ELEC ENG 3BB3:

Cellular Bioelectricity

Notes for Lecture 23

Tuesday, March 4, 2014

Muscle fiber types

1. Fast twitch
   - large number of fibers in each motor unit, for greater contraction strength
   - Motor neurons have larger axon diameters
   - extensive sarcoplasmic reticulum for rapid release of Ca^{2+}
   - large amounts of glycolytic enzymes
   - less extensive blood supply
   - fewer mitochondria

2. Slow twitch
   - smaller number of fibers in each motor unit
   - innervated by smaller nerve fibers (axons)
   - more extensive blood vessel system
   - more mitochondria
   - large amounts of myoglobin, speeding oxygen transport

Twitch fusion

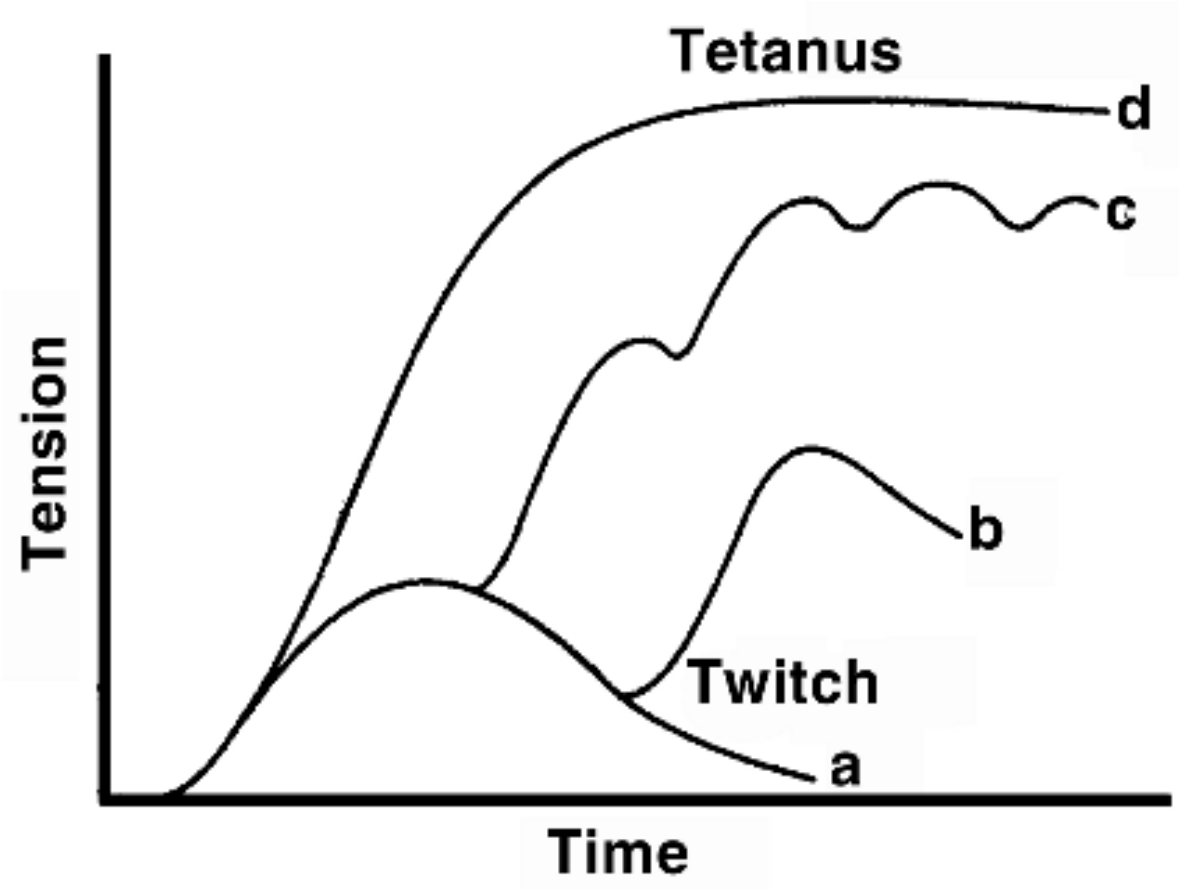


Figure 11.3. Tension versus Time for a Single Stimulus (twitch response) and for a train of stimuli of increasing frequency b, c, d. From Keynes RD, Aidley DJ. 1981. *Nerve and muscle*. Cambridge: Cambridge UP. Reprinted with the permission of Cambridge University Press.

Actin & myosin filament movement:

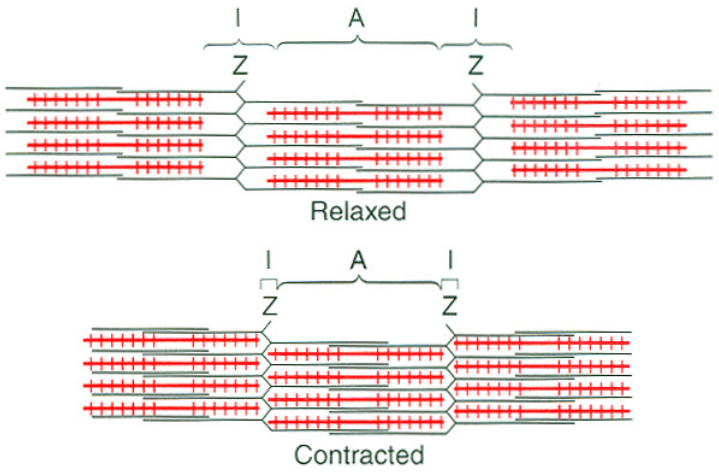


FIGURE 6–4

Relaxed and contracted states of a myofibril showing (*top*) sliding of the actin filaments (*black*) into the spaces between the myosin filaments (*red*), and (*bottom*) pulling of the Z membranes toward each other.

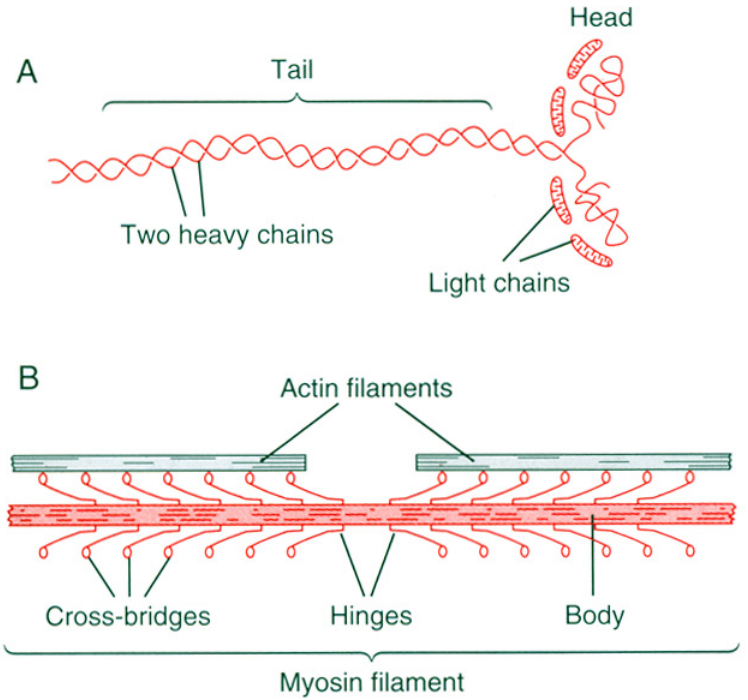


FIGURE 6–5

A, Myosin molecule. *B*, Combination of many myosin molecules to form a myosin filament. Also shown are the cross-bridges and interaction between the heads of the cross-bridges and adjacent actin filaments.

(from Guyton and Hall, 10th Edition)

Actin & myosin filament movement:

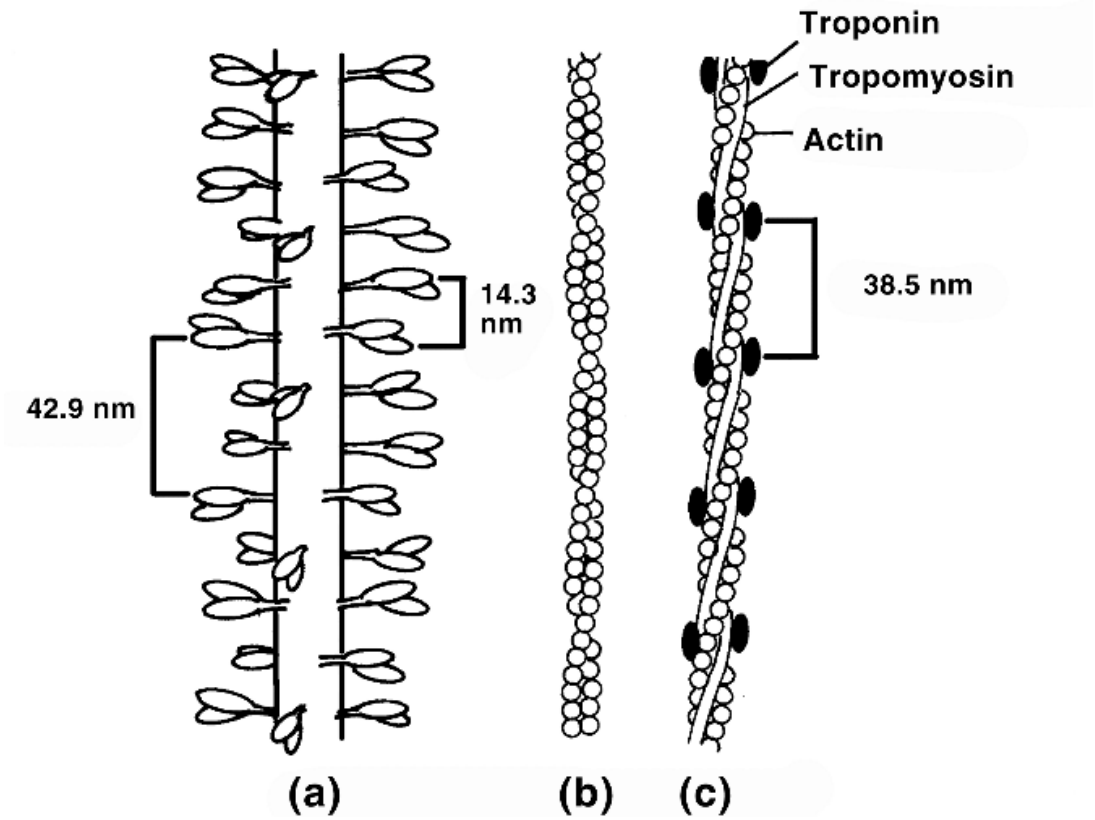


Figure 11.9. Models of the Structure of the Thick and Thin Filaments: (a) myosin; (b) F-actin; (c) thin filament. In (a) the two globular heads of myosin, which split ATP, are shown (a more detailed view is given in Figure 11.11). From Keynes RD, Aidley DJ. 1981. *Nerve and muscle*. Cambridge: Cambridge UP. Reprinted with the permission of Cambridge University Press. Based on Offer G. 1978. The molecular basis of muscular contraction. In *Companion to biochemistry*, Ed AT Bull et al. London: Longman; Huxley HE, Brown W. 1967. The low angle x-ray diagram of vertebrate striated muscle and its behavior during contraction and rigor. *J Mol Biol* **30**:383–434; and Huxley HE. 1972. Molecular basis of contraction in cross-striated muscles. In *Structure and function of muscle*, 2nd ed., pp. 301–387. Ed GH Bourne. New York: Academic Press.

Actin & myosin filament movement:

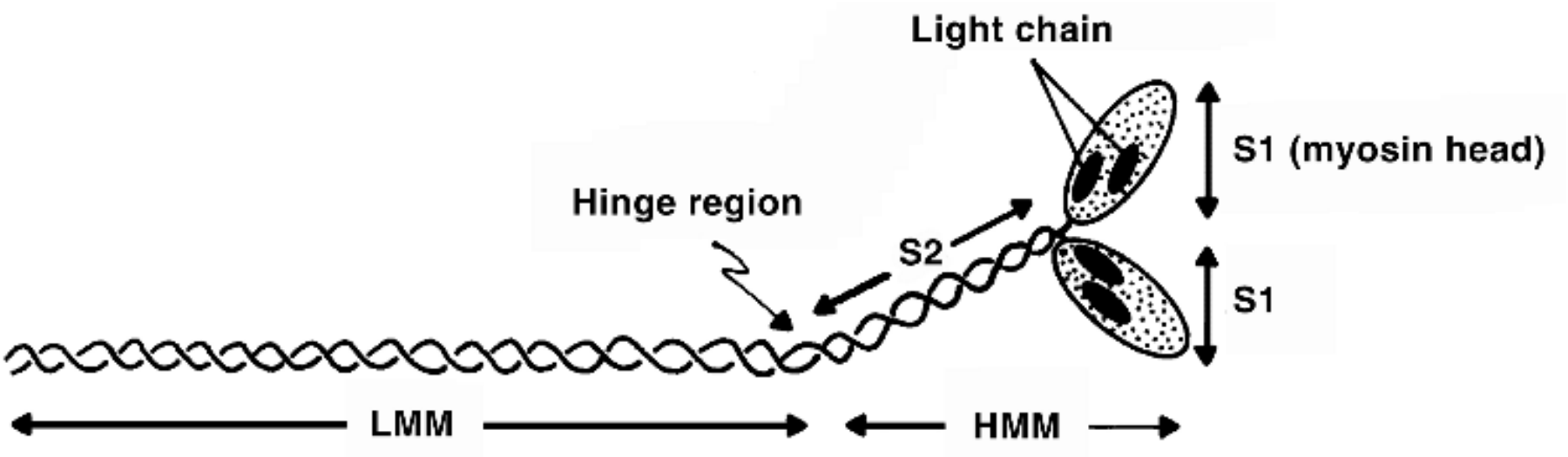


Figure 11.7. Different Components of the Myosin Molecule. Proteolytic enzymes cleave the molecule into heavy meromyosin (HMM) and light meromyosin (LMM). The HMM comprises a short segment of the α-helical rod (S2) and the two globular heads (S1), to which the light chains are attached. The globular heads form the cross-bridges. Reprinted by permission from McComas AJ. 1996. *Skeletal muscle*, Champaign, IL: Human Kinetics. Based on Vibert P, Cohen C. 1988. Domains, motions, and regulation in the myosin head. *J Muscle Res Cell Motility* **9**:296–305, and Rayment I, et al. 1993. Structure of the actin–myosin complex and its implications for muscle contraction. *Science* **261**:58–65.

Actin & myosin filament movement:

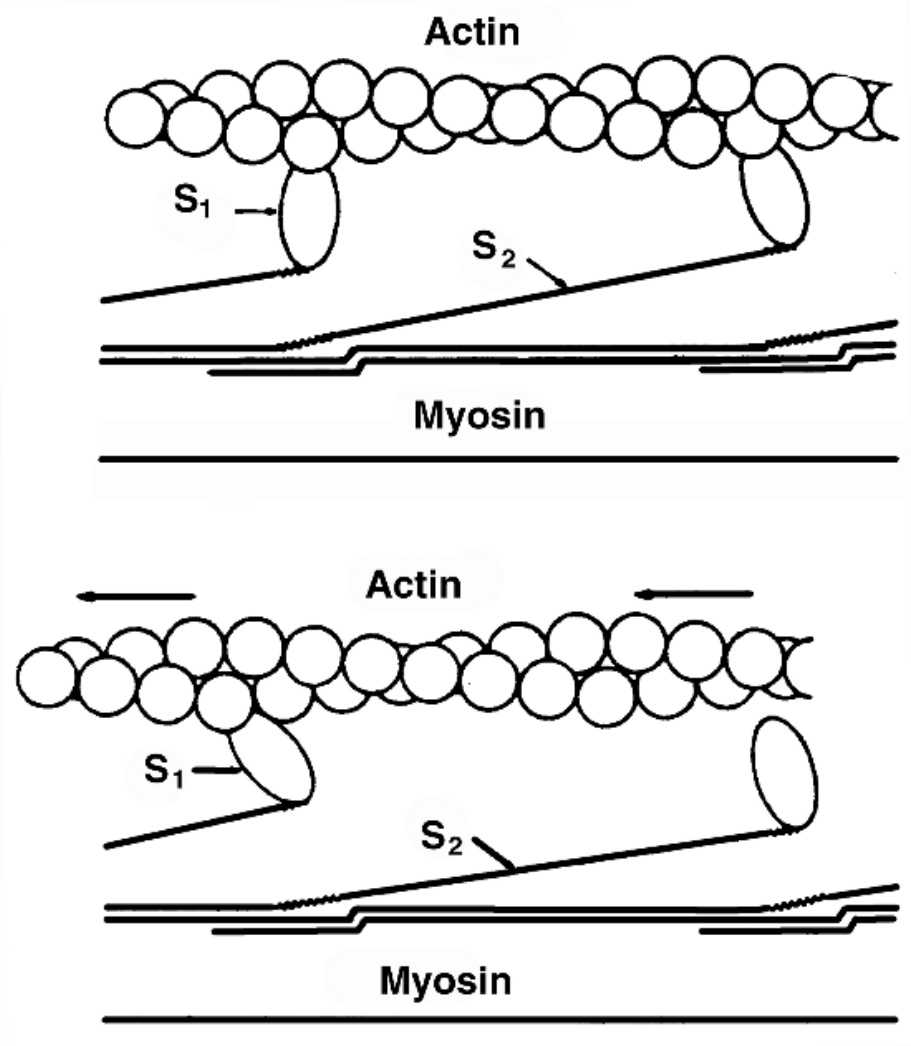


Figure 11.14. Interaction of Actin and Myosin on a Molecular Level. From Huxley HE. 1975. The structural basis for contraction and regulation in skeletal muscle. In *Molecular basis of motility*. Ed LMG Heilmeyer et al. Berlin: Springer.

Actin & myosin filament movement:

1. Myosin is released from a cross-bridge with actin. This results from the action of ATP with which the myosin combines. That is,

$$\mathrm{AM} + \mathrm{ATP} \rightarrow \mathrm{A} + \mathrm{M} \cdot \mathrm{ATP},$$

   where A ≡ actin and M ≡ myosin).
2. ATP is split into ADP + P, while the myosin (S_2) repositions for reattachment with the thin filament. The products remain attached to the myosin, which now has a high affinity for actin.
3. Myosin cross-bridges attach to a new actin monomer.

Actin & myosin filament movement:

4. This results in products being released and the energy so derived utilized as the power stroke (rotation of S_2 and linear movement of actin). At this point, return to step 1.

While actin will react with pure myosin so as to split ATP in the absence of calcium ions, when tropomyosin and troponin are also present, calcium ions are required. In the case of myofibrils, the tropomyosin and troponin are part of the thin filament (see Fig. 11.9c).

Goals of Electromyography

- **Diagnosis (Identify Neuromuscular Disease, e.g. ALS, muscular dystrophy)**
- **Determine extent of disease and monitor progress**
- **Measure dysfunction and propose solutions**
- **Study normal anatomy and physiology**

EMG measurement and interpretation:

Dynamic

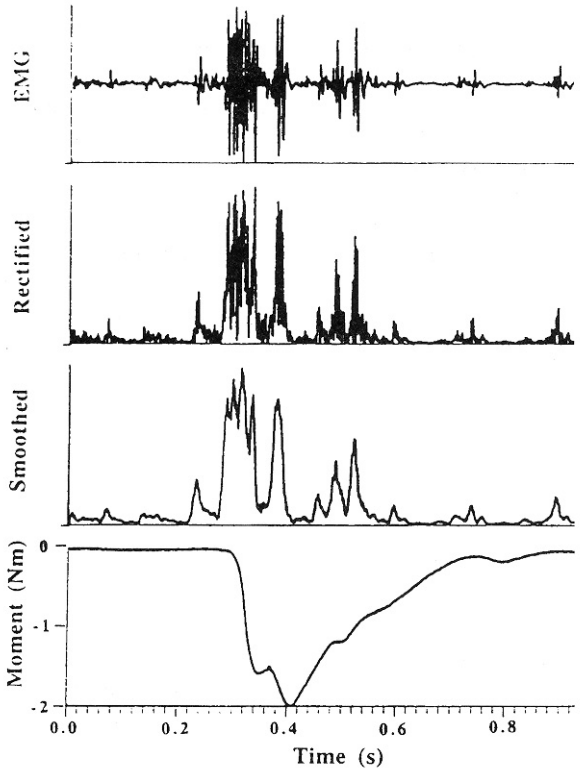


Fig. 10.20 The raw electromyogram is often processed by rectification and smoothing, and is often assumed to vary proportionally with the muscle's 'active state'. In 'phasic contractions', the smoothed rectified EMG does not correlate well with force.

Isometric

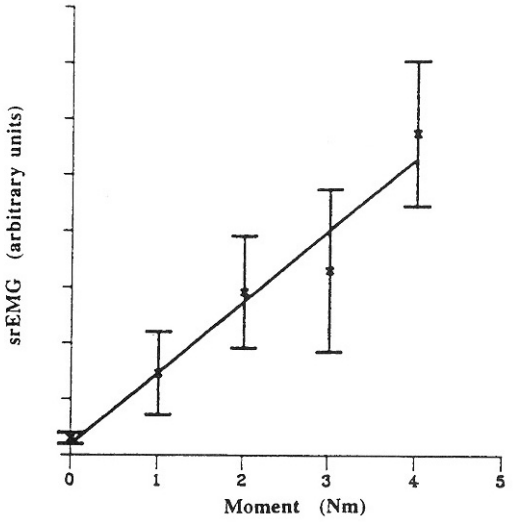
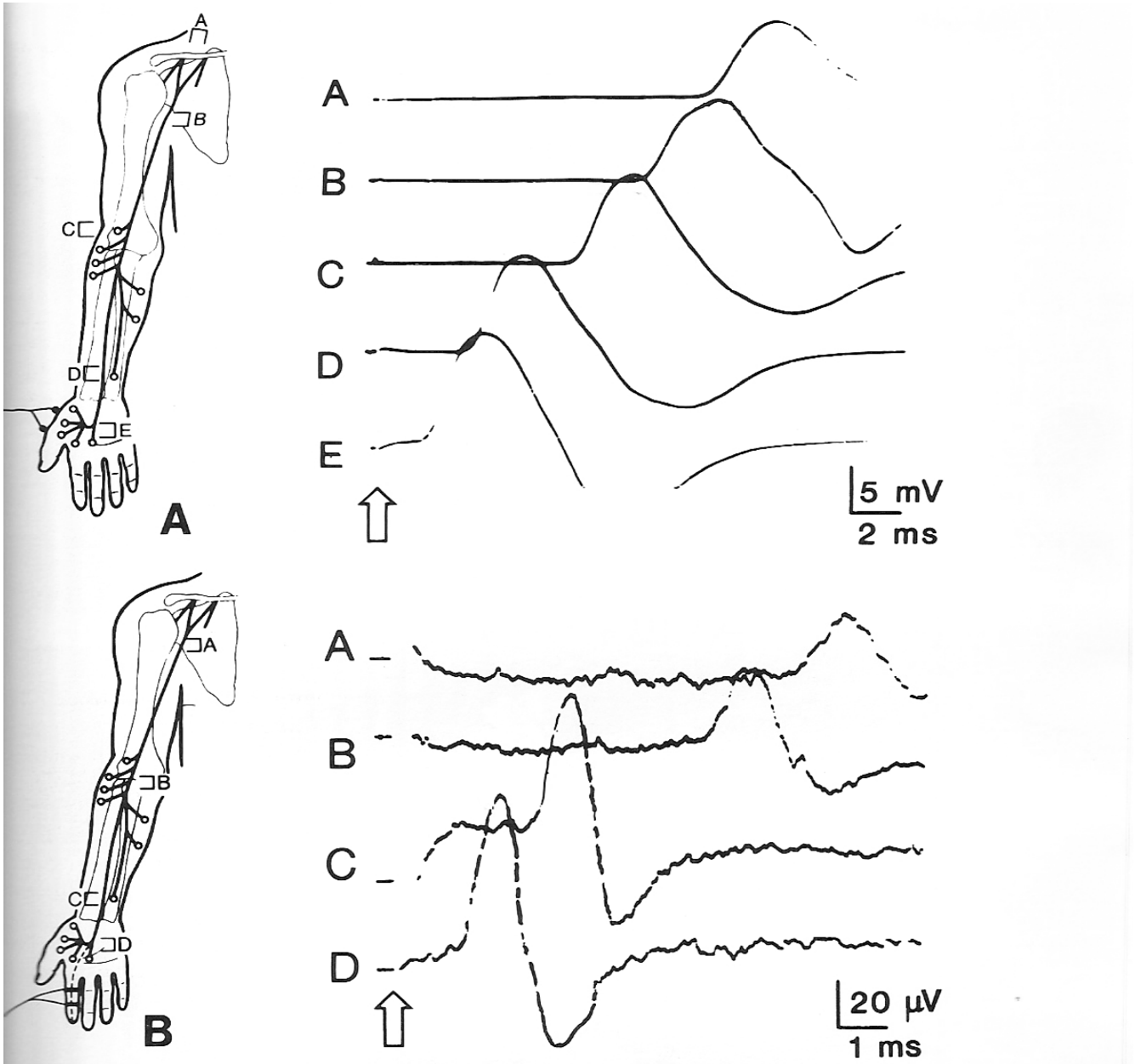


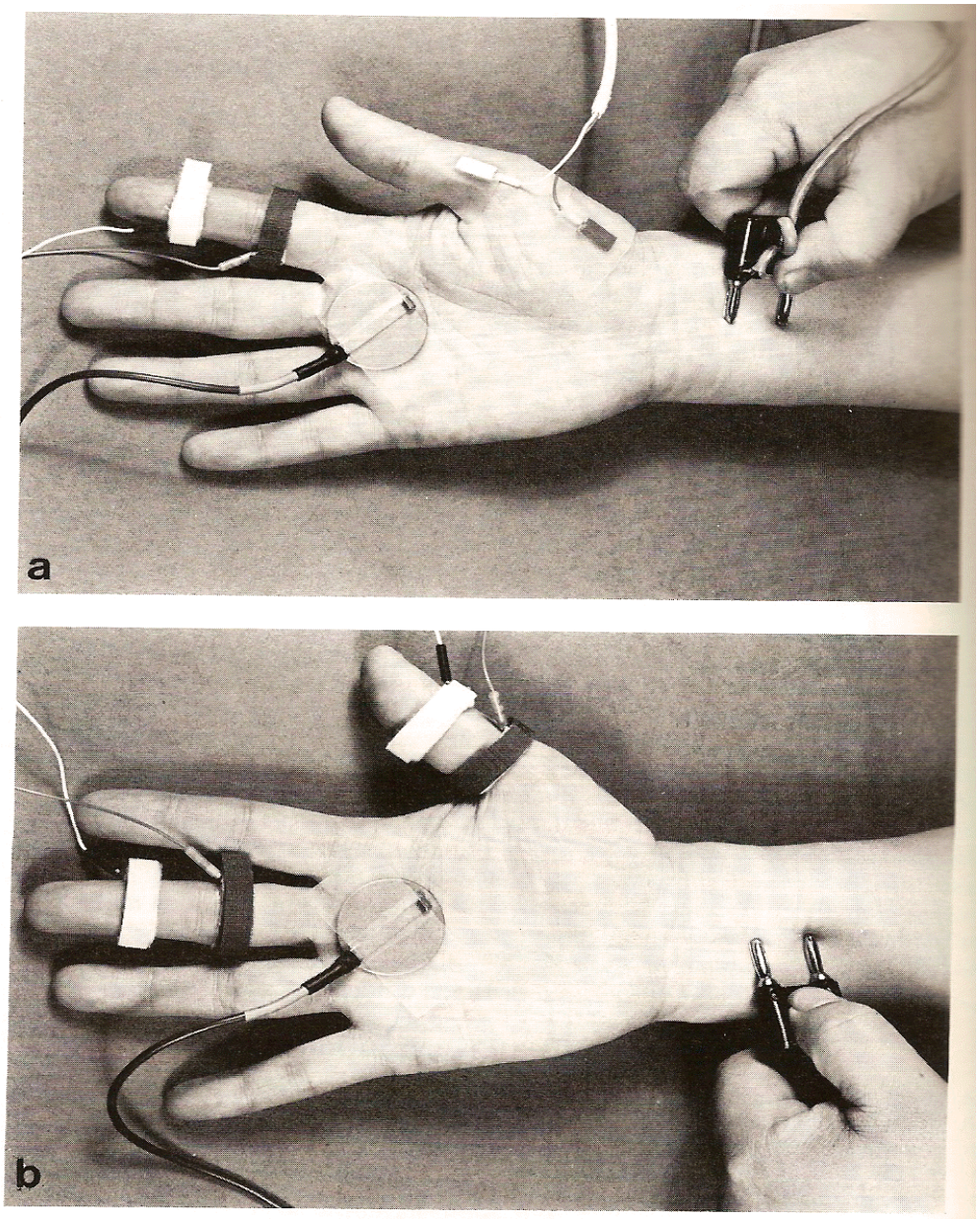
Fig. 10.21 For isometric contractions, integrated smoothed rectified EMG can often be correlated with force.

(Berger et al.)

Surface Diagnostic Techniques (Motor and Sensory Conduction)



Patient Instrumentation
(Sensory and Motor Conduction Velocity)



Stimulation at Wrist

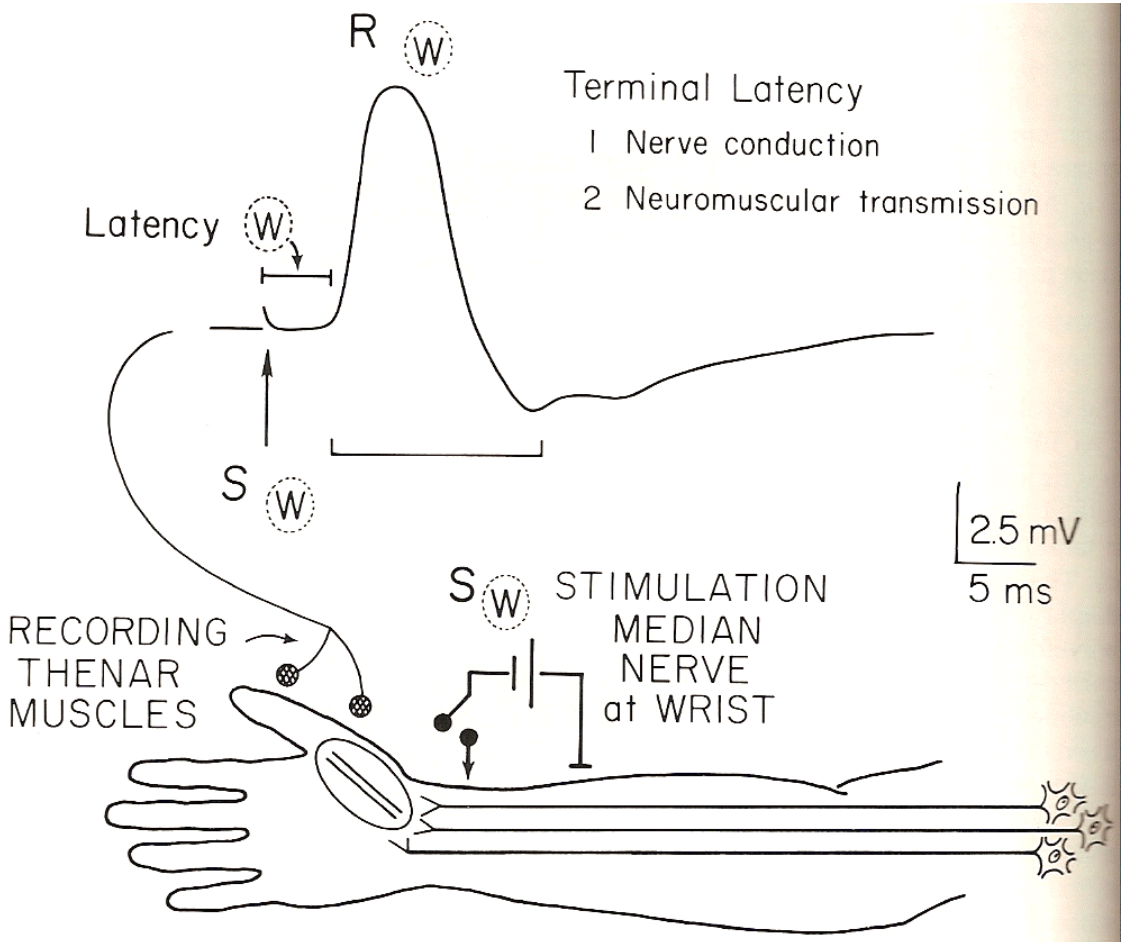


Figure 5-1

Compound muscle action potential recorded from the thenar eminence following stimulation of the median nerve at the wrist. The distal or terminal latency includes (1) nerve conduction from the stimulus point to the axon terminal; and (2) neuromuscular transmission including the time required for generation of the muscle action potential after depolarization of the end-plate.

Stimulation at the Elbow

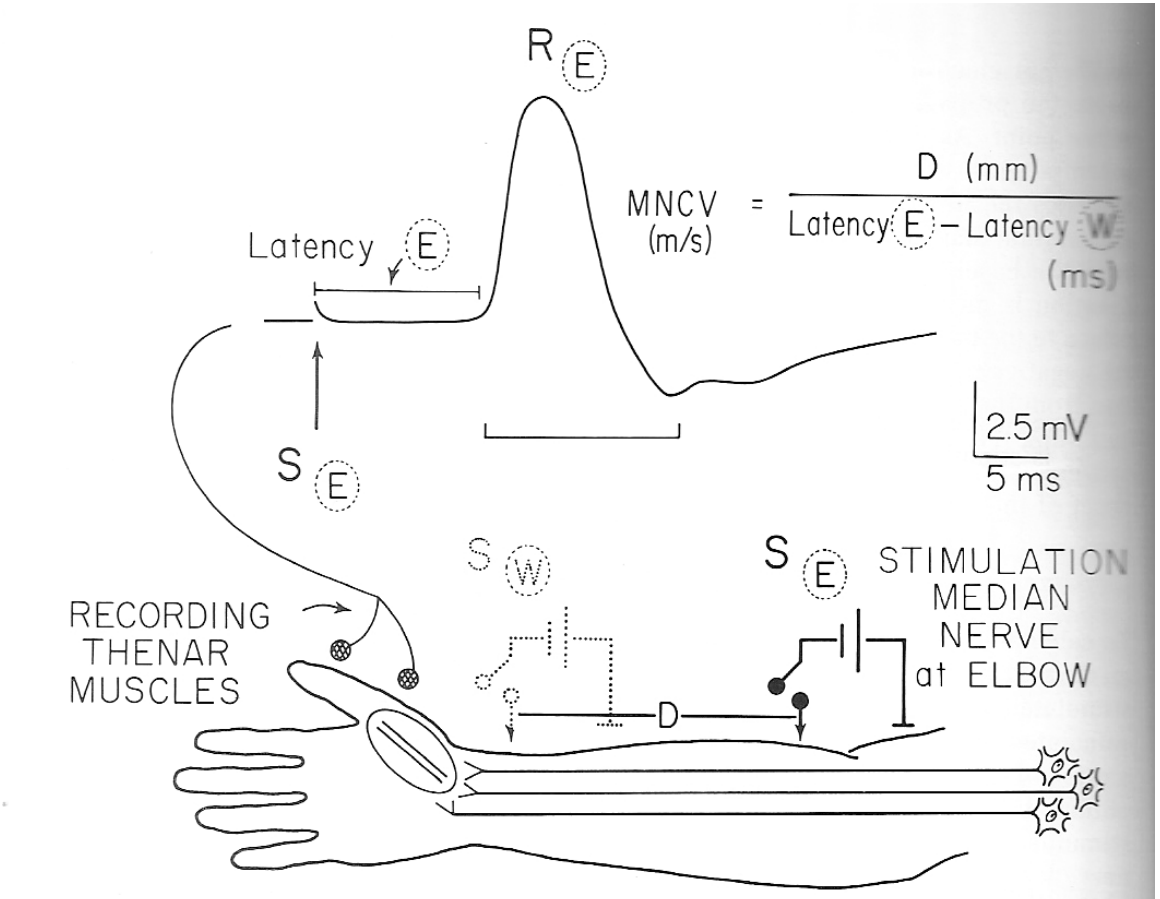
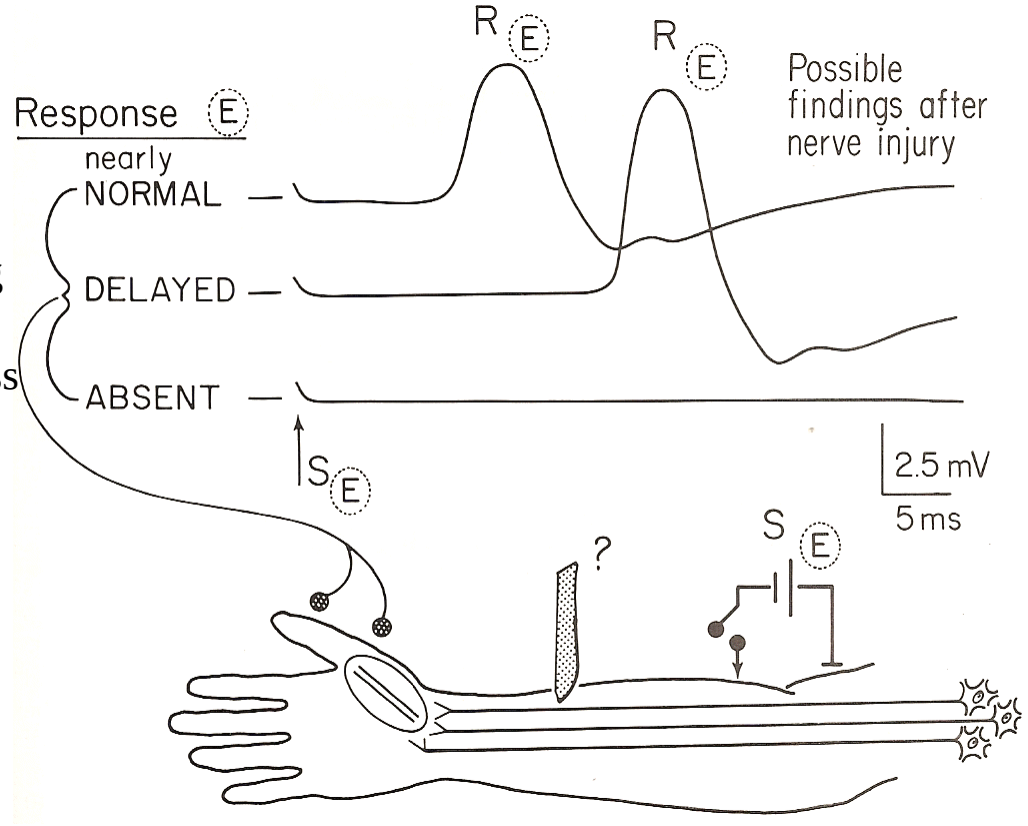


Figure 5-2
Compound muscle action potential recorded from the thenar eminence following stimulation of the median nerve at the elbow. The nerve conduction time from the elbow to the wrist can be determined as the latency difference between the distal and proximal stimulations. The motor nerve conduction velocity (MNCV) is then calculated by dividing the surface distance between the stimulus points by the latency difference.

Pathological Findings

Some Loss of Axons Demyelinating Condition

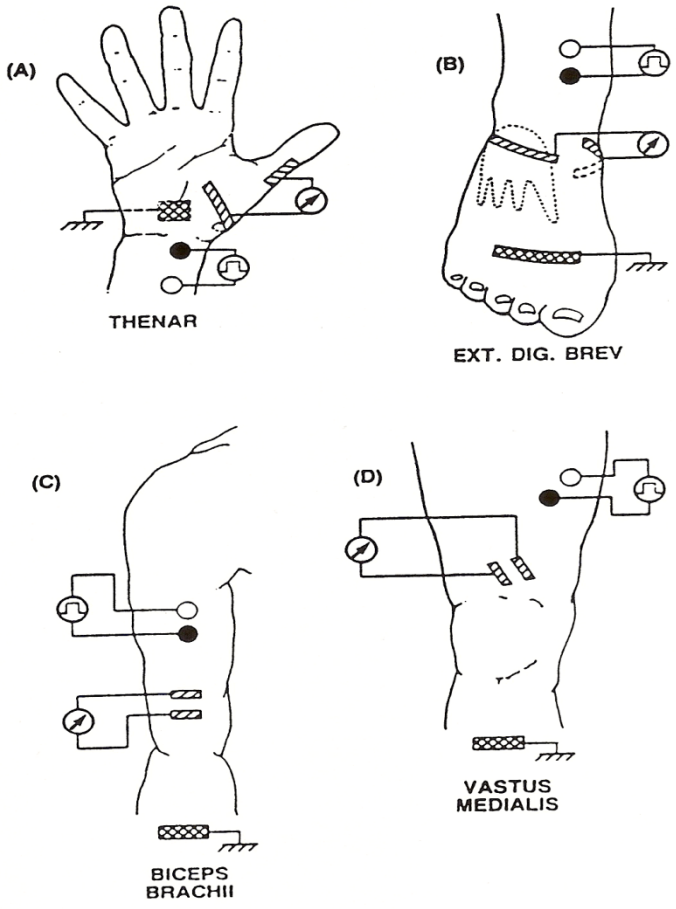
Complete Loss of Axons



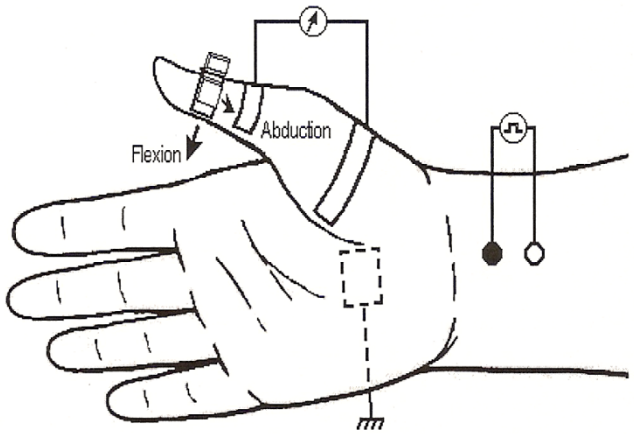
Motor Unit Number Estimation

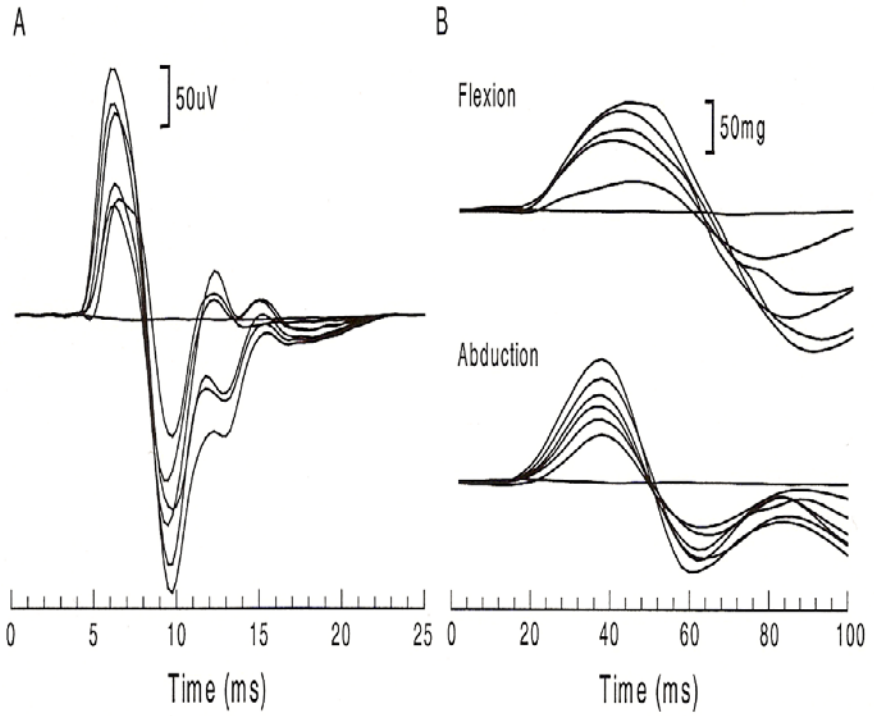
- Estimate the number of alpha motor neurons
- Determine anatomy of normal nerves and muscles
- Determine presence and extent of neuronal disease (diagnostic)
- Monitor disease progression or response to therapy (drug trials, etc)

Patient Instrumentation

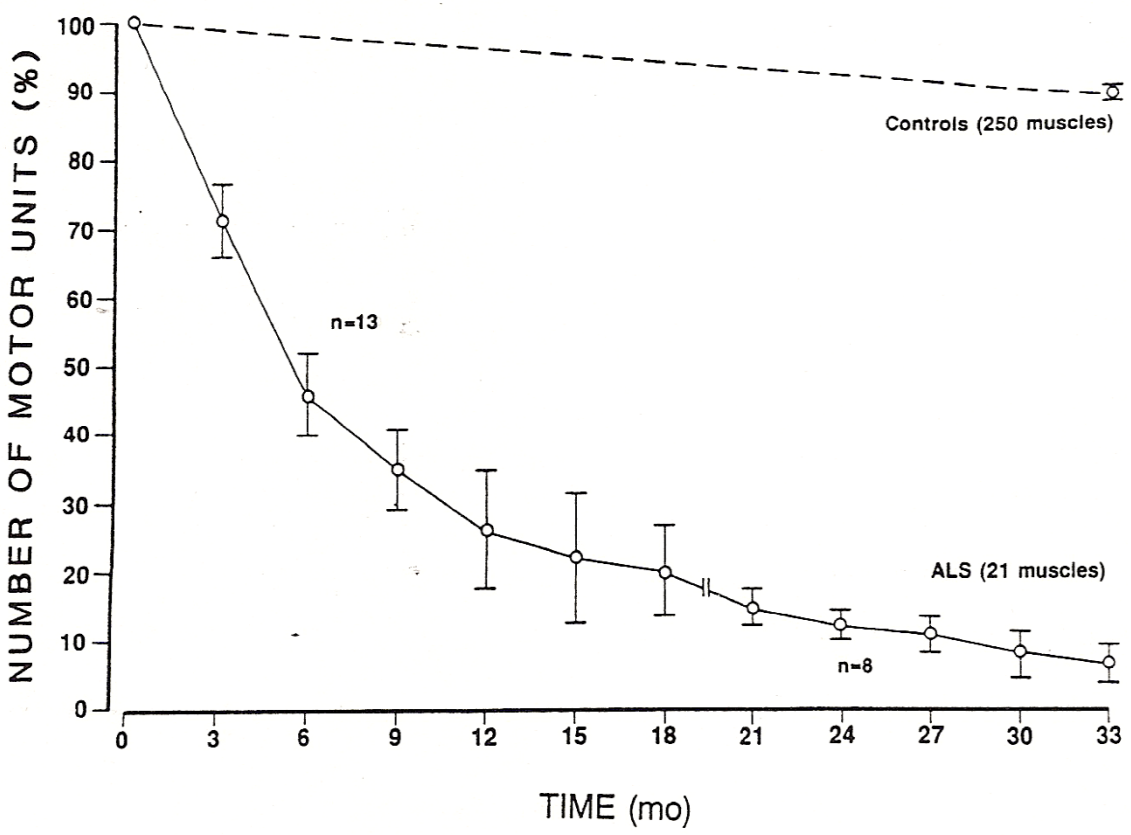


Motor Unit Electrical and Mechanical Responses





Results in ALS



Central Nervous System Identification

- Diagnose and monitor diseases of the central nervous system (Parkinsonism)
- Assess dysfunction following trauma
- Assess effects of intervention (drugs, physiotherapy, surgery)
- Study normal muscle control

Motor Unit Firing Rates

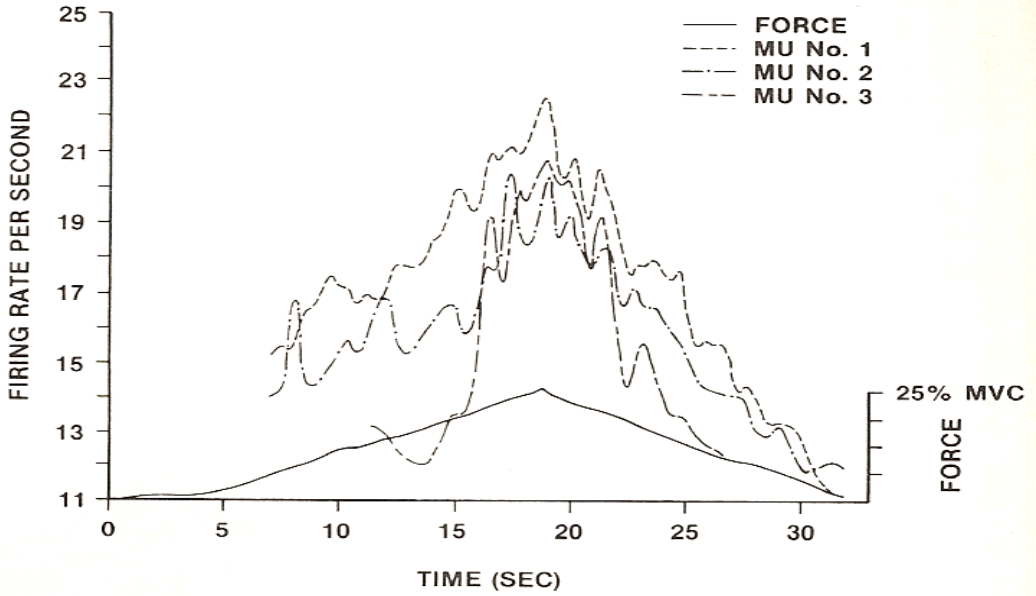


Fig. 7. Rate coding results for a ramped (to 25 percent MVC) contraction of the first dorsal interosseous muscle.

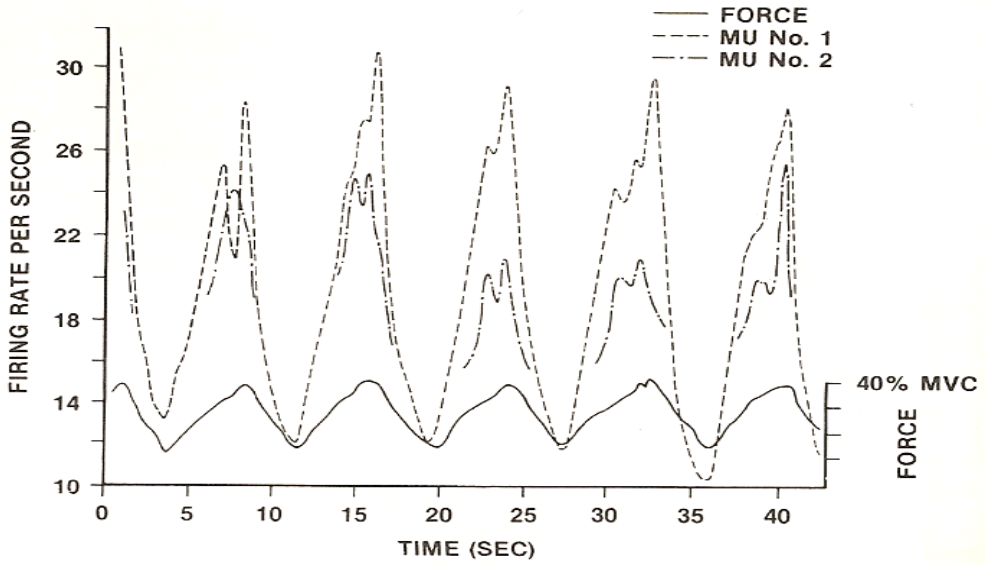


Fig. 8. Rate coding results for a contraction modulated about 25 percent MVC of the first dorsal interosseous muscle.