

**ANATOMICAL GUIDE FOR THE  
ELECTROMYOGRAPHER**



# ANATOMICAL GUIDE FOR THE ELECTROMYOGRAPHER

## The Limbs and Trunk

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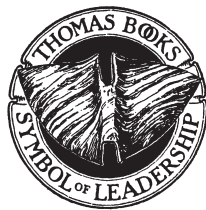
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**CHARLES C THOMAS • PUBLISHER, LTD.**  
*Springfield • Illinois • U.S.A.*

*Published and Distributed Throughout the World by*

CHARLES C THOMAS • PUBLISHER, LTD.  
2600 South First Street  
Springfield, Illinois 62704

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ISBN 978-0-398-08648-0 (Hard)  
ISBN 978-0-398-08649-7 (Paper)  
ISBN 978-0-398-08650-3 (Ebook)

Library of Congress Catalog Card Number: 2011001172

First Edition, 1975  
Second Edition, 1980  
Third Edition, 1994  
Fourth Edition, 2005  
Fifth Edition, 2011

*With THOMAS BOOKS careful attention is given to all details of manufacturing  
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*Printed in the United States of America  
SM-R-3*

**Library of Congress Cataloging-in-Publication Data**

Perotto, Aldo.

Anatomical guide for the electromyographer : the limbs and trunk / by  
Edward F. Delagi . . . [et al.] ; illustrated by Phyllis B. Hammond, Aldo O.  
Perotto, and Hugh Thomas. -- 5th ed. / by Aldo O. Perotto.  
p. ; cm.

ISBN 978-0-398-08648-0 (hard) -- ISBN 978-0-398-08649-7 (pbk.) -- ISBN  
978-0-398-08650-3 (ebook)

1. Electromyography. 2. Extremities (Anatomy). 3. Abdomen--Anatomy. I.  
Delagi, Edward F. II. Title.

[DNLM: 1. Electromyography--methods. 2. Extremities--innervation. 3.  
Muscles--innervation. WE500]

RC77.5.A5 2011

2011001172

*To the memory of my Mentor and friend  
Dr. Edward Delagi*

*And to*

*My granddaughter Laura Adriana  
and to my grandson Martin Alejandro*



## PREFACE

This new edition of the *Anatomical Guide for the Electromyographer* incorporates updated information concerning the basic principles of electromyography. This edition was written at the suggestion of numerous readers who had read the previous edition.

Once again, a vast amount of kinesiological information is included that gives this book a functional angle for the reader. The extensive information also provides the electromyographer with a reminder of the functional anatomy that allows him to understand and analyze the electrical findings.

Great emphasis is placed on describing the insertion of the needle electrode in the intended muscle and to avoid common pitfalls during this phase of the procedure.

The technique described for the study of the diaphragm is not based on my own experience, in contrast to the other muscles in this book. The technique for this muscle was described by Doctor P. Saadeh in 1993. See details of the technique in the footnote of the diaphragm muscle.

In the Appendix, a drawing of the “Nerves Entrapment in the Upper and Lower Extremities Appendix” has been added to facilitate the use and comprehension of anatomic and electromyographic knowledge. It is hoped this new edition will help in the development of future generations of electromyographers.

A.O.P.

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## INTRODUCTION

The primary goal of this book is to be able to reach the intended muscle by using simple techniques. Obtaining distances from easily recognizable anatomical landmarks are sufficient to acquire the target muscle and this is essential for the interpretation of the electrical findings.

The fact that the muscles, especially in the upper and lower extremities are in close proximity to each other, and there is more than one nerve supplying the muscles in the limbs, makes this goal extremely important. The proper technique for each muscle was obtained after many anatomical dissections. These dissections were performed at the Anatomy Laboratory of the Albert Einstein College of Medicine (A.E.C.O.M.) and were shared with the residents of the Department of Rehabilitation Medicine and the Electrodiagnostic Laboratory at Jacobi Hospital and at the Albert Einstein Hospital. We arrived at the conclusion that these techniques were very useful.

At the suggestion of many readers of previous editions of this book, we have decided to incorporate information that describes the essentials of electromyographic testing. Only needle electromyography will be described. Nerve conduction techniques will not be described because they are considered to be outside the scope of the book.



## ACKNOWLEDGMENTS

Once again, my gratitude to Doctor Edward Delagi for all those years of teaching and friendship in the sixth anniversary of his death. The vacuum left by his passing will never be filled. I dedicate this edition to his memory.

Special thanks to my wife for her support and encouragement. To my son Oscar for the editorial work.

My thanks to Doctor B. Nori, Chief of the Department of Rehabilitation Medicine at Elmhurst Hospital, Bronx, New York for her help in preparing this revision.



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## BASIC PRINCIPLES IN ELECTROMYOGRAPHY

This test enables the physician to evaluate the functional activity of the motor unit. It can be a major contributor in the diagnosis of conditions affecting the nerves and the skeletal muscle fibers. The purpose of the test is not to make a diagnosis of a disease, but to assess the functional status of the nerves and the skeletal muscle fibers as mentioned above. The proper localization of the intended muscle with the needle electrode is of paramount importance. The small size of the muscles (as in the hand and facial muscles) and the proximity among them (as in the forearm) makes the use of a clean technique a very important part of the test.

The motor unit (M.U.) is a functional unit which is composed of the following structures:

- (A) The anterior horn cell
- (B) Its axon cylinder
- (C) Terminal branches
- (D) The neuromuscular junction
- (E) The muscle fiber

This test essentially consists of displaying the electrical activity of a living muscle using an intramuscular wire electrode which is placed transcutaneously on the intended muscle. This signal can be converted into a sound and played to a loudspeaker; it can be stored on magnetic tape or printed out using electronic printer or light-sensitive paper.

The muscle under study is evaluated during four different stages:

1. During electrode insertion
2. When the muscle is at rest

3. When the muscle is minimally activated
4. When the muscle is maximally activated

### **A: DURING ELECTRODE INSERTION**

At this moment, a burst of electrical activity occurs which lasts a very short time (approximately 50 msec. or less) (Kimura, 2001). This burst of electrical activity originates in the muscle fiber which is temporarily injured or stimulated by the needle electrode. When the activity ceases, the muscle goes into electrical silence. If the electrical burst becomes prolonged, it could indicate an early sign of neuromuscular dysfunction. If the burst is absent, it indicates that the muscle is no longer viable (severe compartment syndrome, when the entire muscle has been replaced by connective tissue). At this time, an assessment of the amount of fibrotic changes can be made (i.e., resistance to needle insertion).

### **B: AT REST**

A normal muscle is electrically silent at rest. For those muscles in which the innervations become defective, special electrical events develop. The presence of this electrical activity when the muscle is at rest represents one of the most important parts of needle electromyography (Preston & Shapiro, 2005). The presence of abnormal electrical activity can yield such information as: (a) to suggest the neuroanatomic localization of a lesion; (b) the type of spontaneous electrical activity can provide specific diagnostic information (e.g., myotonic discharges are seen only in myopathies); (c) the amount of abnormal activity may determine the severity of the lesion; and (d) this abnormal activity may suggest the time of the lesion, since it takes between 2–3 weeks for them to appear (Preston & Shapiro, 2005).

These spontaneous activities are:

- (a) *Fibrillations*: They represent the spontaneous firing of individual muscle fibers secondary to increased excitability of the muscle membrane following the separation of that muscle fiber from the motor unit (M.U.) (Pease, Lew, & Johnson, 2007). They are usually very small in amplitude (50 to 300 microvolt) and short in duration (~2 msec). It takes approximately 2–3 weeks to appear after the separation has occurred.

Although they may be found in some types of myopathy, they are by far more typical of neuropathies. Fibrillations can be seen in all types of neuropathies affecting the anterior horn cell (Poliomyelitis, ALS) or the axon cylinder (radiculopathies, nerve injuries, systemic diseases). For conditions where the myelin is the primary target (e.g., early stages of diabetes neuropathies; Guillian-Barre syndrome), fibrillations may not be present at the early stages of the conditions. In compression neuropathies, during initial stages where only myelin is affected, fibrillations may not be present. But if the compression continues and the axon cylinder is damaged, fibrillation potentials will appear.

In localized neural insult (entrapment neuropathies), there are three stages of nerve injury:

- (1) Minimal insult, which produces a rapidly reversible nerve block due mainly to edema. No abnormal electrical activity is seen at this stage.
  - (2) Moderate neural injury in which there is failure of an action potential to propagate due to a local demyelization, but the axon cylinder is intact.
  - (3) Severe neural insult: The axon cylinders are damaged, followed by Wallerian degeneration; fibrillation potentials will be present (Dumitru, Amato, & Zwarts, 2002).
- (b) *Positive sharp waves*: These are electrical events whose origin is not well established. It is believed that they originate from a single motor fiber as fibrillation potentials but they last longer than fibrillation (about 10 msec) and the amplitude varies between 50–200 microvolts. The significance of these waves is similar to that of fibrillations; namely, increased membrane excitability secondary to interruption of the connection of the muscle and neural portion of the motor unit. They can also be seen at the last stage of muscular dystrophy (Dumitru et al., 2002; Preston & Shapiro, 2005).
- (c) *Fasciculations*: They represent the firing of the entire motor unit. Two types of fasciculation are recognized: benign (myokynias) and malignant. The former occurs in normal individuals and following stressful, unusual activity. The latter occurs in patients with problems affecting the proximal end of the motor unit, such as anterior horn cell or radiculopathies. According to Buchthal and