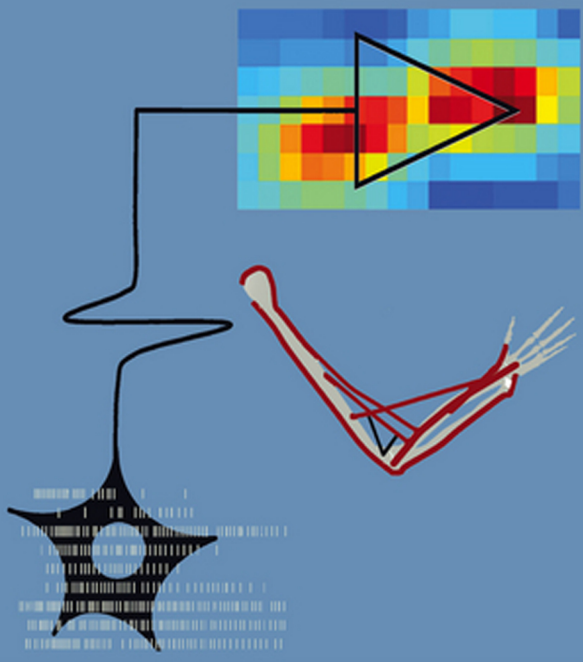
Surface Electromyography

PHYSIOLOGY, ENGINEERING, AND APPLICATIONS

Edited by
ROBERTO MERLETTI AND DARIO FARINA



IEEE Press Series in Biomedical Engineering
Metin Akay, Series Editor

IEEE Engineering in Medicine and Biology Society, Sponsor

IEEE PRESS

WILEY

SURFACE ELECTROMYOGRAPHY

IEEE Press
445 Hoes Lane
Piscataway, NJ 08854

Technical Reviewers

Philip A. Parker, *University of New Brunswick*
Dejan Popović, *University of Belgrade*
Cathi Disselhorst-Klug, *RWTH Aachen University*

SURFACE ELECTROMYOGRAPHY

Physiology, Engineering, and Applications

Edited by

ROBERTO MERLETTI
DARIO FARINA

IEEE Engineering in Medicine and Biology Society, *Sponsor*

IEEE Press Series in Biomedical Engineering
Metin Akay, *Series Editor*

Library of Congress Cataloging-in-Publication Data is available.

ISBN: 978-1-118-98702-5

Printed in the United States of America

10 9 8 7 6 5 4 3 2 1

CONTENTS

INTRODUCTION

In 2004, the book *Electromyography: Physiology, Engineering and Noninvasive Applications*, edited by R. Merletti and P. Parker, was published by IEEE Press and Wiley-Interscience. After more than a decade from that publication, the techniques and the equipment adopted in the study of muscles and muscle signals, by means of surface electrodes, underwent major advances. New tools are available for the detection, processing, and interpretation of surface electromyographic (sEMG) signals, new experience and knowledge have been acquired in the field, and new applications are now possible. These advances are related to electrode arrays and "EMG Imaging" techniques, signal amplifiers, signal transmission, EMG decomposition, as well as to many applications of these methodologies.

For many reasons, this work is not a second edition of the 2004 publication but rather a completely new book. First, it focuses only on surface EMG and not on invasive methods. Second, although it still provides the basic background, it emphasizes the new developments on grid recordings and EMG imaging in several applications. In this perspective, some topics discussed in the previous book have been eliminated while new chapters have been added.

The technical progresses in signal sensing, conditioning, processing, and interpretation techniques, however, have not always been exploited in the applied fields. Clinical applications of new methodologies are still lagging, mostly because of insufficient activities in technology transfer and in education/training efforts. The gap between researchers and practitioners widened in the last decade because of the acceleration of research and the inertia of educational and clinical institutions. This issue requires attention by research-supporting agencies at the European and national levels, especially when economical restrictions limit the spread of innovations.

OUTLINE OF THE BOOK

The areas concerning basic physiological and biophysical issues overlap with those of the book published in 2004, although new knowledge and new points of view are presented, especially in Chapters 1 and 3. Recent advanced approaches for signal detection are illustrated in Chapter 3, which also deals with the issue of electrode–skin interface (impedance and noise), while signal processing approaches for single channel EMG are described in Chapter 4.

One of the relevant new developments of the last decade is the technology of two-dimensional EMG (2D-EMG) or EMG imaging, based on electrode grids. A similar technology was developed earlier for EEG to facilitate its interpretation. 2D-EMG (or high-density EMG, HDEMG) provides a wealth of anatomical and physiological data concerning the muscle(s) below the electrode grid, including information about the innervation zone, the recruitment, de-recruitment, discharge rate, and conduction velocity of the detected motor units.

A wide spectrum of new applications in rehabilitation and movement sciences is opened up by this technology, described in Chapter 5, including ergonomics, occupational medicine, posture analysis, obstetrics, and new forms of biofeedback and rehabilitation training, described in the last 11 chapters.

A long-lasting theory in motor control is based on the modular organization of spinal neuronal networks, which can be identified by the analysis of sEMG. Muscular activation patterns, associated with a large number of movements, appear to be based on a limited number of fundamental patterns (basis functions) whose linear combinations produce such movements which are therefore defined by the weights of the linearly combined basis functions. Chapter 6 illustrates this concept and its applications.

The signals obtained from geometrically different viewpoints of the signal sources in the muscle comprise the inputs to unscrambling algorithms designed to identify and separate the contributions of the individual sources. This process is referred to as decomposition of the sEMG and is described in Chapter 7. It provides a window not only on the muscle but also on the control mechanisms and driving signals provided by the spinal cord networks to the muscle(s).

Mathematical modeling of sEMG is an important research and teaching tool for acquiring and transferring knowledge and for answering questions such as "what if . . .?" that cannot be answered by experiments. Testing new signal processing algorithms and defining their performance and limitations is another important application of the models described in Chapter 8.

Surface EMG data experimentally recorded from the major superficial muscle groups have been successfully used as a direct input drive to musculoskeletal models of human limbs. These models were demonstrated to be an effective way to predict muscle dynamics and joint moments in both healthy and pathological subjects and are described in Chapter 9.

Chapter 10 deals with myoelectric manifestations of muscle fatigue, which is one of the earliest fields of application of surface EMG and also the most treacherous. The statement made more than 20 years ago by Professor Carlo J. De Luca (Wartenweiler Memorial Lecture, International Society for Biomechanics, 1993)—"To its detriment,

electromyography is too easy to use and consequently too easy to abuse."—is still very much true in this field, despite the advances made in the last decade.

Muscles can be activated voluntarily or by electrical stimulation. In the second case the discharge frequency and the number of motor units are respectively controlled by the frequency and the amplitude of the stimulation pulses, thereby reducing a number of confounding factors and allowing external control of these parameters. Chapter 11 describes the electrical stimulation technique and the muscle features that can be investigated with it.

The chapters that follow deal with clinical applications of the techniques described in the previous 11 chapters. The list is certainly not comprehensive. Chapter 12 deals with neurophysiological investigations, with particular focus on reflex studies, while Chapter 13 addresses the applications in ergonomics and occupational medicine whose social and economic relevance are substantial.

Chapter 14 addresses applications in obstetrics and proctology and the issue of prevention of iatrogenic lesions due to episiotomy, a surgical intervention performed too frequently during child delivery and potentially increasing the likelihood of later fecal incontinence.

The issue of posture analysis is addressed in Chapter 15, which deals with monitoring the activity of the triceps surae during quiet standing. The muscles involved are pinnate, and the information provided by sEMG is different from that provided by muscles with fibers parallel to the skin.

Movement and, in particular, gait analysis is one of the fields with current clinical applications. This topic is addressed in Chapter 16 and is also a treacherous one because, in dynamic situations, the movement of the muscle under the skin causes sEMG alterations which reflect geometrical changes too often wrongly attributed to neurophysiological factors.

Physical therapy is the main field of sEMG application where the technique is used to plan and monitor treatment and assess its effectiveness. Timing, amplitude, and distribution of muscle activities, as well as monitoring of fatigue and of changes due to rehabilitation treatments, are the issues presented in Chapter 17. Chapter 18 expands the issue of sEMG biofeedback, which is gaining interest because of the recent sEMG imaging techniques.

Exercise physiology and sports is another important field of sEMG application. The issues of co-activation, muscle timing, and characterization of exercise are analyzed in Chapter 19 with focus on muscle coordination. Chapter 20 describes the use of surface EMG in man–machine interfacing for rehabilitation technologies. Examples of these applications include active prostheses and orthoses.

Other applications of more limited current clinical relevance, such as in space medicine, yoga relaxation studies, and other fields, are not discussed in this book.

OPEN TECHNICAL AND SCIENTIFIC ISSUES

Despite recent progress, sEMG technology is still developing and relatively far from being perfected. As the number of electrodes increases, the cable connection between

the electrodes, the amplifiers, and the computer becomes more problematic because of the high rate of information transfer that is required. The availability of gloves or sleeves with up to a few hundred electrodes covering a limb is very near in the future. The development of thin multi-lead connections between electrodes and amplifiers, with reduced movement artifacts, is a challenge being addressed. A possible solution is the incorporation of battery-powered amplifiers and wireless transmitters into such sleeves.

At the moment, wearable (pocket-size) devices with thin and wide-band fiber-optic connections (up to 50 m long) to a PC are commercially available, but a wireless connection is obviously preferred. In this case the bandwidth limitation is a bottleneck constraining the number of channels. A number of solutions are being considered in research laboratories. Since the signals to be transmitted are highly correlated, compression is a possible solution to reduce the bit rate. This approach has been investigated by researchers who demonstrated that lossless compression can reduce the bit rate by ~60% whereas lossy compression can reduce it by >90% while maintaining a signal-to-noise ratio greater than 20 dB. An alternative approach is the use of a data logger with a removable memory card and wireless transmission of a fraction of the data (for example, 0.1 s every second) to allow the operator to check for signal quality during acquisition.

High-density arrays imply small electrodes whose electrode–skin impedance and noise increase as the contact surface decreases. Skin treatments and paste-less electrode technologies are being investigated to reduce both impedance and noise and increase wearability of electrode arrays. Noninvasive detection of sEMG from deep muscles is still an open problem.

The estimation of force produced by individual muscles and the sharing of the global load among agonists and antagonists muscles, as well as the issue of co-contraction, are still unsolved problems. This issue is still biasing the study of sEMG–force relationship because sEMG is measured from one or few muscles whereas force (or torque at a joint) is produced by many more muscles whose electrical activity, at this time, cannot be entirely detected by noninvasive techniques.

Estimating crosstalk among muscles, and compensating for it, is not a satisfactorily solved problem, although high-density sEMG is a promising tool to address it. The relatively poor repeatability of the surface EMG measures is also an unresolved issue, although also for this problem high-density EMG may be a good approach to the solution.

The absolute values of EMG amplitude often need to be normalized because of confounding factors that should be compensated for. However, despite the many approaches proposed for sEMG normalization, no consensus exists on the most appropriate normalization technique.

Most of the scientific investigations are still limited to isometric conditions that are, however, far from the natural functioning of the muscles in daily-life activities. Extensions of the advanced methods developed for EMG analysis to fully dynamic tasks is challenging and still at a preliminary stage of investigation.

Although methods for decomposing the sEMG into the constituent single motor unit activities have progressed substantially in the last decade, these approaches still

have limitations with respect to the number of conditions and muscles that can be analyzed.

EDUCATION, TRAINING, AND STANDARDIZATION IN THE FIELD OF EMG IMAGING

Despite the availability of free teaching material on the web (www.lisin.polito.it, www.seniam.org, among others) and textbooks on the topic, very few Schools of Movement Sciences, Physical Therapy, or Rehabilitation Medicine include either traditional or advanced EMG technology in the training curricula of rehabilitation, sport, and occupational medicine. This fact results in limited user awareness of the potentialities of some of the new EMG tools available from research laboratories. This is a general problem which hinders clinical applications of sEMG, but its relevance is higher for multichannel sEMG because recently developed techniques are removing many of the problems that were pointed out, in the past, as limiting clinical applications of the technique. Clinical application of sEMG is much more limited by lack of dissemination than by technical limitations.

The issue of EMG best practice was addressed by the European Project "Surface Electromyography for Non-Invasive Assessment of Muscles (SENIAM, www.seniam.org)" whose recommendations (2000) are becoming outdated and do not include multichannel sEMG. A strong need is felt for upgrading recommendations to encompass recent advances. Activities in this direction were proposed in a special section of the 2014 Congress of the International Society for Electrophysiology and Kinesiology (ISEK).

FINAL REMARKS

The contributors to this book include only a very small number of senior members of the community of sEMG researchers. We chose them with the aim of merging as smoothly as possible, in the same book, physiology, engineering, and some important applications by providing suggestions and recommendations to the authors. We believe that all contributors did an excellent job in producing a harmonious result that can be appreciated by readers of different backgrounds. Any errors or failings of this work are certainly not attributable to the contributors but are strictly the responsibility of the editors.

ACKNOWLEDGMENTS

The Editors had the privilege of coordinating an excellent team of contributors and are greatly indebted to them for their efforts and results. They have devoted, without compensation, a considerable portion of their time to this endeavor.

The Editors owe a great debt to the reviewers who dedicated time and patience in reading this book. Their comments and criticisms have been of great help, not only for detecting a number of inconsistencies and drawbacks but also for placing the material in a better framework. Mrs. Antonietta Stango and Mr. Domenico Signorile greatly contributed to the preparation of the material for this book.

The book reports and disseminates knowledge that was in large part acquired within the following European and National Projects:

- "Surface Electromyography for Non-Invasive Assessment of Muscles (SENIAM)"
- "Prevention of Neuromuscular Disorders in the Use of Computer Input Devices (PROCID)"
- "Neuromuscular Assessment of the Elderly Worker (NEW)"
- "Decomposition of Multichannel Surface Electromyograms (DEMUSE)"
- "Cybernetic Manufacturing Systems (CyberManS)"
- "On Asymmetry in Sphincters (OASIS)"
- "Technologies for Anal Sphincter Analysis and Incontinence (TASI)"

- "Decoding the Neural Code of Human Movements for a New Generation of Man–Machine Interfaces (DEMOVE)"
- "A Novel Concept for Support to Diagnosis and Remote Management of Tremor (NeuroTREMOR)"

PROFESSOR ROBERTO MERLETTI

Laboratory for Engineering of the Neuromuscular System, Politecnico di Torino, Torino, Italy

PROFESSOR DARIO FARINA

Department of Neurorehabilitation Engineering, University Medical Center, Göttingen, Germany

CONTRIBUTORS

B. Afsharipour
Laboratory for Engineering of the Neuromuscular System, Politecnico di Torino, Torino, Italy

U. Barone
Laboratory for Engineering of the Neuromuscular System, Politecnico di Torino, Torino, Italy

S. Baudry
Laboratory of Applied Biology and Neurophysiology, ULB Neuroscience Institute (UNI), Université Libre de Bruxelles (ULB), Brussels, Belgium

T. F. Besier
Department of Engineering Science and Auckland Bioengineering Institute, University of Auckland, Auckland, New Zealand

G. Boccia
Motor Science Research Center, SUISM University of Turin, Turin, Italy; and CeRiSM Research Center "Sport, Mountain, and Health," Rovereto (TN), Italy

A. Botter
Laboratory for Engineering of the Neuromuscular System (LISiN) Politecnico di Torino, Torino, Italy

I. Campanini
LAM—Motion Analysis Laboratory, Department of Rehabilitation, AUSL of Reggio Emilia, Correggio, Italy

E. A. Clancy
Electrical & Computer Engineering Department; Biomedical Engineering Department, Worcester Polytechnic Institute, Worcester, Massachusetts

A. D'avella
Laboratory of Neuromotor Physiology, Santa Lucia Foundation, Rome, Italy
Department of Biomedical and Dental Sciences and Morphofunctional Imaging, University of Messina, Messina, Italy

J. Dideriksen
Department of Neurorehabilitation Engineering, Universitätsmedizin Göttingen, Georg-August-Universität, Göttingen, Germany

Jacques Duchateau
Laboratory of Applied Biology and Neurophysiology, ULB Neuroscience Institute (UNI), Université Libre de Bruxelles (ULB), Brussels, Belgium

Roger M. Enoka
Department of Integrative Physiology, University of Colorado, Boulder, Colorado

D. Falla
Institute for Neurorehabilitation Systems, Bernstein Center for Computational Neuroscience, University Medical Center Göttingen, Georg-August University, Göttingen, Germany
Pain Clinic, Center for Anesthesiology, Emergency and Intensive Care Medicine, University

D. Farina
Department of Neurorehabilitation Engineering, Bernstein Focus Neurotechnology Göttingen, Bernstein Center for Computational Neuroscience, University Medical Center Göttingen, Georg-August University, Göttingen, Germany

J. W. Fernandez
Department of Engineering Science and Auckland Bioengineering Institute, University of Auckland, Auckland, New Zealand

A. Gallina
Laboratory for Engineering of the Neuromuscular System, Politecnico di Torino, Italy
University of British Columbia—Rehabilitation Science, Vancouver, BC, Canada

M. GAZZONI
Laboratory for Engineering of the Neuromuscular System, Politecnico di Torino, Torino, Italy

A. HOLOBAR
Faculty of Electrical Engineering and Computer Science, University of Maribor, Maribor, Slovenia

Y. P. IVANENKO
Laboratory of Neuromotor Physiology, Santa Lucia Foundation, Rome, Italy

F. LACQUANITI
Laboratory of Neuromotor Physiology, Santa Lucia Foundation, Rome, Italy
Center of Space Biomedicine, University of Rome "Tor Vergata", Rome, Italy
Department of Systems Medicine, University of Rome "Tor Vergata", Rome, Italy

D. G. LLOYD
Centre for Musculoskeletal Research, Griffith Health Institute, Griffith University, Gold Coast, QLD, Australia

I. D. LORAM
Cognitive Motor Function Research Group, School of Healthcare Science, Manchester Metropolitan University, United Kingdom

M. M. LOWERY
School of Electrical & Electronic Engineering, University College, Dublin, Ireland

R. MERLETTI
Laboratory for Engineering of the Neuromuscular System (LISiN), Politecnico di Torino, Torino, Italy

A. MERLO
LAM—Motion Analysis Laboratory, Department of Rehabilitation, AUSL of Reggio Emilia, Correggio, Italy

M. A. MINETTO
Division of Endocrinology, Diabetology and Metabolism, Department of Medical Sciences, University of Turin, Turin, Italy

T. MORITANI
Professor of Applied Physiology, Graduate School of Human and Environmental Studies, Kyoto University, Sakyo-ku, Kyoto, Japan

F. NEGRO
Department of Neurorehabilitation Engineering, Bernstein Center for Computational Neuroscience, University Medical Center Goettingen, Goettingen, Germany

A. RAINOLDI
Professor of Physiology, Department of Medical Sciences, Motor Science Research Center, SUISM University of Turin, Turin, Italy

M. SARTORI
Department of Neurorehabilitation Engineering, Bernstein Focus Neurotechnology Göttingen, Bernstein Center for Computational Neuroscience, University Medical Center Göttingen, Georg-August University, Göttingen, Germany

D. F. STEGEMAN
Department of Neurology/Clinical Neurophysiology, Radboud University Nijmegen Medical Centre and Donders Institute for Brain, Cognition and Behaviour, The Netherlands

T. M. VIEIRA
Laboratory for Engineering of the Neuromuscular System, Politecnico di Torino, Torino, Italy, and Escola de Educação Física e Desportos, Universidade Federal do Rio de Janeiro, Rio de Janeiro, Brasil

D. ZAZULA
Faculty of Electrical Engineering and Computer Science, University of Maribor, Maribor, Slovenia

1

PHYSIOLOGY OF MUSCLE ACTIVATION AND FORCE GENERATION

R. M. Enoka[1] and J. Duchateau[2]

[1]*Department of Integrative Physiology, University of Colorado, Boulder, Colorado*
[2]*Laboratory of Applied Biology and Neurophysiology, ULB Neuroscience Institute (UNI), Université Libre de Bruxelles (ULB), Brussels, Belgium*

1.1 INTRODUCTION

To extract information about the control of movement by the nervous system from electromyographic (EMG) signals, it is necessary to understand the processes underlying both the generation of the activation signal and the torques exerted by the involved muscles. As a foundation for the subsequent chapters in this book, the goal of this chapter is to describe the physiology of muscle activation and force generation. We discuss the anatomy of the final common pathway from the nervous system to muscle, the electrical properties of motor neurons and muscle fibers, the contractile properties of muscle fibers and motor units, the concept of motor unit types, and the control of muscle force by modulating the recruitment and rate coding of motor unit activity.

1.2 ANATOMY OF A MOTOR UNIT

The basic functional unit of the neuromuscular system is the motor unit. It comprises a motor neuron, including its dendrites and axon, and the muscle fibers innervated by the axon [28]. The motor neuron is located in the ventral horn of the spinal cord or

Surface Electromyography: Physiology, Engineering, and Applications, First Edition.
Edited by Roberto Merletti and Dario Farina.

brain stem where it receives sensory and descending inputs from other parts of the nervous system. The axon of each motor neuron exits the spinal cord through the ventral root, or through a cranial nerve in the brain stem, and projects in a peripheral nerve to its target muscle and the muscle fibers it innervates. Because the generation of an action potential by a motor neuron typically results in the generation of action potentials in all of the muscle fibers belonging to the motor unit, EMG recordings of muscle fiber action potentials provide information about the activation of motor neurons in the spinal cord or brain stem.

1.2.1 Motor Nucleus

The population of motor neurons that innervate a single muscle is known as a motor nucleus or motor neuron pool [51]. The number of motor neurons in a motor nucleus ranges from a few tens to several hundred [40,58] (Table 1.1). The motor neuron pool for each muscle typically extends longitudinally for a few segments of the spinal cord (Fig. 1.1), and at each segmental level the pools for proximal muscles tend to be more ventral and lateral than those for distal muscles and the pools for anterior muscles are more lateral than those for posterior muscles [59]. Nonetheless, the extensive dendritic projections of motor neurons intermingle across motor neuron pools.

1.2.2 Muscle Fibers

The muscle fibers innervated by a single axon are known as the muscle unit (Fig. 1.1), the size of which varies across each motor unit pool. The motor units first recruited

TABLE 1.1 Motor Neuron Locations and Numbers for Selected Forelimb Muscles

Muscle	Spinal Location	Number
Biceps brachii	C5–C7	1051
Triceps brachii	C6–T1	1271
Flexor carpi radialis	C7–C8	235
Extensor carpi radialis	C5–C7	890
Flexor carpi ulnaris	C7–T1	314
Extensor carpi ulnaris	C7–T1	216
Extensor pollicis longus	C8–T1	14
Abductor pollicis longus	C8–T1	126
Flexor digitorum superficialis	C8–T1	306
Extensor digitorum communis	C8–T1	273
Flexor digitorum profundus	C8–T1	475
Extensor digiti secundi proprius	C8–T1	87
Abductor pollicis brevis and flexor pollicis brevis	C8–T1	115
Adductor pollicis	C8–T1	370
First dorsal interosseus	C8–T1	172
Lateral lumbricalis	C8–T1	57

Data are from Jenny and Inukai [58] and are listed as pairs of antagonistic muscles.

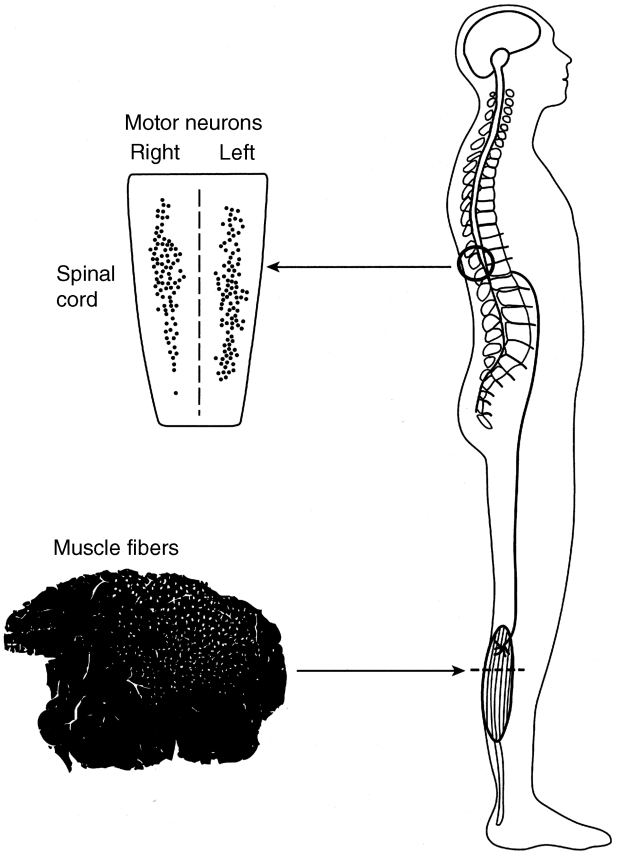


FIGURE 1.1 Muscle force is controlled by a population of motor units (motor unit pool) located in the spinal cord with each motor unit innervating a number of muscle fibers (muscle unit). The muscle fibers belonging to a single muscle unit are indicated by the white dots in the cross-sectional view of the muscle. A typical motor unit pool spans several spinal segments, and muscle units are usually limited to discrete parts of the muscle. Modified from Enoka [30] with permission.

during a voluntary contraction innervate fewer muscle fibers and hence have smaller muscle units than those that are recruited later in the contraction. Most motor units in a muscle have small muscle units and only a few have large muscle units [76,102,107] (Fig. 1.2A). Based on the association between muscle unit size and maximal motor unit force, Enoka and Fuglevand [32] estimated the innervation numbers (muscle unit size) for the 120 motor units in a human hand muscle (first dorsal interosseus) ranged from 21 to 1770 (Fig. 1.2B). Similar relations likely exist for most muscles [51]. Due to the exponential distribution of innervation number across a motor unit pool, it is necessary to distinguish between the number of motor unit action potentials discharged from the spinal cord and the number of muscle fiber action potentials recorded in the muscle with EMG electrodes. This distinction is indicated with the

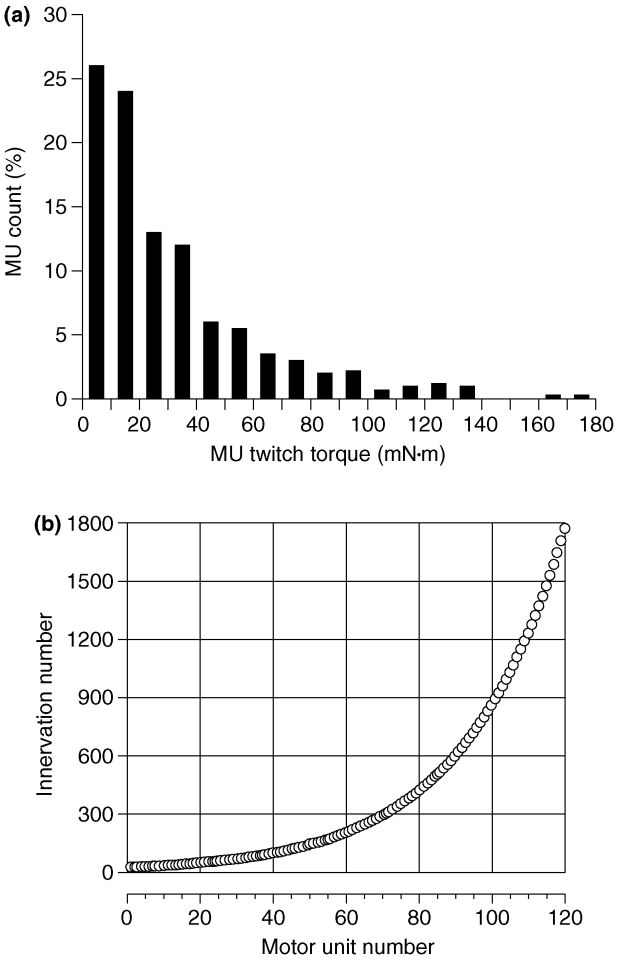


FIGURE 1.2 Variation in muscle unit size across the motor unit pool. (**a**) Distribution of motor unit (MU) twitch torques for 528 motor units in the tibialis anterior muscle of 10 subjects [107]. (**b**) Estimated distribution of innervation numbers across the 120 motor units comprising the first dorsal interosseus muscle [32].

term "neural drive" to denote the motor unit action potentials and "muscle activation" to indicate the muscle fiber action potentials [26,31,36].

The fibers in each muscle unit are located in a subvolume of the muscle and intermingle with the fibers of other muscle units (Fig. 1.1). The spatial distribution of the fibers belonging to a muscle unit is referred to as the motor unit territory. Counts of muscle unit fibers indicate that motor unit territories can occupy from 10% to 70% of the cross-sectional area of a muscle and that the density of muscle unit fibers ranges from 3 to 20 per 100 muscle fibers [51]. Moreover, the fibers of a single muscle unit often do not extend from one end of the muscle to the other, but instead terminate

within a muscle fascicle [48,108]. As a consequence of muscle unit anatomy, the forces generated by individual muscle fibers must be transmitted through various layers of connective tissues before reaching the skeleton and contributing to the movement. Such interactions attenuate the unique contribution of individual fibers to the net muscle force during a movement and thereby reduce the influence of differences in contractile properties among muscle fibers.

1.3 MOTOR NEURON

The motor unit is classically considered to be the final common pathway in that sensory and descending inputs converge onto a single neuron that discharges an activation signal to the muscle fibers it innervates [28]. The motor neuron has extensive dendritic branches that receive up to 50,000 synaptic contacts with each contact capable of eliciting inward or outward currents across the membrane and thereby generate an excitatory or inhibitory postsynaptic potential. The inputs are integrated and will generate an action potential in the trigger zone (axon hillock) when the change in membrane potential exceeds voltage threshold (Fig. 1.3). Motor neurons have four main types of receptors and ion channels that produce the responses to the synaptic inputs [50]:

1. *Leak Channels.* These primarily pass an outward K current and are largely responsible for establishing the resting membrane potential, which is approximately −70 mV in motor neurons.
2. *Voltage-Gated Channels.* These receptors are activated by a change in the membrane potential, such as activation of Na, K, and Ca channels by depolarization of the membrane. Na currents are the key elements in the generation of action potentials, and Ca-activated K channels are responsible for the afterhyperpolarization phase of the action potential.
3. *Ionotropic Synaptic Channels.* These are ligand-gated receptors that bind neurotransmitters and pass currents that produce excitatory or inhibitory postsynaptic potentials. Excitatory currents that depolarize the membrane potential are mainly produced by glutamate-gated receptors, whereas inhibitory currents that hyperpolarize the membrane typically involve either glycine- or GABA-gated receptors.
4. *Neuromodulatory Receptors.* Once a neurotransmitter binds to these receptors, they activate intracellular second messenger pathways that can modulate the function of leak, voltage-gated, and ionotropic channels. Neuromodulatory receptors, therefore, control motor neuron excitability by modulating its responsiveness to ionotropic input. Two neurotransmitters with potent neuromodulatory effects on motor neuron excitability are serotonin and noradrenaline.

The change in motor neuron membrane potential in response to the synaptic inputs it receives depends on the electrical interaction among its ion channels. These interactions can be characterized with Ohm's law: $V = I/g$, where V = potential difference across a

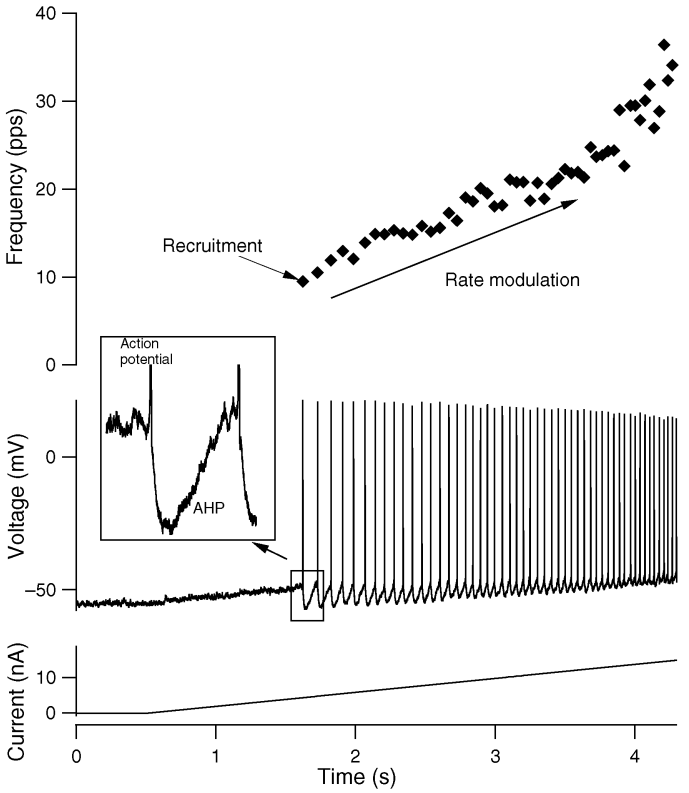


FIGURE 1.3 Relation between the current received by a motor neuron, the rate at which it discharges action potentials, and the force exerted by the muscle unit. (**Bottom trace**) The current injected into a motor neuron with a microelectrode. (**Middle trace**) The change in membrane potential (voltage) of the motor neuron in response to the progressive increase in injected current. When the change in membrane potential exceeds voltage threshold, the motor neuron is activated (recruitment threshold) and begins discharging action potentials. The inset shows the membrane potential trajectory between action potentials, which have been truncated to emphasize the afterhyperpolarization (AHP) phase. (**Upper trace**) Plot of the instantaneous discharge rate (pps = pulses per second) in response to the increase in current and the corresponding increase in the force produced by the muscle unit. Modified from Heckman and Enoka [50] with permission.

patch of the cell membrane, I = membrane current density (current per unit area), and g = input conductance (inverse of resistance) per unit area. The change in motor neuron membrane potential in response to a synaptic current varies with input conductance, which is largely determined by the number and size of its dendrites. Small motor neurons have the least extensive network of dendrites (low input conductance) and therefore experience the greatest change in membrane potential in response to synaptic current [50]. With similar values for voltage threshold among all motor neurons, the change in membrane potential in the trigger zone (axon hillock) will exceed the voltage

threshold with the least amount of synaptic current in small motor neurons, and therefore these neurons will be recruited first with progressively increasing synaptic input. Moreover, the rate at which action potentials are discharged by a motor neuron (rate coding) after it has been recruited (recruitment threshold) increases in direct proportion to the current it receives (Fig. 1.3).

The dendrites account for 95% of the surface area of a motor neuron and ~95% of the synaptic contacts occur on the dendrites. Ionotropic inputs to the motor neuron arise from the cerebral cortex, brain stem, and peripheral sensory receptors, but are transmitted via interneurons to the motor neurons. The key descending pathways that contribute to the control of movement include the corticospinal, rubrospinal, vestibulospinal, and reticulospinal tracts. The distribution of synaptic input within the dendrites of motor neurons is known for only a few systems [93]. Although excitatory input is generally greatest in the largest motor neurons and least in the smallest, recruitment order still proceeds in the order from smallest to largest motor neurons when combined with the intrinsic properties of motor neurons. As an exception to this pattern of input distribution, however, the excitation arising from the length detector in muscle—the muscle spindle—is greatest in the smallest motor neurons. In contrast, the inhibitory inputs studied to date often seem to generate approximately equal currents in all motor neurons.

Transmission of the postsynaptic potentials elicited by the synaptic currents to the trigger zone was initially assumed to occur passively by electrotonic conduction. More recent evidence, however, indicates that the postsynaptic potentials in the dendrites of motor neurons are augmented by the modulation of voltage-sensitive channels [51]. For example, motor neurons contain ~1000 to 1500 synapses with receptors that bind serotonin or noradrenaline and are capable of amplifying and prolonging synaptic inputs with persistent inward Ca and Na currents. Due to the profound influence of the persistent inward currents on the gain of relation between synaptic input and discharge rate [51], the neuromodulatory input is considered critical in defining the excitability of the motor neuron pool, including recruitment threshold [50]. Neuromodulatory input, for example, can amplify ionotropic input by as much as fivefold [8,49,55], which can saturate the capacity of the motor neuron to respond to further increases in synaptic input [56,68]. The function of these monosynaptic projections from the brain stem is likely to enhance motor neuron excitability during motor activity (serotonin) and conditions that require modulation of physiological arousal (noradrenaline).

1.4 MUSCLE UNIT

The muscle unit comprises the muscle fibers innervated by a single motor neuron and corresponds to the peripheral element of the motor unit. In a healthy person, the discharge of an action potential by a motor neuron invariably results in the activation of the muscle fibers it innervates. The force produced by a muscle unit largely depends on its innervation number, which varies exponentially within the motor unit pool (Fig. 1.2B), and average innervation numbers differ across muscles (Table 1.1). The

ability to grade force precisely during weak contractions likely depends on the size and number of small muscle units in the involved muscles. In contrast, the largest muscle units may only be engaged during rapid or powerful contractions.

1.4.1 Muscle Fiber Action Potentials

As nerve–muscle synapses typically provide secure transmission between a motor neuron and its muscle fibers, axonal action potentials invariably generate end-plate potentials that exceed voltage threshold and produce muscle fiber action potentials that engage the contractile proteins. Despite both the electrical signal (EMG) and the contractile activity (muscle fiber force) originating from the convolution of the neural drive to the muscle, the summation of the two signals diverges due to differences in the shape and sensitivity of each basic element. Because each action potential comprises positive and negative phases, the summation of multiple action potentials is algebraic. In contrast, the twitch, which is the force response of muscle to a single action potential, has only a positive phase and the summation of multiple twitches is only positive. Moreover, the shapes of the action potential and twitch are affected differently by changes in the physiological state of the neuromuscular system.

A muscle fiber is electrically similar to a large-diameter, unmyelinated axon and requires high transmembrane currents to propagate the action potential along the sarcolemma. The transmembrane current density associated with the action potential is proportional to the second derivative in the spatial domain of the potential recorded in extracellular space [97]. The currents underlying the propagation of action potentials along multiple muscle fibers sum to generate extracellular field potentials that can be readily detected with appropriate electrodes. The shapes of the recorded potentials depend on the properties and location of the electrodes and on the anatomy and physiology of the muscle fibers and associated tissues [37].

Each muscle fiber action potential begins at the nerve–muscle synapse and propagates in both directions toward the ends of the muscle fiber [37,48,78]. Although the speed at which the action potential propagates along the muscle fiber, which is referred to as its conduction velocity, depends on the diameter of muscle fiber [9,81], it is modulated by such factors as changes in muscle length [104], skin temperature [34], and extracellular concentrations of metabolites [46]. Nonetheless, there is a statistically significant, albeit moderate, association between the contractile properties and conduction velocity of motor units in tibialis anterior [2].

Although the presence of a muscle fiber action potential provides an index of muscle activation, the actual interaction of the contractile proteins to produce the force depends on the controlled release and reuptake of Ca^{2+} from the sarcoplasmic reticulum to the sarcoplasm and the level of phosphorylation of myosin light chains. When Ca^{2+} kinetics are compromised, such as during some types of fatiguing contractions [92], the association between the number of muscle fiber action potentials and muscle force is disrupted. As an example of the magnitude of the dissociation between muscle activation (number of muscle fiber action potentials) and force due to dysfunction of excitation-contraction coupling and other impairments, Dideriksen et al. [25] compared the simulated relations between EMG amplitude and

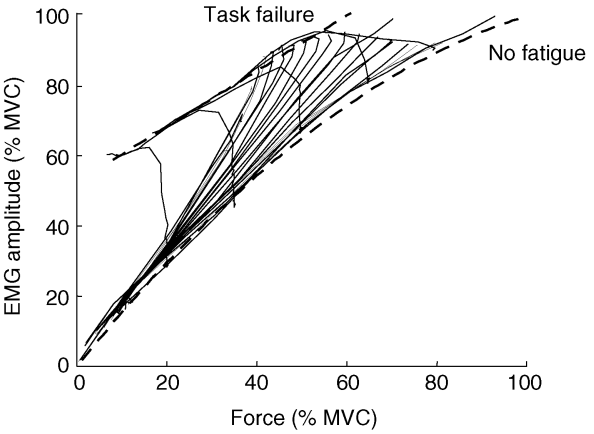


FIGURE 1.4 The relation between the amplitude of the surface EMG signal and muscle force during three simulated protocols that involved fatiguing contractions. The thin lines denote the simulated relations for the different fatigue protocols, the details of which are described in Dideriksen et al. [25]. The observed associations between EMG amplitude and force were bounded by the absence of fatigue (*lower dashed line*) and the relation when the simulated contractions were continued beyond task failure for sustained submaximal contractions (*upper dashed line*). The results indicate that EMG amplitude was not uniquely related to muscle force during the simulated fatiguing contractions. Modified from Dideriksen et al. [25] with permission.

muscle force after three fatigue protocols. The associations between EMG amplitude and muscle force across the three protocols were bounded by the absence of fatigue and the adjustments observed when the simulated contractions were sustained longer than task failure (Fig. 1.4). Fatiguing contractions, therefore, resulted in the same muscle force being associated with EMG amplitudes that differed by up to 25% of the MVC value.

Conversely, the peak force achieved during a muscle twitch in response to a single electrical stimulus is transiently increased immediately after a maximal voluntary contraction [99]. The increase in twitch force, referred to as post-activation potentiation, is obtained without any change in the size of the compound muscle action potential [3]. Therefore, as with the adjustments during fatiguing contractions, the force exerted by a muscle is not directly related to the amplitude of the activation signal (number of muscle fiber action potentials) when the muscle is in a state of post-activation potentiation [4].

1.4.2 Muscle Unit Force

The maximal force capacity of a muscle unit depends on the average cross-sectional area of the muscle fibers (μm^2), the specific force of the fibers ($mN/\mu m^2$), and the innervation number. Of these three factors, the most significant is the number of fibers in the muscle unit [10,63,103]. Therefore, the weakest muscle units have the lowest

innervation numbers, whereas the strongest muscle units comprise the greatest number of muscle fibers. Consequently, adaptations in motor unit force can be associated with changes in innervation number. For example, the greater tetanic force of the weakest motor units in the medial gastrocnemius muscle of old rats was associated with an increase in innervation number and a decrease in the number of strong motor units compared with middle-aged rats [60,62]. Nonetheless, both the cross-sectional area and specific force of muscle fibers [13] can change in response to interventions that modulate physical activity level and thereby contribute to adaptations in the peak tetanic force of motor units [60,62,91].

Many activities of daily living, however, are limited by the capacity of motor units to produce power rather than tetanic force. Because power is the product of force and velocity, muscle unit function is also modulated by differences in maximal shortening velocity, which varies with the dominant myosin heavy chain (MHC) isoform in the muscle fibers that comprise the muscle unit. Adult human muscle fibers can express three types of MHC isoforms (types 1, 2A, and 2X) that can also be combined in two types of hybrid fibers (types 1–2A and 2AX) [13]. Type 1 fibers have the slowest unloaded shortening velocity and type 2X fibers the fastest, mainly due to differences in the time that ADP is bound to myosin during the power stroke of the crossbridge cycle [14]. Due to differences in tetanic force and unloaded shortening velocity, the power production capacity of muscle fibers is least in type 1 fibers and greatest in type 2X fibers, although there is considerable overlap between the different type 2 fibers (Fig. 1.5).

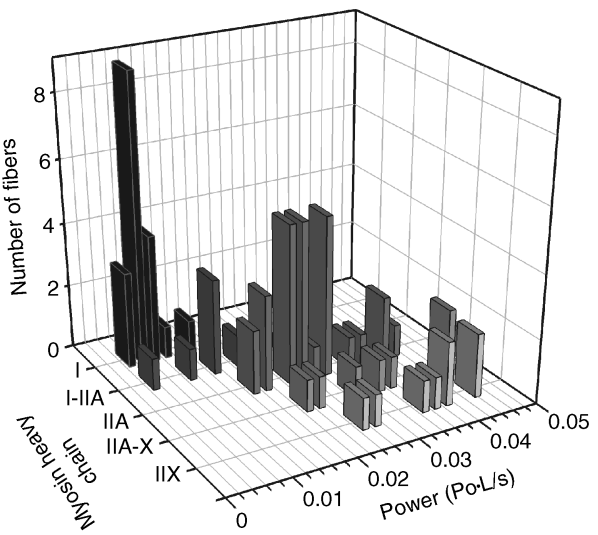


FIGURE 1.5 Maximal power production for 67 skinned fibers from the vastus lateralis muscle of humans. The fibers were classified on the basis of the three myosin heavy chain isoform (I, IIA, and IIX) or combinations of isoforms (I–IIA, IIA–IIX). Despite differences in the average values for the different types of fibers, there was considerable overlap in the distributions across fiber types. Data from Bottinelli et al. [11].