Chapter 25: Basic Principles of Exercise Physiology

INTRODUCTION

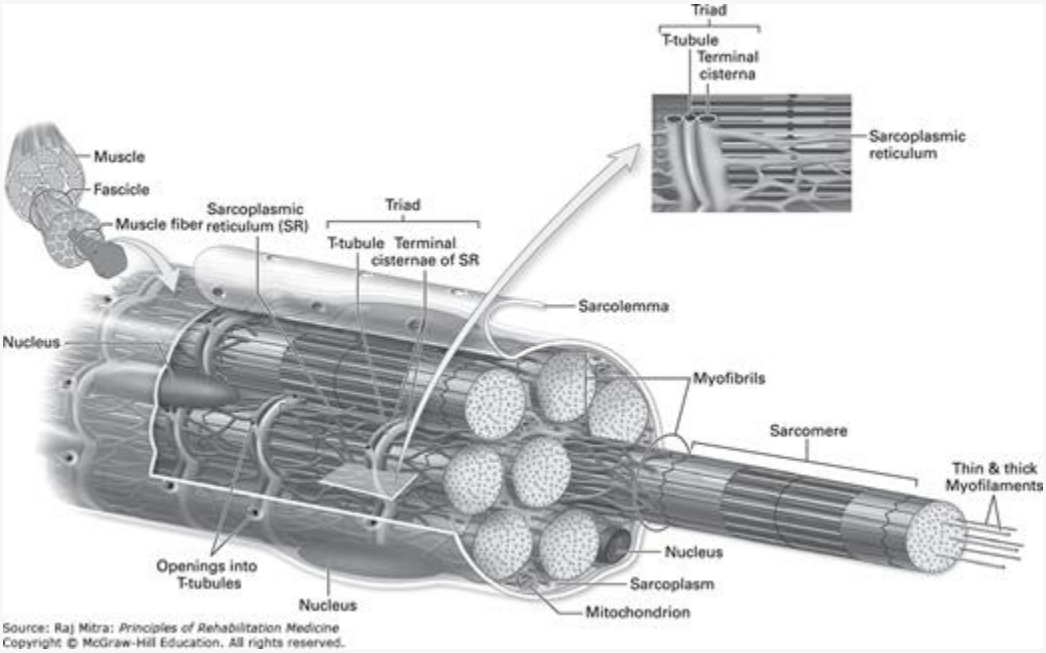
Exercise has a positive and significant impact on the human body by improving physiological function, physical fitness, and health. Physical activity can lead to numerous physiological changes that may lower the prevalence of chronic disease and cause significant adaptions in many physiological systems of the body. There are potential benefits of exercise on the cardiovascular, pulmonary, and neuroendocrine systems. Increased physical activity can directly affect energy expenditure, metabolic change, neurobiological effects, and neuroendocrine changes.

BASIC PHYSIOLOGY OF SKELETAL MUSCLE FIBERS

Skeletal muscle fibers are responsible for the generation of force in order to produce movement. Myofibrils are organized to compose skeletal muscle fibers. These myofibrils are surrounded by portions of the sarcoplasmic reticulum and have deep channels (T tubules) (Fig. 25–1).

FIGURE 25–1

Organization of a skeletal muscle fiber. (Reproduced with permission from Muscle Tissue. In: Mescher AL, eds. *Junqueira's Basic Histology*, 14e New York, NY: McGraw-Hill; 2016.)



Muscle fibers vary in terms of their mechanical, physiological, and biochemical properties, making skeletal muscle a heterogeneous tissue. Skeletal muscle fibers have been classified by using histochemical techniques (ATPase and oxidative enzymes stains), measurements of contraction (twitch) or fiber shortening velocity, and the identification of myosin heavy chain isoform with use of protein electrophoresis. Skeletal muscle may contain up to three types of fibers in varying proportion: fiber types I, IIa, and IIx.[1] It is important to note that skeletal muscle fibers can express more than one type of myosin heavy chain isoform simultaneously.[1] Type I myosin heavy chain isoform muscle fibers are the slow, oxidative, fatigue-resistant fibers. Type IIx are very fast contracting, glycolytic, and fatigable fibers. Type IIa have intermediate properties. These fibers are fast contracting but with an oxidative metabolic profile. Fibers that express more than one type of myosin heavy chain isoform are known as hybrid fibers; various combinations (I/IIa, IIa/IIx, I, IIa, IIx) have been reported.[1]

Force can be generated while skeletal muscle remains static, shortens, or increases in length.[2] During most types of movement or exercise, muscles alternate between static and dynamic muscle actions.[3]

The generation of force by muscle fibers is dependent on a complex series of well-orchestrated events initiated by a nerve impulse, which triggers the release of acetylcholine (ACH) into the synaptic cleft. The ACH initiates muscle contraction by binding to the ACH receptors in the neuromuscular junction. This impulse quickly spreads from the sarcolemma as calcium ions are released into the sarcoplasm. The calcium ions eventually bind to troponin, which causes a change in the morphology of the thin filament. Tropomyosin, which is located on actin (on the thin filament) is shifted, and the shift exposes active sites on the thin filament. Concomitantly, myosin heads located on the thick filaments are also exposed to the ...

Chapter 30: Rehabilitation of Musculoskeletal Conditions of the Upper Extremity

SHOULDER

Fractures

Upper extremity fractures account for more than half of all pediatric fractures[1] and only 7% of geriatric fractures.[2] The annual incidence of upper extremity fractures in the United States is 67.6 fractures per 10,000 persons.[3] The most common shoulder girdle fractures occur in the proximal humerus, with fractures of the clavicle being second most prevalent. The annual incidence of proximal humeral fractures is 83.0 events per 100,000 persons,[4] while clavicle fractures account for 24.4 injuries per 100,000 person years.[5] Scapular fractures account for 13 fractures per 100,000 women and 37 fractures

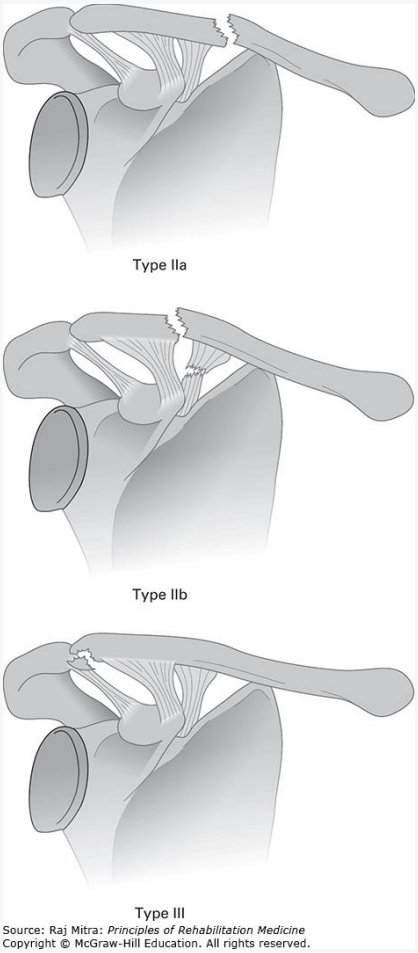
per 100,000 men. Glenoid fractures are rarer and account for 10% of scapular fractures with an overall prevalence of 0.1%.[6]

Clavicle

Eighty percent of clavicle fractures occur within the middle third, while 15% involve the distal third, and 5% involve the proximal third.[7] Distal clavicle fractures can be subclassified into three groups. Type I refers to a clavicle fracture distal to the coracoclavicular (CC) ligament with the ligament remaining intact. Group II refers to a clavicle fracture distal to the CC ligament with (IIB) or without (IIa) disruption of the CC ligament. Type III distal clavicle fractures refer to a fracture with intra-articular extension (Fig. 30–1).

FIGURE 30–1

Classification of distal clavicular fractures. A-P = anteroposterior. (Reproduced with permission from Bjoernsen L, Ebinger A. Shoulder and Humerus Injuries. In: Tintinalli JE, Stapczynski J, Ma O, Yealy DM, Meckler GD, Cline DM, eds. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 8e New York, NY: McGraw-Hill; 2016.)

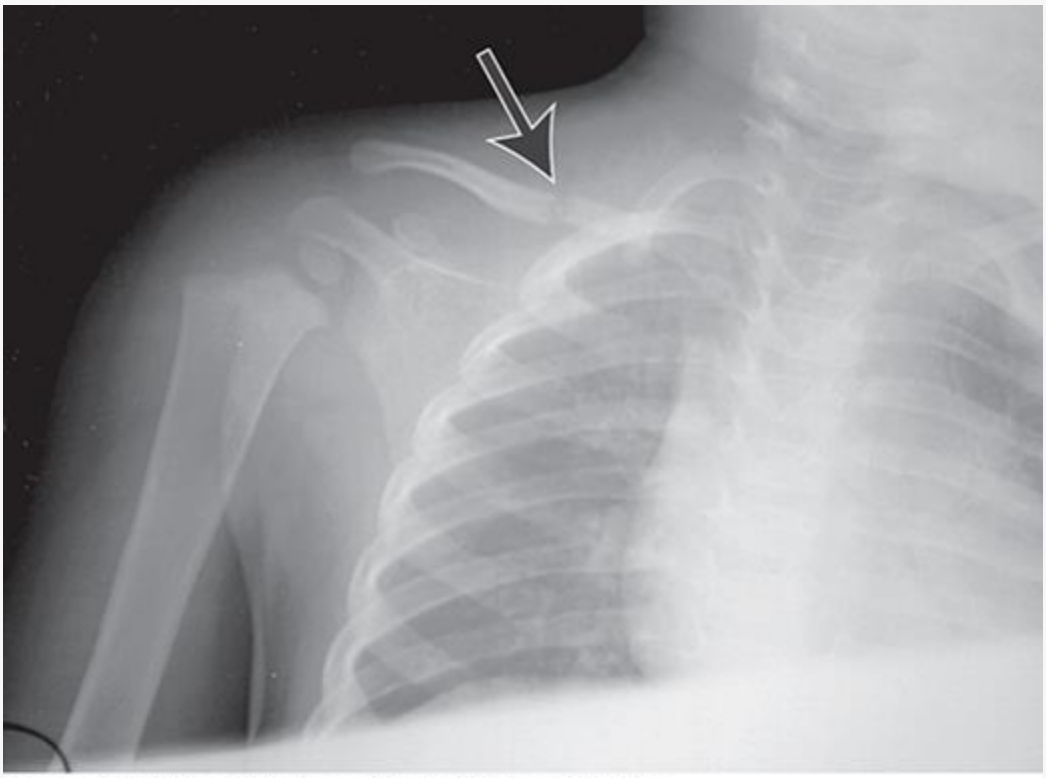


Trauma accounts for the most common etiology of clavicular fractures. Translational force from the lateral shoulder, falling on an outstretched hand, and direct contact with the clavicle may all lead to fracture. Less common causes include childbirth in the neonate, malignancy, and radiation therapy.[8] In rare cases, stress fractures of the clavicle can be seen. Case reports typically center on gymnasts or weight lifters.

A clavicle radiograph series is used for diagnosis. This series includes an anteroposterior (AP) view of the clavicle, acromioclavicular (AC) joint, and sternoclavicular (SC) joint, as well as an AP view with 15 degrees of cephalad angulation[9](Fig. 30–2). Computerized tomography (CT) scans can be performed in instances of equivocal radiographs or in cases of fracture near the AC or SC joint.

FIGURE 30–2

Nondisplaced clavicle fracture in an infant (*arrow*). (Reproduced with permission from Black KL, Duffy C, Hopkins-Mann C, Ogunnaiki-Joseph D, Moro-Sutherland D. Musculoskeletal Disorders in Children. In: Tintinalli JE, Stapczynski J, Ma O, Yealy DM, Meckler GD, Cline DM, eds. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 8e New York, NY: McGraw-Hill; 2016.)



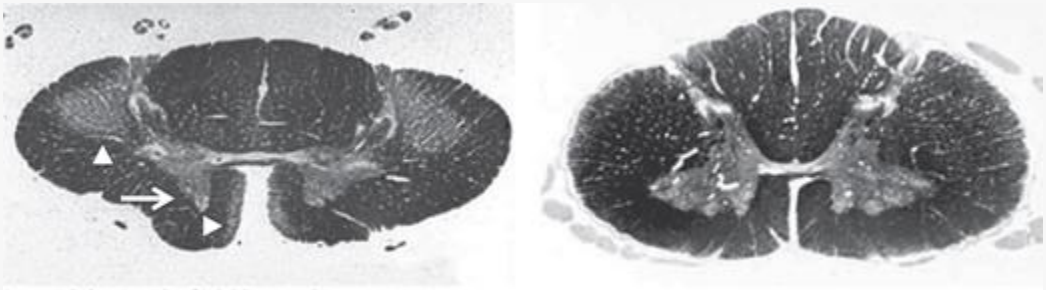
Chapter 71: Neuromuscular Disease: Motor Neuron Disorders

INTRODUCTION

Motor neuron diseases (mnds) are a heterogeneous group of neurologic conditions characterized by corticospinal tract and anterior horn cell degeneration of the brain and spinal cord[1] (Fig. 71–1). While the etiology of MNDs is broad and not clearly elucidated, there is evidence supporting multifactorial processes that can be inherited genetically or acquired sporadically through environmental exposures such as toxins, infections, and autoimmune or other causes.[2–8] The common pathology is the destruction of motor neurons and encompasses disorders with selective loss of lower motor neurons (LMNs), upper motor neurons (UMNs), or a combination of both.[2] This results in muscle weakness and disability with loss of normal limb, speech, swallow, and respiratory functions.

FIGURE 71–1

(Left panel) Cervical spinal cord in amyotrophic lateral sclerosis shows dramatic atrophy of gray matter in anterior horns (arrow) due to loss of motor neurons. The pale-staining areas in the lateral and anterior columns (arrowheads) of the spinal cord reflect the great loss of myelinated axons in the lateral and anterior corticospinal tracts. (Right panel) Normal cervical spinal cord. (Photo contributor: Kinuko Suzuki, MD, Tokyo Metropolitan Institute of Gerontology; retired faculty, Department of Pathology and Laboratory Medicine, University of North Carolina, Chapel Hill, NC.)

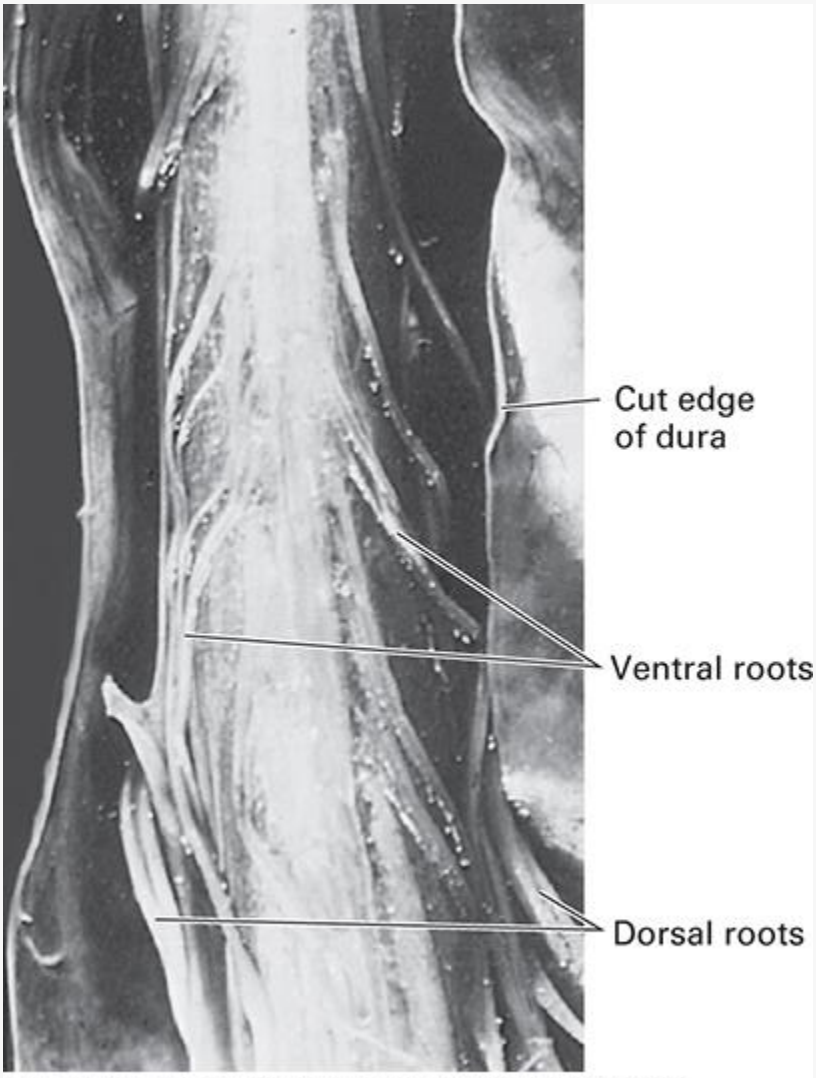


The most common adult form of MND is amyotrophic lateral sclerosis (ALS), which is characterized by both UMN and LMN findings[1] (Fig. 71–2). ALS, first described by Jean-Martin Charcot in the 1870s, is considered to be the classic model of MND.[1]In the United States, for three-quarters of a century, ALS was referred to as “Lou Gehrig's disease,” named after a beloved American baseball player who publicly announced the end of his career due to the disability caused by the disease. This moniker, however, has fallen out of favor since a very successful research fund-raising campaign (known as the “ALS Ice Bucket Challenge”) caused a shift in the public's recognition of the disease. In this chapter, we discuss MND classification, diagnostic and clinical

evaluation, and treatment and management strategies from the perspective of reaching a diagnosis and treating the patient found to have ALS.

FIGURE 71–2

Ventral view of the spinal cord (with the dura opened) of a patient with motor neuron disease (amyotrophic lateral sclerosis). Notice the reduction in size of the ventral roots (resulting from the degeneration of the axons of motor neurons) compared with the normal dorsal roots. (Reproduced with permission from Discussion of Cases. In: Waxman SG, eds. *Clinical Neuroanatomy*, 28e New York, NY: McGraw-Hill; 2017.)



Source: Raj Mitra: *Principles of Rehabilitation Medicine*