CHAPTER 3

The Movement System: Nerve and Muscle Physiology and the Control of Human Movement

"The key to success is discipline. Teens don't want to hear that. They think that they can just snap their fingers, and voila! But with discipline come knowledge, coordination, balance, muscle memory, confidence — things that make it possible to hit the bulls-eye three times in a row."

—*Carlos Santana, musician, 1947*

CHAPTER OUTLINE

LEARNING OUTCOMES

This chapter provides an overview of nerve and muscle interaction and how they function together. After reading this chapter, you should be able to:

- ❑ Describe the properties of irritability, excitability, and transmission that are the unique capabilities of nerve and muscle tissue;
- ❑ Give an overview of the nervous system's physiological, anatomical, and functional divisions and explain their functions;
- ❑ Describe the basic structure of skeletal muscle and how skeletal muscle contracts;
- ❑ Describe the different types of muscle fibers and their contribution to functional movement;
- ❑ Describe the function of proprioceptors—Golgi tendon organ, joint receptors, and muscle spindle—and explain how they contribute to human movement control;
- ❑ Define and describe what motor control means;
- ❑ Describe the functional contributions to motor control from the spinal region, the brainstem, the cerebellum, the basal ganglia, and the motor cortex, and summarize the functional consequences of damage to those areas;
- ❑ Describe the following common movement system impairments—weakness, abnormal muscle tone, coordination problems, and involuntary movements.

CLINICAL SCENARIO

Joseph is a 5-year old child with spastic diplegic cerebral palsy. He has spasticity in both lower extremities and weakness in his trunk, but he is able to ambulate around school and home using a walker. While playing outside, he fell, landing his elbow onto a broken glass bottle and partially severed his left ulnar nerve at the medial epicondyle. Jay, his clinician, needs to explain to Joseph's mother the differences between the weakness that he is displaying in his left hand and the weakness and spasticity that is present in his legs.

Introduction

Since this is a clinical kinesiology textbook, you may wonder what a chapter whose primary information regards physiology and movement theory is doing in such a text. As clinicians, it is important for us to understand how the body works, what makes it work, and how we can influence how it works in the exercise and rehabilitation programs we create for our patients. Since kinesiology is the study of human movement, we must appreciate the elements that produce motion. Human movement occurs as a result of an intimate relationship between anatomy and physiology. This chapter presents information relevant to the connections between these two systems; melding this chapter's information with the subsequent chapters will provide you with a clear understanding and appreciation of human movement.

Providing a basic understanding of neural physiology for the development and appreciation of the kinesiological functions in human performance is the goal of this chapter. We will first identify the basic physiology of uniquely excitable nerve and muscle tissue. Then, the building blocks of the components of the neuromuscular system—neurons, skeletal muscle, and sensory receptors—are described, and we will discuss how the entire system is dynamically organized to produce functional, purposeful movement. This information is followed by an overview of all of the central motor control areas with a focus on their functional contributions to movement. A discussion of movement impairments and their functional consequences follows and concludes with a discussion of clinical considerations, including a brief summary of some of the more common impairments resulting from motor control dysfunction. Impairment of the central and peripheral nervous systems are compared and contrasted. Some of the material in this chapter may serve as a quick review for those who have previously studied the anatomy and physiology of the neuromuscular system. For those who have not had the benefit of such courses, textbooks on human anatomy and physiology and neuroscience may be consulted for a more complete explanation of the functions of the neuromuscular system.

Purposeful movement is a fundamental characteristic of human behavior. Coordinated human movement is the result of an orchestration of muscles acting on the bony skeleton; this orchestration is organized by the nervous system and refined by multiple sensory mechanisms to produce mechanical responses. Human movement requires activation and integration of multiple parts of multiple systems within a split second. Movement is not the result of one single muscle acting across one joint but is an integrated system of the brain and body that responds, executes, interprets, and adjusts to continual feedback. The use of the term "system" is appropriate in discussions of body motion. A "system" is an assemblage or combination of parts that form a functioning unit. Viewing human movement as a system made up of several contributing elements such as the

nervous, muscular, skeletal, and sensory systems allows us to study both structure and function simultaneously. What we usually think of as separate systems are actually components of a larger system that provides desired and purposeful outcomes. In other words, those structures or systems that contribute to more than one function are actually a part of more than one purposeful system.[1]

Therefore, the human **movement system** involves the functional interaction of structures that contribute to the act of moving.[2] Included in these structures are the nervous system's somatosensory and relay components that facilitate the skeletal and muscular systems.[3,4] The body's movement system changes throughout the life cycle in response to growth, maturation, aging, disease, or environmental demands.

Movement occurs through **biomechanical** responses to this neurological input. These biomechanical responses include skeletal muscle contractions that move the body's system of levers and pulleys which are formed by bones, tendons, and ligaments. A person's individuality is expressed by his or her own pattern of muscular contractions. These individually unique manifestations include facial expressions, body postures, fine motor skill performances such as typing or playing a musical instrument, and gross motor activities such as walking and running. The individual with a normal functioning neuromusculoskeletal system has a remarkable ability to develop just the right amount of muscle force needed to perform an endless variety of motor tasks—from placing a contact lens in one's eye to carrying a heavy load of textbooks to class.

Whatever the muscle activity, it is accomplished through intricate communication between the musculoskeletal and nervous systems. An elaborate nervous system provides fine control of muscle contractions over a wide range of lengths, tensions, speeds, and loads. The nervous system is very complex and has myriad responsibilities, including a variety of sensory and motor functions. The sensory nervous system provides accurate and timely information about the status of each body part and their environmental surroundings through its **afferent** (L. *ad,* to, plus *ferre,* to carry) receptors. Incoming sensory information from these afferent receptors travels to various parts of the nervous system where it is accepted, interpreted, and responded to in context with information previously stored in the brain. Once the nervous system has processed the afferent information, its **efferent** (L. *ex,* out, plus *ferre,* to carry) motor nerve impulses send a response to selected muscles or muscle groups to produce desired movements. Thus, the end product—desired movement—is achieved through the collaborative interaction and coordination of primarily the motor and sensory systems.

The body's ability to produce an appropriate response relies on numerous factors. These factors include the ability of muscles to develop graded amounts of active tension; the ability of the cardiovascular, respiratory, and digestive systems to provide the ingredients that fuel the contractile process; and the ability of the nervous system to regulate the rate and amount of contraction needed to accurately move certain body parts while stabilizing and inhibiting other parts. This entire process from afferent stimulation to motor response occurs within milliseconds.

Physiology of Excitable Tissue: Nerve and Muscle

As you learned in physiology, all living cells are surrounded by membranes formed by a continuous phospholipid bilayer. Embedded throughout these membranes are proteins with various characteristics. This section briefly reviews cell physiology to refresh your memory of the topics relevant to neuromuscular function as they relate to and further add to your understanding of kinesiology.

Uniquely, both nervous and muscular tissue membranes are excitable; that is, their membranes are irritable and thereby sensitive to electrochemical change. Furthermore, this excitability can be communicated between the tissues and from one region or system to another. Because of this unique characteristic, nerve cells and muscle cells are not only excitable but are also able to transmit this electrochemical information to produce movement. Before we discuss the specific interworking between the nervous and muscular systems, we need to understand how these tissues work.

Differences in electrical potential exist across the membranes of all living cells. Fluids bathe the inside and outside of each cell. These intracellular and extracellular fluids contain negatively and positively charged particles, called ions. The ions are predominantly negative inside the cell and positive outside the cell. This imbalance of ions from one side of a cell membrane to the other is called a **potential difference**. Two factors are responsible for the ability of a cell to maintain a potential difference across its membrane:

- The cell membrane has selective permeability. This means that it is relatively impermeable to certain ions and more permeable (lets ions pass through the membrane) to others. However, the permeability of the membrane to an ion can be increased

transiently by certain chemical substances released by nerve endings, as is discussed later.

- The cell can actively move ions across the membrane to maintain a required resting potential.

The potential inside a cell membrane is measured relative to the fluid just outside the membrane (Fig. 3.1). Under resting conditions when no action is occurring, the membrane potential, aptly called the **resting potential,** is negative. Nerve cells, muscle cells, and sensory receptors maintain a negative resting potential in the range of −60 to −90 mV (average = −85 mV) between the inside and outside of their membranes.

A neuron innervating skeletal muscle and the skeletal muscle itself each possess membrane characteristics that allow them to react when a stimulus is provided. This ability to react to a stimulus is called **irritability.** Once nervous and muscular tissues react to a stimulus, the cell's membrane changes its resting potential and it becomes more positive. This process is called **depolarization.** When the nerve or muscle cell membranes are depolarized, they become *excitable* and transmit the electrochemical impulse along their membranes, so that the depolarization propagates, or moves, along the cell's membrane. When this depolarization continues to be transmitted, this impulse, is known as an **action potential.** Action potentials are the *language,* or the electrochemical messages, that are then propagated through the movement system. Let's examine this process step by step.

Think of a light switch turning on a ceiling light: when the switch is moved to the "on" position, a signal is sent through the electrical wire to the light bulb. The body reacts to a stimulus in a similar manner. A sufficient stimulus (electrical, mechanical, chemical, or thermal) applied to a nerve or muscle cell causes the cell membrane to be more permeable to certain ions. This increased permeability results in a rapid exchange of previously separated positive and negative ions as the stimulus moves across the membrane. This rapid ion movement causes the membrane to become more positively charged, or depolarized (Fig. 3.1). The flow of current between immediately adjacent regions serves to excite the polarized region ahead of the current, with the result that this region now contributes a greatly amplified electric signal. With subsequent adjacent regions becoming excited, the action potential propagates or spreads down the length of the axon without a change in its amplitude (intensity) as it moves along the axon. In other words, the excitation that is produced by a stimulus produces a wave of electrochemical activity that moves rapidly along nerve and muscle fibers and is associated with local changes in the electrical potential of each of the fibers. An action potential transmitted over a nerve fiber is a *nerve impulse,* whereas an action potential conducted over a muscle fiber is a *muscle impulse.* Immediately after depolarization, an active process, termed **repolarization,** returns the membrane to its resting potential.

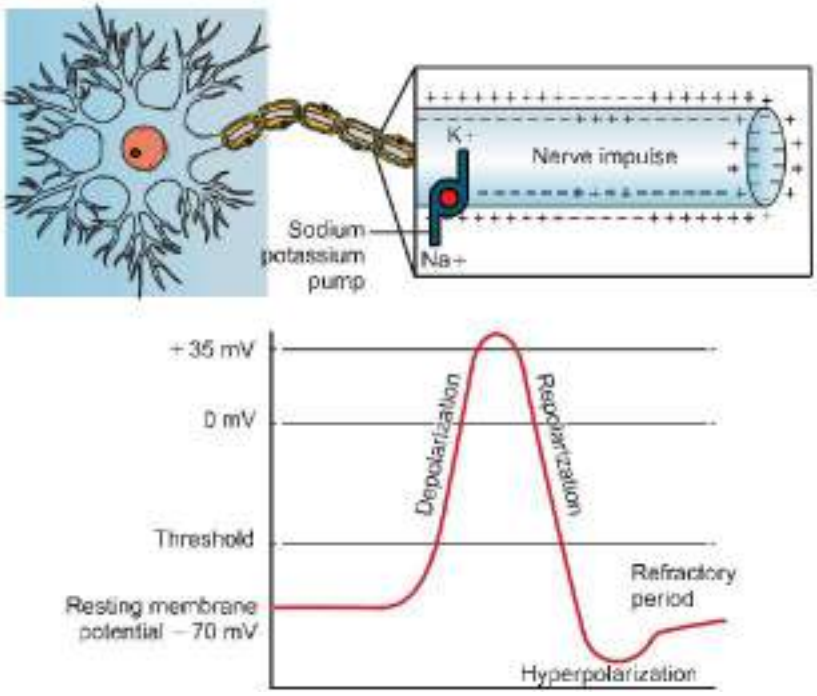

Figure 3.1 Generation of an action potential.

Neurons send "control signals" to other neurons or to muscles by releasing small amounts of chemicals termed **neurotransmitters**. Each time a nerve impulse arrives at the **synapse** (Gr. *synapsis*, a connection), a junction between nerves or at the junction between a motor neuron and a muscle fiber, neurotransmitters are released at the synapse. The chemical synapse between two neurons may be either excitatory or inhibitory. Excitatory synapses cause depolarization of the postsynaptic membrane to produce an action potential. In contrast, inhibitory synapses result in a **hyperpolarization** (more negative potential) of the postsynaptic membrane. This inhibition increases the voltage requirement so it is more difficult to create an action potential.[3]

Nervous System Anatomy Overview

Now that we understand how nerves and muscles "talk" to each other, let's examine the anatomical components of the nervous and muscular systems before we investigate how they work together in a functional movement system.

Nervous System Classifications

The most basic structure of the nervous system is the neuron. Neurons have many different shapes and sizes, depending on their location and functions in the nervous system. A typical neuron consists of a cell body containing the nucleus; several short radiating processes called dendrites; and one long process, the axon, which terminates in twig-like branches. The axon may also have branches or collaterals projecting along its course. The axon, together with its covering, or sheath, forms the nerve fiber.

Beyond comprehending the neuron, the nervous system becomes very complex. To make such a complex system easier to understand, science has divided the nervous system into smaller units. Because the nervous system performs so many functions and is comprised of various structures, it can be divided using a variety of methods. The most common methods used to discuss the nervous system include physiological, anatomical or functional divisions. *Physiologically*, the nervous system is divided into the somatic and visceral nervous systems. The somatic system includes all of the receptors and nerves that innervate muscles and skin. The visceral system is the autonomic system that is further subdivided into the sympathetic and parasympathetic systems. Further discussion of physiology is available in human physiology texts. This text, however, focuses on anatomical and functional classifications of the nervous system as they relate to movement.

Anatomically, the nervous system is divided into the central nervous system (CNS) and the peripheral nervous system (PNS). The central nervous system is composed of the brain and spinal cord and includes all of the nerves that communicate with each other within those areas. These neural structures are enclosed within the bony vertebral column and skull. On the other hand, the peripheral nervous system includes the cranial nerves, the afferent sensory nerves to the spinal cord and the efferent motor neurons from the spinal cord to the muscles. A cross section of a thoracic spinal cord segments and locations of major motor and sensory tracts are illustrated in Figure 3.2. The term **tract** describes a group of axons with common origin, function, and termination. The name of a tract often indicates the general origin and destination of the axons which make up the tract. For example, the spinocerebellar tracts convey sensory impulses from the spinal cord to the cerebellum. Likewise, axons in the corticospinal tract descend from the cerebral cortex and terminate within the spinal cord. General anatomic features of the neural pathways transmitting from the brain to individual muscle fibers are also illustrated schematically in Figure 3.2. Axons of **upper motor neurons** are located in the cerebral cortex and descend in the spinal cord. These upper motor neurons form axonal bundles as corticospinal pathways or tracts. Corticospinal tracts are in the lateral and medial portions of the spinal cord (Fig. 3.2) and are the lateral and medial corticospinal tract, respectively. The axons of the corticospinal tracts make synaptic contact, usually via interneurons, with **lower motor neurons**. Lower motor neurons are in the ventral horn gray matter of the spinal cord. Each lower motor neuron innervates a set of muscle fibers within a muscle. These connections between a nerve and a muscle are illustrated in Figure 3.2.

Functionally, the nervous system is divided similarly to the anatomically based system but with additional clarification. For example, the peripheral nervous system includes afferent and efferent nerves. In functional terms, the afferent system includes all nerves associated with the transmission of sensory information into the CNS. Afferent nerves includes peripheral axons, often called primary or first order afferents, originating from the receptors and entering into the dorsal horn of the spinal cord. Once in the spinal cord, afferent signals synapse and continue transmission within the central nervous system via second and third order afferents at various neural regions between the spinal cord and cortex.

The efferent system includes nerves that regulate movement and motor behavior. The initial efferent nerves within the central nervous system are upper

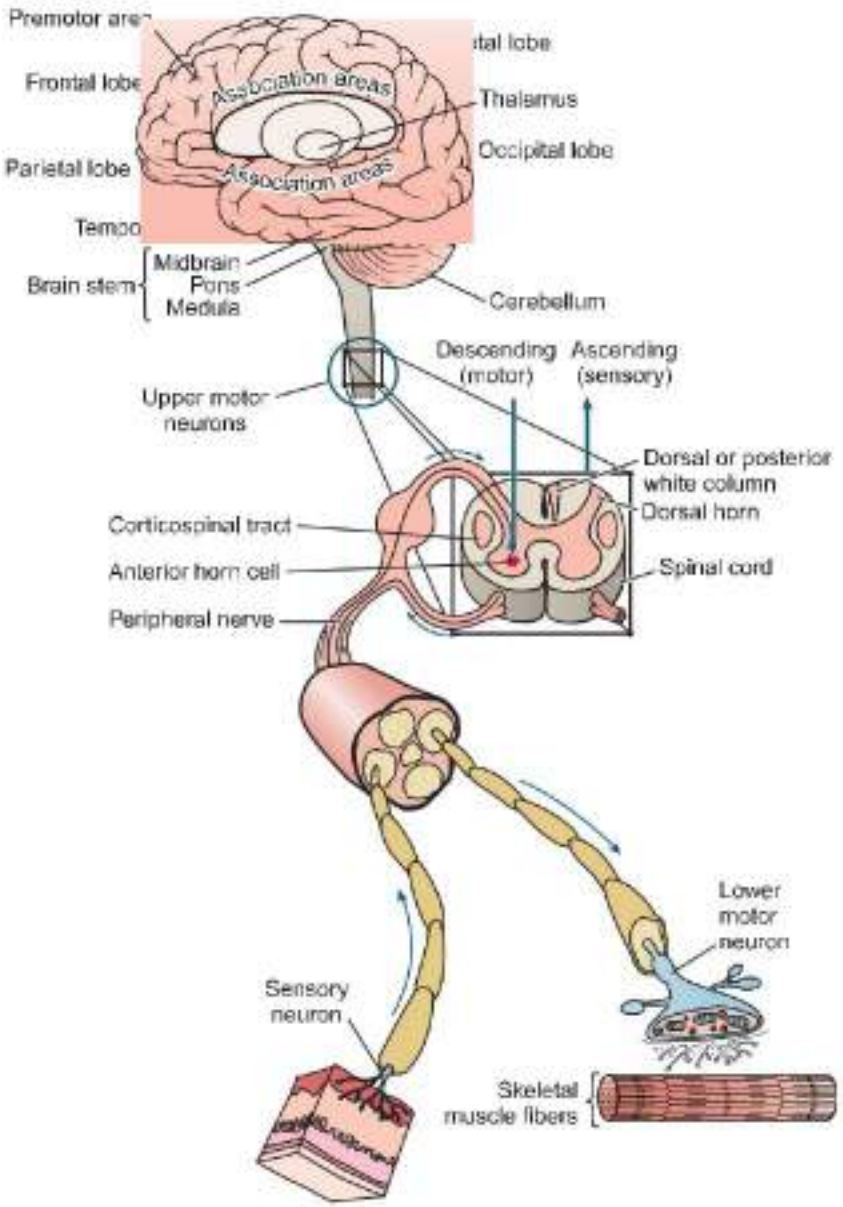

Figure 3.2 Major structures involved in the transmission of impulses to activate skeletal muscle fibers; central and peripheral components of the descending (motor) pathways from the cerebral cortex to the spinal cord and to muscles; cross section of the spinal cord at the thoracic level, illustrating the central gray matter and the peripheral white matter with ascending and descending tracts. Peripheral nerve fibers are enlarged to illustrate sensory and motor components. Note that structures and tracts exist on both the left and right sides; for simplicity, however, only one side is illustrated.

motor neurons since their connections, cell bodies, and axons lie within the brain and spinal cord. **Interneurons** are those neurons within the ventral horn and intermediate areas of the spinal cord. Interneurons transmit efferently to alpha and gamma lower motor neurons. Alpha and gamma lower motor neurons innervate extrafusal muscle fibers and intrafusal muscle fibers, respectively; muscle fibers are discussed in more detail later in this chapter. Interneurons function as a neural *bridge* between the upper and lower motor neurons.

Nerve Fibers

As we know, neurons are the functional structure of the nervous system. Afferent and efferent nerves must relay messages quite rapidly if they are to perform their responsibilities effectively. The motor and sensory nerves are wrapped with a myelin sheath to provide for this necessary transmission speed. Myelin is a white lipid substance that insulates the neural axon. Along this myelin sheath are regular indentations the length of the axon; these are nodes of Ranvier, named after a French histologist, Louis Ranvier (1835–1922). The myelin sheath increases the speed of neural transmission by allowing the excitation to jump along the axon from one node of Ranvier to the next rather than flowing to make contact with the entire axon. Characteristics of sensory and motor neurons are depicted in Figure 3.3.

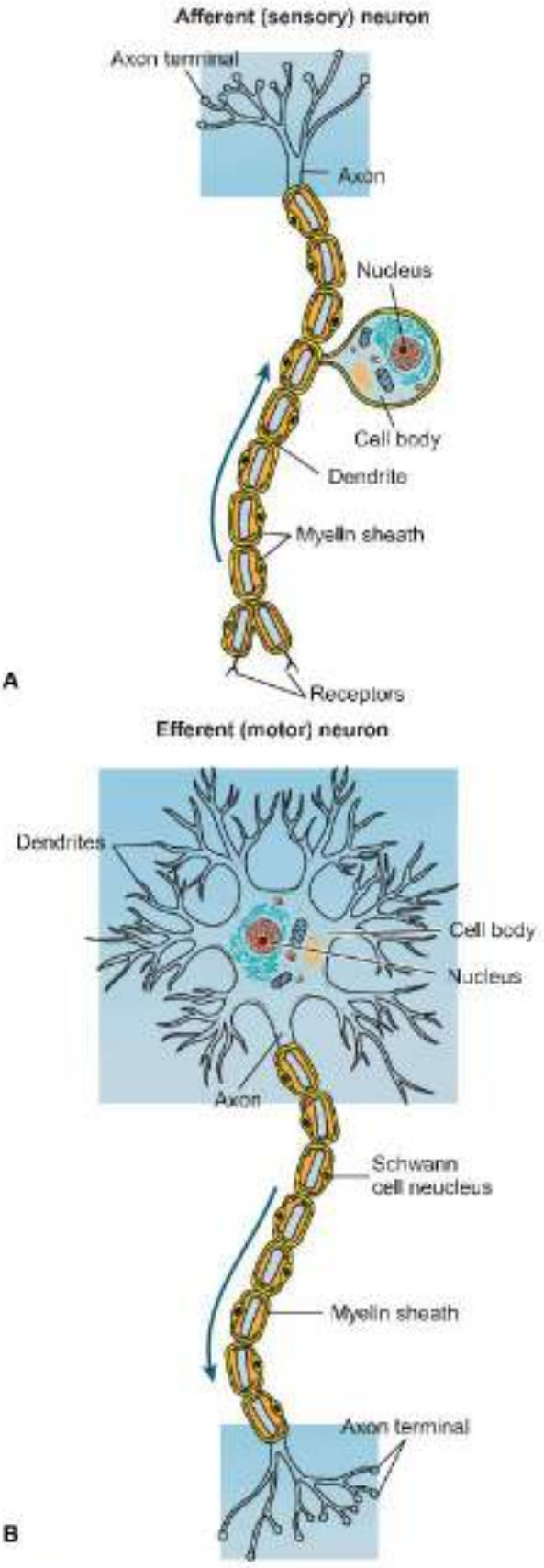

Figure 3.3 Diagrammatic representation of a sensory neuron and a motor neuron.

Nerve Fibers in the Peripheral Nervous System

A peripheral nerve trunk coming off the spinal cord is composed of many nerve fibers, both sensory and motor (Fig. 3.2). Functionally, peripheral nerves include the following fibers:

- Sensory nerves are functionally referred to as afferent nerve fibers. Their cell bodies lie in special **ganglia**. Sensory fibers carry impulses arising from various receptors in the skin, muscles, and special sense organs to the central nervous system, where the impulses are interpreted.
- Motor nerve fibers are functionally referred to as efferent nerve fibers. These motor fibers conduct impulses from the spinal cord to skeletal muscle fibers for voluntary muscle activity control. Their cell bodies are located in the gray matter of the spinal cord and brainstem. Lower motor neuron is the term used to describe a motor (efferent) nerve whose cell body and axon originate in the ventral horn of the spinal cord and synapse directly onto skeletal muscle. It is also often referred to as the *final common path* between the nervous system and the muscular system.
- Autonomic neurons are concerned with the involuntary control of glandular activities and smooth muscles, including smooth muscles surrounding arterioles and venules within muscles. Comprehensive explanations of the autonomic nervous system are beyond the scope of this text but may be found in physiology and neuroscience textbooks.

Classification of Motor and Sensory Nerve Fibers on the Basis of Axonal Diameter in the Peripheral Nervous System

When histologists and anatomists began studying the characteristics of the nervous system, neurons supplying various motor and sensory structures in the body were classified according to the diameter of their axons. Their classification system remains in use today (Table 3–1). The largest axons are classified as type A, and the smallest fibers are C; those of intermediate diameter are referred to as type B. Both A and B fibers are myelinated, whereas C fibers are unmyelinated. Type A fibers are further divided, based on fiber diameter. Type A subdivisions include type A-alpha (α), type A-beta (β), type A-gamma (γ), and type A-delta (δ).

The reason classification according to size is important has to do with the speed of nerve conduction. The speed at which a nerve impulse travels along the length of an axon is related to the diameter of the axon and whether it is enclosed in a myelin sheath. Larger axons conduct impulses at a faster velocity. This makes intuitive

TABLE 3-1 | NERVE FIBER TYPES

Fiber Type	Fiber Diameter (μm)	Conduction Velocity (m/sec)	Peripheral Organ	Function
A-alpha (α) (motor)	12–20	70–120	Skeletal muscle	Motor, skeletal muscle efferent
A-alpha Ia (sensory)	12–20	70–120	Muscle spindle afferent	Proprioception
A-alpha Ib (sensory)	12–20	70–120	Golgi tendon organs afferent	Proprioception
A-Beta II (sensory)	5–12	30–70	Muscle spindle and touch/pressure receptors	Touch, pressure, vibration
A-gamma (γ) (motor)	3–6	15–30	Intrafusal muscle fibers of muscle spindle	Motor, muscle spindle efferent
A-delta (δ) (sensory)	2–5	12–30	Skin	Pain and temperature afferent
B fibers	1–3	3–15	Autonomic Sympathetic	Autonomic efferent
C fibers IV	.5–1	.5–2	Skin, autonomic postganglionic	Pain and temperature afferent

Source: Berbiti, DB. *Functional Neurorehabilitation Across the Life Span*. Philadelphia: F. A. Davis Company, 2004. Reprinted with permission from F. A. Davis Company.[1]

sense, and a simple comparison helps us understand this concept. If you compare the largest-diameter nerve to a garden hose and the smallest-diameter nerve to a straw, it is much faster to move one gallon of water through the hose than through the straw. As we have already discussed, adding a myelin sheath causes the axon to conduct an impulse even faster. Type Aα are the largest myelinated axons (diameter = 20 μm) and conduct an impulse at a maximum velocity of approximately 120 m/s. The longest sensory and motor axons extend from the lumbar spinal cord segments to the foot muscles, a distance of approximately 1 meter in an adult of average height. Therefore, a minimum of 8 msec (0.008 second) is needed for a nerve impulse to travel the length of type Aα axon. As seen in Table 3–1, the smallest nerve fibers, type C (0.5 μm in diameter), convey nerve impulses from sensory endings in the skin that appear to produce sensations of pain when stimulated. Pain impulses are conducted at a velocity of approximately 0.5 m/s.

Classification of Sensory Fibers on the Basis of Fiber Origin within the Peripheral Nervous System

Afferent nerve fibers within the peripheral nervous system may also be classified according to the type of sensory receptor from which impulses are conducted. This method includes four groups. The first group (group I) is subdivided into subgroups Ia and Ib. Group Ia fibers carry impulses from the primary sensory receptor in muscles, the **muscle spindle**. Group Ib fibers carry impulses from sensory receptors located in tendons and are referred to as **Golgi tendon organs (GTO)**. The Golgi tendon organs are located at the interface of a muscle with its tendon. Diameters of group Ia and Ib fibers are approximately 12 to 20 mm. Muscle spindles and Golgi tendon organs are type A-α fibers. Group II fibers are equivalent to type A-β in diameter size (4 to 12 μm) and carry impulses from the secondary receptors in the muscle spindle.[6] The structure and function of muscle spindles and GTOs are described in the section on receptors.

Classification of Motor Fibers on the Basis of Fiber Destination within the Peripheral Nervous System

Efferent nerve fibers are classified into two groups based on which muscle fiber that they innervate. Alpha (α) motor neurons innervate **extrafusal** skeletal muscle. **Gamma (γ) motor neurons** innervate the contractile element called the **intrafusal** (within the spindle) **muscle fibers**. As the name indicates, the intrafusal muscle fibers lie within the muscle spindle while extrafusal muscle fibers are normal skeletal muscle fibers.

Nerve Fibers in the Central Nervous System

As with the peripheral nervous system classifications, the CNS also has different methods of classifying the nerves which comprise it. Within the central nervous

system, nerves are classified typically by their physical characteristics such as size or shape. For example, the giant cortical neuron is so named for its size, and a pyramidal neuron is named for the shape of its cell body.

Another way of classifying neurons references their function. For example, the *association* neuron is a neuron within the association cortices of the brain. These nerves communicate with each other within the brain and literally create associations between neural areas, similar to the bridging function of the interneurons within the spinal cord; hence, their name—**association neurons**.

As previously mentioned, all sensory and motor axons travel in tracts within the CNS. Recall that tracts consist of bundled axons and are named for their origin, location and destination. Because many nerve fibers are covered with a myelin sheath, tracts appear white in unstained histologic sections; therefore, the term **white matter** is used to describe areas in the central nervous system that contain predominantly fiber tracts. Within various regions of the central nervous system, aggregations of anatomically and functionally related neurons (cell bodies) are distinguished from one another, and these aggregates are referred to as **nuclei** or **ganglia**. Regions of the central nervous system in which nerve cell bodies are concentrated appear gray in color because they are not covered with myelin; these sections are referred to as **gray matter**. The central region of the spinal cord contains nerve cell bodies (gray matter) surrounded by tracts of myelinated axons (white matter) (Fig. 3.2). Some tracts carry ascending or sensory impulses whereas others carry descending motor impulses. In the cerebrum, the cell bodies and tract positions reverse; the cortex appears gray because cell bodies of cortical neurons lie in the superficial surface layers. Tissue beneath the gray matter is white because here is where the myelinated axons that connect cortical neurons with other regions of the CNS are located.

Upper motor neurons lie within the central nervous system and carry impulses from the brain to motor neurons in the spinal cord, and **lower motor neurons** of the peripheral nervous system transmit motor impulses from the spinal cord to activate skeletal muscle fibers. Some neurons, termed **interneurons**, reside entirely within the spinal cord and transmit impulses from one neuron to the dendrites or cell body of another neuron nearby.

Most neurons discharge nerve impulses intermittently; that is, the neurons exhibit a level of firing even while at "rest." The frequency of discharge is modified by the influence of other neurons. Both facilitatory and inhibitory stimuli are continually transmitted from motor centers in the brain to interneurons throughout the spinal cord. Motor neurons receive synaptic connections from thousands of other neurons. Whether a given motor neuron becomes more active or less active depends on the net effect of all the facilitatory and inhibitory stimuli that arrive at the motor neuron at any given instant.

Afferent nerves also have various possible connections when they enter the spinal cord. After entering through the dorsal horn of the spinal cord, the sensory axon may give off a branch that synapses with interneurons in the spinal cord. However, the main fiber usually ascends through the spinal cord to synapse with other neurons in the central nervous system. A peripheral neuron with an uninterrupted axon like this is a **first order neuron.** Sensory neurons that receive synaptic input from a peripheral sensory neuron (first order neuron) and then carry the impulse to the brainstem and other lower centers in the central nervous system are **second order neurons.** Second order neurons frequently transmit the impulse to **third order neurons**, which are located in higher centers of the central nervous system.

Muscular System

Cognizant that the nervous and sensory systems constantly coordinate and refine our movements, we next examine muscles in terms of their structure and function. Like nerves, muscles are also excitable and respond dynamically; similarly, just as an action potential occurs as a single excitation and response of a nerve, a muscle twitch results as a single excitation and response of a muscle. (Fig. 3.4).

Structure of Skeletal Muscle

Just as the neuron is the basic element of the nervous system, the muscle fiber is at the core of the muscular system. Although very different from neurons, muscle fibers are also complex in their structure.

Muscle and Muscle Fiber Structure

Each segment of the body contains several skeletal muscles (Fig. 3.5A). A muscle is surrounded by a thin connective tissue covering called **epimysium.** The epimysium helps to keep each muscle separate from adjacent muscles. If we delve into a muscle, we see that another connective tissue layer called perimysium subdivides the muscle. **Perimysium** divides a muscle into sections within the entire muscle. Each subsection of a muscle is a **fasciculus** (Fig. 3.5B). If we move deeper into the muscle's **fasciculi**, we realize that they are made up of lots of muscle fibers. The muscle fibers are the basic structure of muscle. **Muscle fibers** are **muscle cells**

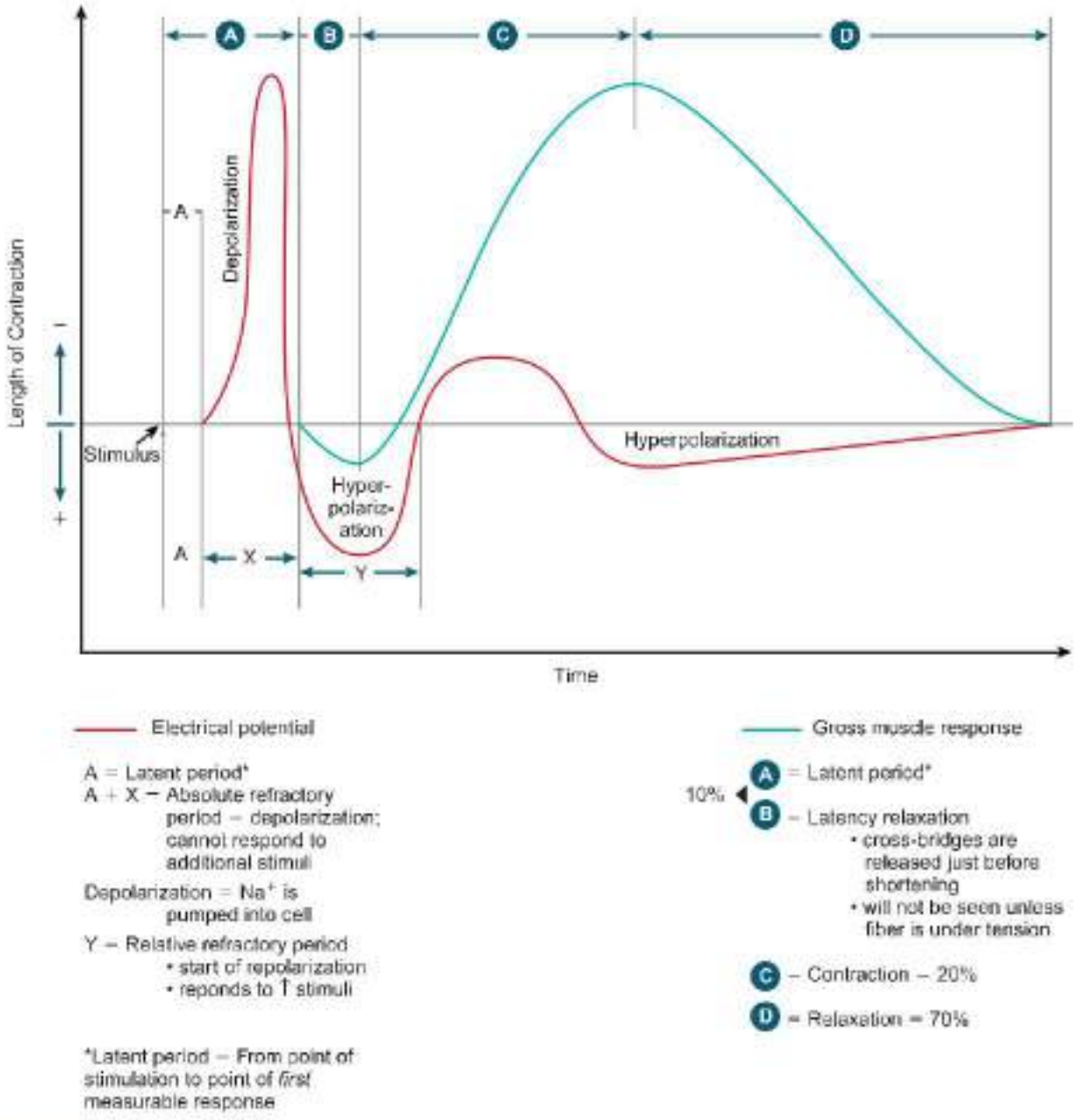

Figure 3.4 Time relationship of the electrical, chemical, and mechanical responses in a simple muscle twitch.

(Fig. 3.5C). Each muscle fiber is made up of multiple rod-like myofibrils, spanning the entire length of the muscle fiber (Fig. 3.5D). **Myofibrils** are bundles of filaments within a muscle fiber; myofibrils are also called **myofilaments**. The length of a muscle fiber varies from a few millimeters to many millimeters. The diameter of an individual muscle fiber ranges from 10 to 100 **micrometers (μm)**. Each muscle cell has several nuclei. Each myofibril has a covering or membrane, the **sarcolemma**, and is composed of a gelatin-like substance, sarcoplasm (Fig. 3.5C). Hundreds of muscle fibers and other vital structures, such as mitochondria and the sarcoplasmic reticulum, are imbedded in the sarcoplasm. Mitochondria serve as "tiny factories" where metabolic processes occur.

A myofibril (Fig. 3.5D) is composed of units, and each unit is referred to as a **sarcomere**. A sarcomere lies between two Z-lines. Between these two Z-lines are many myofilaments. **Myofilaments** are made up of fine threads of two protein molecules, **actin** (thin filaments) and **myosin** (thick filaments) (Fig. 3.5E). These two filaments provide skeletal muscle with the appearance of

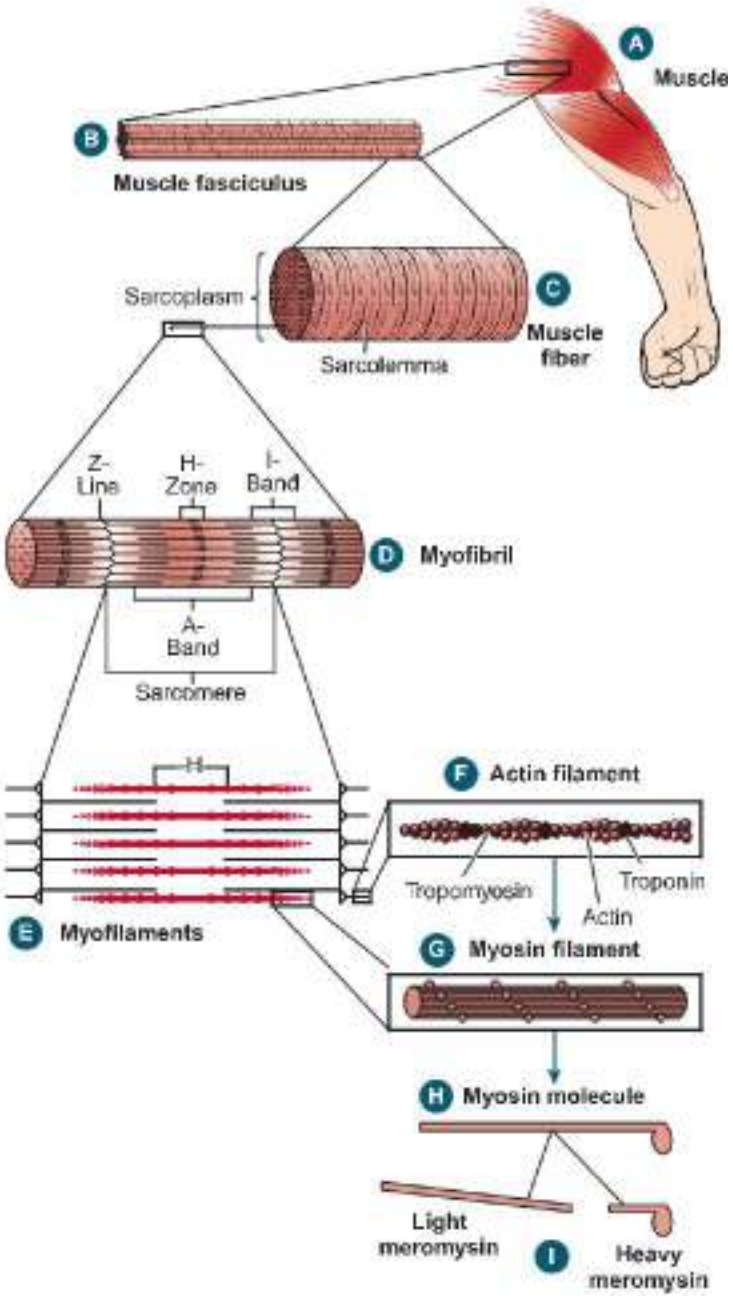

Figure 3.5 Diagram of the organization of skeletal muscle at rest, from **A)** the gross to **E)** the molecular levels. **F, G,** and **H** are cross sections of the myofibrils at the levels indicated. **I** is a diagram of the composition of the myofilaments. (Adapted from Bloom, W. and Fawcett, D.W. *A Textbook of Histology*, ed 10. WB Saunders, Philadelphia, 1975, p 306.)

light and dark striations, which is why skeletal muscle is referred to as "striated" muscle. The striations are alternate bands of light and dark light-refractive materials that, when viewed under a microscope, are seen to be alternately lighter and darker bands (Figs. 3.5D).

The darker band in skeletal muscles, referred to as the anisotropic or A-band, contains both actin and myosin filaments (Fig. 3.5F–I). In the three-dimensional arrangement of these filaments, six actin filaments surround each myosin and three myosin filaments (Fig. 3.5) surround each actin filament. A-bands have an isotropic middle zone: the H-band which contains only myosin filaments (Fig. 3.5D, G). The lighter band in skeletal muscle—the isotropic or I-band—contains only actin filaments and is bisected vertically by a Z-line (Fig. 3.5D, F). One end of each actin myofilament within the I-band is anchored to the Z-line.

The thin actin filaments of the I-bands contain two proteins, troponin and tropomyosin (Fig. 3.5F). Actin is polymerized (linked together) to form two-stranded filaments that are twisted together (Fig. 3.5F) to form part of the actin filament. Tropomyosin is a rod-shaped molecule and composed of two separate polypeptide chains that are wound around each other to form a long,

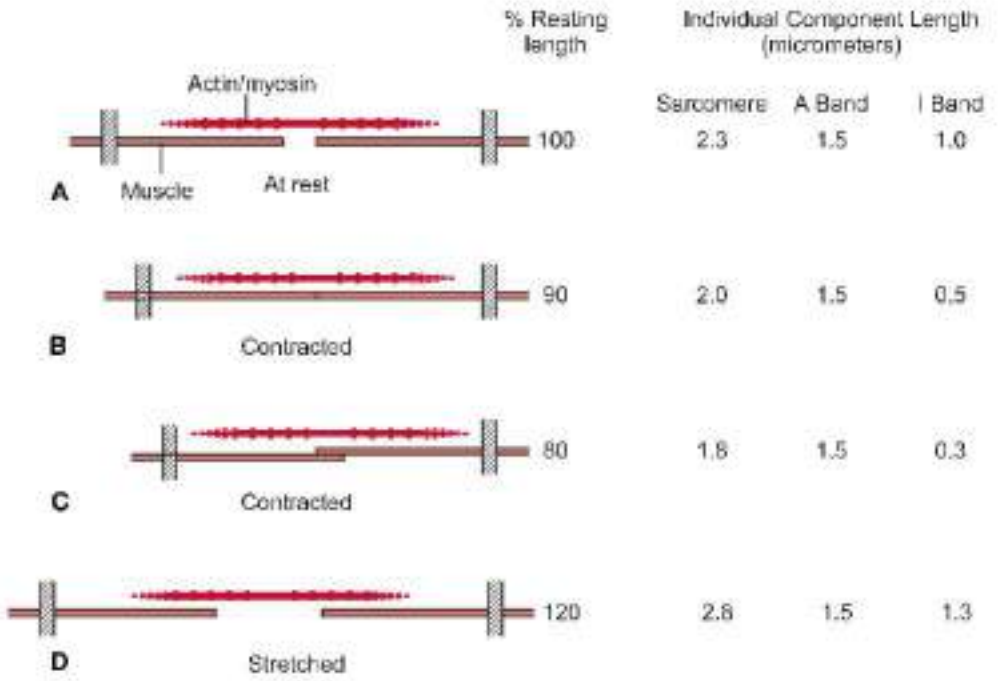

Figure 3.6 The structural basis for changes in muscle length: **A)** A sarcomere at resting length showing changes **B, C)** in arrangement of filaments under various degrees of contraction; and **D)** when stretched. Relative changes in length of the sarcomere are indicated on the *left*, and the approximate lengths (in micrometers) of the sarcomere, the A-band, and the I-band are listed on the *right*. Note the constancy of the A-band length. (Adapted from Schottelius, BA, and Schottelius, DD: *Textbook of Physiology*, ed 17. St. Louis: CV Mosby, 1973, p 87.)

rigid insoluble chain. Tropomyosin is about 40 nm long. These straight molecules are arranged along the actin filament so one tropomyosin is coupled with approximately six actin molecules (Fig. 3.5F). Troponin is a regulatory protein that is bound to a specific region of the tropomyosin filament. This arrangement provides one troponin globule per 40 nm of tropomyosin filament. Regulatory proteins, such as troponin, impact the interactions between an actin filament and its adjacent myosin filaments. An important function of troponin is based on its enormous avidity for calcium ions (Ca^{++}), a property that is important in activating the contractile process. This arrangement of actin, tropomyosin, and troponin forms the actin filaments in the sarcomere. In simple terms, the functional purpose of actin is to provide a binding site for the myosin during a muscle contraction.

Myosin filaments (Fig. 3.5G) are thicker than actin filaments and are composed of myosin molecules (Fig. 3.5H). Myosin consists of polypeptide chains, one pair of heavy chains and two pairs of lighter chains, which are coiled together into one large chain (Fig. 3.5G-I). Myosin molecules form a rod about 1.6 μm long and 1.5–2.0 nm in diameter, about 1/10,000 of the diameter of a hair from your head. The end of each heavy chain has a globular structure that forms two "heads" of myosin. These heads look similar to the distal end of a hockey stick (Fig. 3.5). These globular heads are at the end of an "arm" portion that is "hinged" to the myosin. These "hinges" allow the arms to project out laterally from the myosin filament and move during muscle activation. These heads are called **crossbridges** because they bridge the thick filaments to the thin filaments during muscle activity (Fig. 3.8). Crossbridges are not present at the central portion of the myosin filament, and the crossbridges on the two halves of the myosin project in opposite directions. These crossbridges align in groups of three called "crowns." Each consecutive crown is positioned such that it is rotated on the myosin from the previous crown. This arrangement provides crossbridges for each actin that is adjacent to the myosin. There are about 300 to 400 crossbridges on a 1.6 μm-long myosin filament.[7] Myosin exhibits enzyme-like qualities capable of splitting adenosine triphosphate (ATP) into adenosine diphosphate (ADP) and phosphate (PO_4) plus energy. The significance of this reaction is discussed in the section dealing with the energetics of muscle contraction.

Investigators using light and electron microscopy have observed relaxed and contracted states of muscle tissue. The length of each serially repeating sarcomere unit is approximately 2.5 μm when the muscle is relaxed (Fig. 3.6A). The length of each sarcomere decreases to about 1.5 μm when the muscle is fully contracted (Fig. 3.6C). In contrast, the sarcomere unit may be increased to about 3.0 μm when the muscle is stretched (Fig. 3.6D).

As mentioned, a sarcomere is secured at each of its ends by a Z-line (Figs. 3.5D and 3.7). Widths of individual A-bands do not change during contraction. However, the I-band where only actin filaments are seen does become narrower, and the H-zone, where only myosin filaments are seen within the A-band, is obliterated. These observations demonstrate that the free ends of the actin filaments slide toward each other into the central H-zone of the A-bands when muscles contract. As the actin filaments move toward each other, the Z-lines are pulled closer together so that the I-bands shorten (Fig. 3.6A, B, C). Although the amount of shortening of each sarcomere unit is small (0.5–1.0 μm) the shortening of several thousands of these sarcomere units linked in series produces a noticeable reduction in the entire muscle's overall length. For example, a muscle fiber 10 cm in length, like the biceps brachii muscle, has approximately 40,000 sarcomere units lined up end to end.* If each of these 40,000 sarcomere units shortened by 1 μm, the ends of the entire muscle fiber would move 40,000 μm (or 4 cm) closer together. Thus, an overall shortening of 40% of the length of the muscle occurs.

This concept of actin and myosin filaments sliding past each other to produce muscle contraction is known as the **sliding filament model** of muscle contraction.[8,9] The specific way in which actin filaments are drawn past myosin filaments to develop muscle tension and muscular shortening is complex. Simply summarized, however, the myosin's heads attach to and pull on the actin causing the actin filament to slide over the myosin into the H-zone to shorten the sarcomere during muscle contraction (Fig. 3.5 E, Fig. 3.8). Let us delve deeper into the sliding filament model to gain additional understanding of how muscles contract.

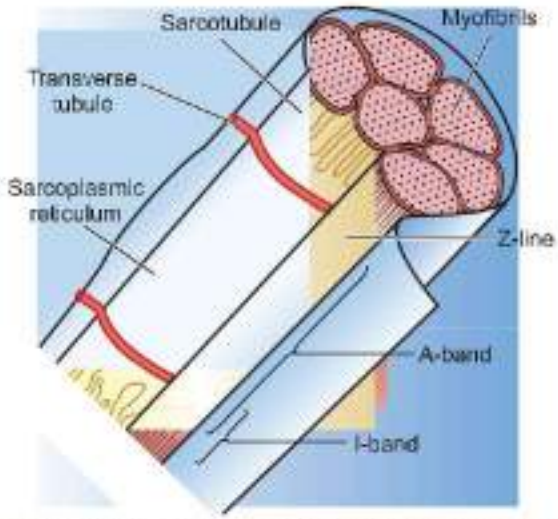

Figure 3.7 Endoplasmic reticulum of the skeletal muscle fiber. Sketch of the fine structure of part of a muscle fiber based on an electron micrograph. The cell membrane invaginates at the level of each Z-line, sending transverse tubules across the interior of the fiber. Between the Z-lines and parallel to the myofibrils runs the sarcoplasmic reticulum, of which sacklike enlargements (the terminal cisternae) adjoin the transverse tubules.

Myoneural Junction: Transmission of Impulses from Nerves to Skeletal Muscle Fibers

The nervous system regulates the activity of muscle fibers by sending control signals in the form of action potentials. Conversion of a nerve impulse to a muscle impulse, however, occurs through a complex process. The nerve fiber branches at its end to form a motor end plate, which adheres tightly to surface of the muscle fiber but does not penetrate the muscle fiber membrane (Fig. 3.2). This connection is a type of synapse referred to as the **myoneural** (Gr. *myo*, muscle, plus *neuron*, nerve) **junction**, commonly referred to as the **neuromuscular junction**. The motor neuron end plate contains mitochondria that synthesize a neurotransmitter, acetylcholine. Molecules of acetylcholine are stored in small vesicles located in the presynaptic ending of motor neurons. The arrival of a nerve impulse at the myoneural junction causes release of acetylcholine from some of the vesicles. When freed from storage in the vesicles, acetylcholine diffuses rapidly across the short distance between the motor end plate and muscle fiber membrane. Acetylcholine then interacts with receptor sites on the muscle fiber membrane. The interaction increases the permeability of the muscle cell membrane to ions in the fluid bathing the junction. Movement of these ions into the muscle cell depolarizes the muscle fiber (postjunctional) membrane and triggers a muscle action potential that moves along the muscle fiber by an

*1 cm = 10^4 μm = 10,000 μm; thus, the 10 cm muscle fiber = 100,000 μm in length. 100,000 μm divided by 2.5 μm per sarcomere = 40,000 sarcomere units.

electrochemical mechanism similar to that of a nerve impulse (Fig. 3.4).

After causing increased permeability at the postjunctional membrane, acetylcholine is rapidly inactivated by an enzyme, cholinesterase. Cholinesterase is present in the fluid bathing the synaptic space and immediately splits acetylcholine when it comes in contact with it. The very short time that acetylcholine remains in contact with the muscle fiber membrane, about 2 msec, is usually sufficient to excite the muscle fiber, and yet the rapid inactivation of acetylcholine by cholinesterase prevents re-excitation after the muscle fiber repolarizes.

Conduction of Muscle Impulses to the Interior of the Muscle Fiber: Endoplasmic Reticulum

Change in electrical potential in the immediate vicinity of actin and myosin filaments triggers a process that leads to shortening of each sarcomere. The interior of a muscle fiber contains two interlaced systems of tubes that play an important role in excitation of and contraction of muscle fibers (Fig. 3.7). One system, the **transverse tubular system (T-system)**, runs perpendicular to the myofibrils and speeds the transmission of a muscle action potential to all portions of the muscle fiber. The other system, the **sarcoplasmic reticulum (SR)**, is found deep to the sarcolemma, running parallel and superficial to the myofibril. The sarcoplasmic reticulum stores and releases calcium ions during the contractile process. The two systems, the transverse tubular system and the sarcoplasmic reticulum, together comprise the **endoplasmic reticulum** (Fig. 3.7).

Excitation–Contraction Coupling

Energy must be supplied to myofilaments to cause movement of the actin filaments toward the center of the A-bands. Energy for this purpose is available from adenosine triphosphate (ATP) molecules, which are coupled to myosin crossbridges. The energy is provided when myosin acts as a **catalyst** to split molecules of ATP into adenosine diphosphate (ADP) and inorganic phosphate (P_i). Calcium stimulates the myosin to split ATP. This process is called myosin **ATPase activity**. The following section describes how this process works.

Sliding Filament Model of Muscle Contraction

A sequential series of events explains how sliding filaments develop tension and shorten (Fig. 3.8). Projections of **crossbridges** are located on the myosin myofilaments. At rest, the crossbridges between myosin and actin myofilaments lie perpendicular to the myosin filaments and are prevented by regulatory mechanisms from making contact with the actin filaments (Fig. 3.8A). Also, at rest, calcium is stored in the SR, and the ATP molecules are coupled near the end of each crossbridge (Fig. 3.8A). Potential reactive sites on actin myofilaments are covered by troponin and, therefore, are not available to the myosin crossbridges.

When a pulse of depolarization descends the T-tubules, quantities of calcium are released from storage sites in the SR. Some of the calcium ions interact with troponin, causing a deformation in the shape of

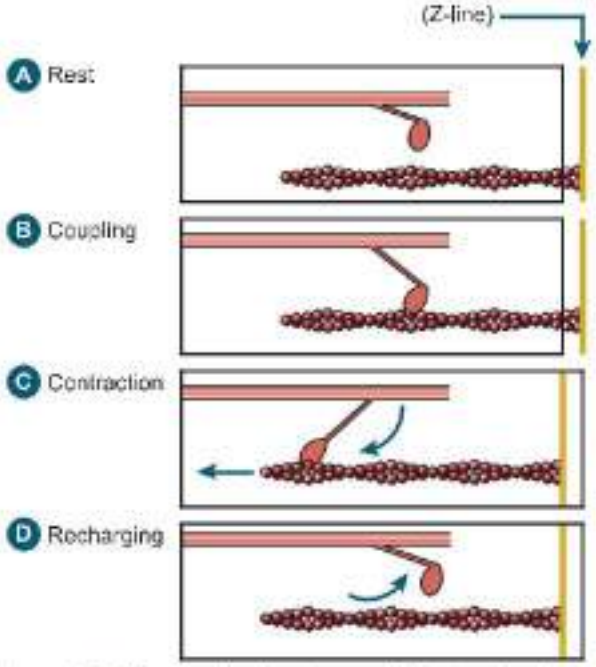

Figure 3.8 The hypothesized sequential series of reactions between active sites on actin and myosin filaments that pull the actin filament along the myosin filament to produce shortening of the sarcomere. The recovery process is also illustrated. Many repetitions of the cycle at a large percentage of the active sites are needed to produce a strong contraction. **A)** Rest. Crossbridges project from a myosin myofilament but are not coupled with an actin myofilament. Adenosine triphosphate (ATP) is attached near the head of the crossbridge; troponin covers the active sites on the actin myofilament; calcium ions are stored in the sarcoplasmic reticulum. **B)** Coupling. Arrival of the muscle action potential depolarizes the sarcolemma and T-tubules; calcium ions are released and react with troponin; change in the shape of the troponin-calcium complex uncovers active sites on actin; a crossbridge couples with an adjacent active site, thereby linking myosin and actin myofilaments. **C)** Contraction. Linkage of a crossbridge and an active site triggers adenosine triphosphatase (ATPase) activity of myosin; ATP splits to adenosine diphosphate (ADP) + PO_4 + energy; the reaction produces a transient flexion of the crossbridge; the actin myofilament is pulled a short distance along the myosin myofilament; Z-lines are moved closer together. **D)** Recharging. The crossbridge uncouples from the active site and retracts; ATP is replaced on the crossbridge. The recoupling, flexion, uncoupling, retraction, and recharging processes are repeated hundreds of times per second.

the troponin molecule (Fig. 3.8B). The changes in shape caused by the interaction of calcium with troponin uncover an active site on the thin actin filament that electrostatically attracts the myosin crossbridge. So, in the presence of calcium, myosin and actin are attracted to each other and a globular head of a myosin filament and an active site on an actin filament forms an actomyosin crossbridge. The actomyosin crossbridge, in turn, triggers the ATPase activity of myosin to cause ATP to split into ADP plus phosphate and produce energy. This chemical energy is converted into mechanical energy by the muscle cell to produce motion. Mechanically, this ATP hydrolysis (split into ADP and P_i) produces transient "flexion" of the crossbridge (Fig. 3.8C), which pulls the actin filament a short distance. Once this motion occurs, the ADP and P_i are released to allow reformation of ATP which binds once again to myosin. In the presence of ATP, the myosin's affinity for actin diminishes and linkage is broken (Fig. 3.8D). A cycle of coupling, flexion, uncoupling, retraction, recharging, and recoupling repeats hundreds of times each second as long as calcium and ATP are present. If either calcium is removed from the myoplasm or the ATP supply is exhausted, the cycle of actomyosin crossbridge formation and release ceases.

Muscle Relaxation

As depolarization of the muscle fiber ends (5–10 msec), intracellular calcium concentration drops very quickly, and relaxation occurs. The rapid drop in intracellular calcium results from an active "pumping" of calcium ions from the region of the myofilaments back into the SR storage sites. Active transport of calcium against a concentration gradient continues until the concentration of calcium remaining in the intracellular fluid bathing the myofilaments reaches a very low resting-condition level. Removal of calcium ions from the vicinity of the actin filaments results in the troponin returning to its original shape, covering the active sites on the actin filament; thus, the actin and myosin filaments return to their "resting," relaxed state. In effect, an insufficient concentration of intracellular calcium ceases actin and myosin myofilament interaction.

Muscle Fiber Types

To most efficiently perform various functions, different types of skeletal muscle fiber exist. Early research of muscle fibers classified an entire muscle as either fast or slow, based on speeds of shortening.[10, 11] With more recent investigations, an expansion in identification of main fiber types has evolved to include two types: type I and type II, with a further subdivision of type II into type IIa and type IIb.[10, 11] Some investigators have described a more detailed breakdown of fiber types,[11–14] but, for the purpose of this text, only the two main types of muscle fibers are discussed. Each type of fiber has different properties. Most skeletal muscles contain a mixture of both types, with the proportion of one type more predominant than the other type in a muscle. More importantly, the functional unit of muscle contraction, the **motor unit**, which includes the alpha motor nerve and all of the muscle fibers that it innervates, can be classified based on contractile speed, as detailed below.[10, 15, 16]

The **type I** muscle fiber appears dark (like the dark meat of a domestic chicken[*]) because it contains large numbers of mitochondria and a high concentration of myoglobin. Myoglobin is muscle hemoglobin that stores oxygen. Type I also is referred to as **slow-twitch, tonic,** or **slow oxidative (SO)**, because biochemically, these fibers depend on aerobic or oxidative energy metabolism.[11, 12, 18]

The **type II** muscle fiber appears paler (like the white meat of a domestic chicken[*]) because it contains fewer mitochondria and only small amounts of myoglobin. Type II, further subdivided into IIa and IIb, are fiber

PRACTICE POINT

Live muscle has a continual availability of ATP for muscle activity. Rigor mortis is a condition caused by a lack of ATP with an arrest of the actomyosin crossbridge cycle with attachment of the two myofilaments in the presence of calcium. Since there is no ATP, the actin and myosin filaments remain attached until muscle tissue begins decomposing. Rigor mortis is a condition that occurs around 3 hours after death and remains to some degree for up to 72 hours.

[*] A different situation exists in birds that predominantly fly.

types that are fast-twitch in their contractile speed but which rely on glycolytic (anaerobic) processes and oxidative (aerobic) metabolic processes. Type II muscle fibers, also termed **fast-twitch, phasic,** or **fast glycolytic (FG)** or **fast oxidative glycolytic (FOG),** are larger in diameter than type I muscle fibers. Type IIa are the fast oxidative glycolytic and type IIb are the fast glycolytic. Type IIa fibers may seem like a "transition" fiber type between the slow oxidative type I and the fast glycolytic type IIb since it possesses characteristics of each. Type IIb fibers develop a greater contraction force and complete a single twitch in a significantly shorter time than type I muscle fibers. Type IIb fibers, however, fatigue more quickly than type I fibers. Type I fibers are innervated by small diameter axons of the motor nerve and are recruited first in a muscle contraction. On the other hand, type IIb fibers are innervated by larger diameter motor axons and are recruited after type I and type IIa fibers.[17] Table 3–2 summarizes the characteristics of each fiber type.

All human muscles contain various proportions of these different types of muscle fibers. Postural muscles such as the soleus and erector spinae, which are vital for stabilizing the body in positions such as standing for long periods of time, are largely composed of type I fibers, whereas muscles involved in large or quick bursts of activity, such as the biceps brachii, are composed largely of type IIb fibers. Some investigators suggest that the proportions of fast-twitch and slow-twitch muscle fibers in a particular muscle can vary from subject to subject.[20-23] Despite subject-to-subject variation, the proportion of slow-twitch (type I) fibers is high in postural control muscles such as the human soleus muscle in the leg (as great as 85% of the fibers) and low in quick-moving, refined movement muscles such as in the orbicularis oculi of the eyeball (10%).[18] Generally, the more type I muscle fibers contained within a muscle, the more it provides postural stability, and the more type IIb fibers contained within a muscle, the more it provides rapid movement over short periods.

Muscle fiber development and adaptation are good examples of how the movement system can change in response to the demands placed upon it. Muscle fibers can adapt to changing demands by changing ratios of fiber types within muscles.[10, 19] For example, type II fibers are predominant at birth, as evidenced by the characteristically quick jerky movements typical of a newborn baby. As the infant develops postural control, an increase in type I fibers occurs. By the time the child is about 2 years old and has upright postural control, balance, and antigravity control, the ratio of type I and II fiber types is similar to the ratio seen in adults.[1, 20] Muscle fiber development and adaptation is also illustrated in children with developmental disabilities such as cerebral palsy, who demonstrate differences in the morphological properties of skeletal muscle.[21] Fiber types change again with aging, resulting in decreases in the total number of both fiber type I and type II fibers and selective atrophy of the type II fibers, and conversion of fiber types.[10,22-27] These age-associated changes are correlated with some of the declines in motor performance seen in older persons, such as decreased

TABLE 3–2 | CHARACTERISTICS OF SKELETAL MUSCLE FIBERS BASED ON PHYSICAL AND METABOLIC PROPERTIES

Muscle Fiber Type			
Property	**Type I Slow-Twitch SO**	**Type IIA Intermediate FOG**	**Type IIB Fast-Twitch FG**
Muscle fiber diameter	Small	Intermediate	Large
Color	Red (dark)	Red	White (pale)
Myoglobin content	High	High	Low
Mitochondria	Numerous	Numerous	Few
Oxidative enzymes	High	Intermediate	Low
Glycolytic enzymes	Low	Intermediate	High
Glycogen content	Low	Intermediate	High
Myosin ATPase activity	Low	High	High
Major source of ATP	Oxidative phosphorylation	Oxidative phosphorylation	Glycolysis
Speed of contraction	Slow	Intermediate	Fast
Rate of fatigue	Slow	Intermediate	Fast

SO = slow oxidative; FG = fast glycolytic; FOG = fast oxidative-glycolytic.
Source: Burke, RE and Edgerton, VR. Motor unit properties and selective involvement in movement. *Exer Sport Sci Rev* 3:31, 1975.[10] Adapted with permission.

postural control, coordination and dexterity challenges, along with functional problems such as **hypokinesis** (decreased activity) and frequent falls. There is evidence to indicate that muscle fiber types can change from fast- to slow-twitch fibers with exercise or electrical stimulation.[21, 28] Conversely, muscles that experience disuse demonstrate a transformation in the opposite direction, from slow to fast.[21, 28]

This fluctuation of fiber type has implications for rehabilitation, serving as the physiological basis for numerous therapeutic and exercise science intervention programs. Although a fiber-type specification within individuals is thought to be genetically determined, training and rehabilitation can alter contractile and metabolic properties of muscle fibers, allowing for improved responses to functional demands. For example, placing a high metabolic demand on muscle, such as in endurance training, results in an increased oxidative capacity for all muscle fiber types and leads to a conversion from fast glycolytic to fast oxidative glycolytic muscle fibers. In other words, percentages of pure type IIb fibers decrease and the percentages of type IIa fibers increase with endurance training.[32, 34, 35] Researchers have found that the type I fibers become faster and type II fibers convert to slower, more oxidative types with endurance exercise. The converse is also true with type I fibers becoming more plentiful and slower with deconditioning.[29-31]

The Motor Unit

Motor neurons that activate efferent motor responses are located either in the brainstem or in the spinal cord. Those in the brainstem are for the muscles of the face and head, and those in the spinal cord send impulses to the muscles of the neck, trunk, and extremities. Specifically, the motor neurons in the spinal cord are located in the gray matter of the ventral (anterior) horns (Fig. 3.2). Various types of motor neurons exist. The majority, if not all, of the neurons that innervate skeletal muscles are within the A, alpha (α), size classification, and known as **alpha (α) motor neurons**. Motor commands travel from the neuronal cell bodies over peripheral nerve fibers and then across the neuromuscular junction. The number of muscle fibers innervated by a single motor nerve fiber varies from as few as five, as in some of the eye muscles, to as many as 1,000 or more, as in large muscles such as the gastrocnemius. The more control required of a muscle, the fewer muscle fiber to nerve fiber ratios a muscle has. On the other hand, muscles that produce large forces without the need for fine control have much larger ratios of muscle fibers to nerve fibers. The number of motor units and the average number of muscle fibers per motor unit are later summarized in Table 3–3.

As the term "motor unit" implies, all muscle fibers act as one unit, contracting or relaxing nearly simultaneously. Muscle fibers of one motor unit are not adjacent to one another; they are distributed throughout the muscle's length. In addition, if the motor unit's nerve activates its muscle fibers to contract, those fibers will contract maximally. This principle is the **all-or-none law**.

Gradation of Strength of Muscle Contraction

Increased strength of a muscle contraction, as a whole, occurs in three ways and is based on different principles:

- Size principle: The smallest motor units are activated first.
- Recruitment principle: Increasing the number of motor units activated simultaneously increases the overall muscle tension.
- Excitatory input/rate coding principle: Increasing the frequency of stimulation of individual motor units increases the percentage of time that each active muscle fiber develops maximum tension

PRACTICE POINT

Changes in fiber type composition may also be at least partially responsible for some of the impairments and disabilities seen in older persons. As the elderly remain active and experience injuries, the clinician must realize that muscle fiber compositions in this age group will require changes in rehabilitation expectations in areas such as maximal strength or balance abilities. Evidence indicates that strength exercises are beneficial for older persons and these individuals will experience the same types of gains as younger individuals, but the maximal level will be lower than younger patients.[32] Additionally, exercise interventions, especially endurance training, can affect muscle fiber types leading to improvements in conditioning, balance, and performance.[32]

The size principle of recruitment describes the fact that the smallest motor neurons are the first to be recruited and the largest motor neurons are recruited last.[38] Small motor neurons participate in most sustained activities because they tend to innervate the slow-twitch, type I muscle fibers that fatigue slowly. When muscle functions require greater strength, the largest fast-twitch and more quickly fatiguing motor units become active. In terms of recruitment order, the smaller motor units are recruited first. Since these smaller motor units have fewer muscle fibers per nerve, more must be recruited to produce force of a specific level. For example, if a small motor unit is able to produce 0.05 kg, it would take 100 small motor units to produce 5 kg of force. On the other hand, the large motor units produce greater force since there are a greater number of muscle fibers in each motor unit. For example, if a large motor unit is able to produce 0.20 kg, 100 large motor units would produce 20 kg of force. Of course, these numbers are exaggerated, but they serve to explain the concept. In summary, a muscle successively fires its small motor units first. Once those units are all recruited, the larger motor units are recruited in order of their size from smallest large unit to the largest large motor unit. In other words, motor units are normally recruited in an orderly pattern with those which produce low force recruited first, followed by higher force-producing units as force requirements increase.[39]

Firing of a single motor unit results in a twitch contraction of the stimulated muscle fibers. With an increase in firing rate, these twitches summate to increase and sustain a force output. An individual increases muscle force by increasing both the number of active motor units and the firing rates of those active motor units.

Joint, Tendon, and Muscle Receptors

Specialized receptors are present in joint structures, tendons, and skeletal muscles. Because these afferent receptors gather information about one's own joints and joint movements, they are called **proprioceptors** (L. *proprio*, one's own, plus *captive*, to receive). These receptors detect changes in tension and position of the structures in which the receptors are situated. A pattern of nerve impulses is generated in the receptor and transmitted to other parts of the nervous system. As a result, moment-to-moment changes in joint angle (position of the joint), speed of joint motion, amount of joint compression or distraction, as well as changes in muscle length, rate of change in muscle length, and force of muscle contraction are relayed to centers in the spinal cord and brain. In the central nervous system, this information is integrated with that coming in from other sensory organs. Additional sensory organs include the eye's retina and the inner ear's vestibular apparatus, both of which provide input on position, balance, and motion. Integrated sensory signals then are used by motor control centers in the brain to automatically adjust the location, type, number, and frequency of motor unit activation so that appropriate muscle tension is developed to perform desired movements.

Joint Receptors

Several different types of sensory receptors are in joint capsules and ligaments. The major anatomic features of various sensory receptors are illustrated in Figure 3.9. Most of these receptors emit several action potentials per second as a "resting" output, so the body always has a sense of position in space. The receptor is stimulated when it is deformed. Depending on the location and magnitude of deforming forces acting on the joint and receptor location, certain receptors are stimulated and discharge a high-frequency burst of nerve impulses when the joint moves. Receptors typically adapt, which means that the frequency of impulses decreases after movement ceases and then transmit a steady train of nerve impulses thereafter. Further movement of the joint may cause one set of receptors to stop discharging impulses and another set to become active. This continual flow of information allows the nervous system to continually appraise joint position and of the rate of joint movement.

PRACTICE POINT

The largest large motor units are not recruited for normal daily activities. There is some indication that these motor units are not recruited voluntarily. Evidence suggests that these motor units are recruited during times of extreme stress. During these times the autonomic system provides extraordinary functions such as when a nonathletic individual jumps an 8-foot fence to save someone in a house fire.

Figure 3.9 Schematic illustration of types of receptors that are usually distinguished in joints. A diagram of the knee joint, showing the distribution of various receptor types in the capsule and ligaments of the joint. The menisci are free from nerve fibers except at their attachment to the fibrous capsule.

Golgi tendon organs (GTOs) lie within muscle tendons near the point of their attachment to the muscle (Fig. 3.10). An average of 10 to 15 muscle fibers is usually connected in direct line with each GTO. Because they are oriented in line (or in series) with the collagen fibers of the tendon and the muscle fibers, the GTO is ideally suited to detect force or tension in either muscle or tendinous collagen fibers but not changes in muscle length.[40] The GTO is stimulated by tension produced within the muscle fibers or the collagenous tendon to which it is attached. Physiologic data indicate that the GTO responds through force-related neural discharge because it is selectively sensitive to the forces produced by in-series muscle fibers. Nerve impulses discharged by the Golgi tendon organ are transmitted over large, rapidly conducting afferent axons (group Ib fibers) to the spinal cord and cerebellum. In turn, several efferent messages are dispatched. These efferent messages go to the agonist (contracting) muscle to inhibit it and to that muscle's antagonist to facilitate it. Inhibiting the agonist limits that muscle's force production to a level that can be tolerated by the tissues being stressed by the contraction. The GTOs thereby mediate nonreciprocal inhibition, or **autogenic inhibition,** referring to this inhibitory input to an agonist muscle (prime mover) and an excitatory message to the antagonist (opposing) muscle.

Muscle Spindles

Skeletal muscles are composed of **extrafusal** (L. *extra,* outside of or in addition, plus *fusus,* spindle) **fibers,** which are "regular" or skeletal muscle fibers. Lying within muscles, parallel to the extrafusal fibers, are unique proprioceptors called **muscle spindles,** so named for their shape. These small but complex organs have multiple functions, both sensory and motor in nature.

Very specialized muscle fibers, called **intrafusal muscle fibers (IFMF),** lie within muscle spindles. Each muscle spindle contains 3 to 10 of these specialized muscle fibers. The intrafusal fibers of the muscle spindle are encapsulated within a connective tissue sheath

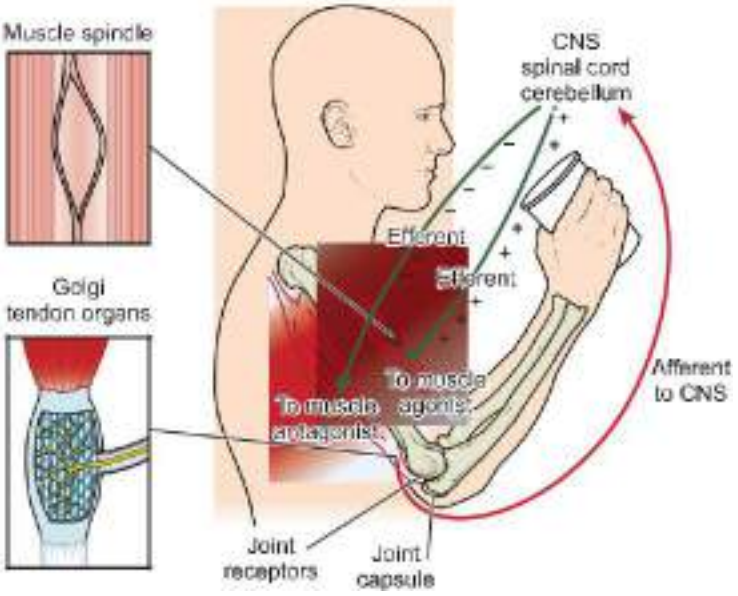

Figure 3.10 A schema to illustrate the anatomic relations among extrafusal muscle fibers, a motor unit, a muscle spindle and a Golgi tendon organ with the attached afferent projection. The force or tension produced by contracting the extrafusal muscle fibers causes the structural fiber network (collagen and elastin) to collapse around the Golgi tendon organ and the muscle spindle to be activated. The muscle spindle activates the agonist while inhibiting the antagonist whereas the Golgi tendon organ does the opposite: inhibits the agonist and activates the antagonist.

PRACTICE POINT

Autogenic inhibition can be effectively applied to therapeutic stretching techniques, especially in situations in which the patient is extremely anxious about movement due to pain. Stretch the muscle to the end of the range and then ask the client to actively isometrically contract the muscle against your resistance. The muscle will then relax temporarily due to the GTO and stretching into an increased range becomes facilitated. In this technique, called hold-relax, the limb is held by the clinician at the end of the muscle's range of motion (for example, the hamstrings), and the patient is asked to perform an isometric or "holding" contraction at that point in the motion. After the ensuing relaxation of the isometric hold, the limb is then more easily moved into the newly achieved range.[1, 41]

(Fig. 3.11). There are two types of morphological arrangements of the intrafusal fiber nuclei: nuclear bag and nuclear chain fibers. These intrafusal fiber names describe their specific anatomical configuration.[42, 43] The nuclear bag intrafusal muscle fiber has its nuclei grouped in the middle (looks like a bag) of the fiber, whereas the nuclear chain has its nuclei spread along its length, in a chain-like arrangement. Both the nuclear bag and the nuclear chain are surrounded in a spiral fashion by branches of an Ia (also called primary sensory ending) afferent neuron. A secondary neuron ending is formed by group II afferent fibers and found primarily on nuclear chain fibers.[44]

Two types of gamma (γ) axons supply the nuclear fibers with motor innervation. The static gamma motor nerve axons supply the nuclear chain fibers, and the dynamic gamma motor nerve axons supply the nuclear bag fibers. These gamma nerve axons are narrower than the alpha motor axons supplying the extrafusal muscle fibers, so intrafusal muscle fibers respond slower to stimuli than do the extrafusal muscle fibers. However, this unique anatomy illustrates that muscle spindles have both sensory and motor functions.

Focus on the Sensory Function of the Muscle Spindle

As part of the afferent or sensory system, muscle spindles function as a stretch receptor. Muscle spindles send sensory impulses over the Ia and II afferent axons that "inform" other neurons in the spinal cord and brain of their length and, therefore, of the length of the extrafusal muscle and of the rate at which a muscle stretch occurs. This muscle spindle's receptor function transpires because of its parallel alignment with the extrafusal muscle fibers. Therefore, a change in the intrafusal fiber length is associated with a change in the extrafusal fiber's length.

There are varying numbers of muscle spindles located within different muscles. Muscle spindles are present in skeletal muscle and are most numerous in the muscles of

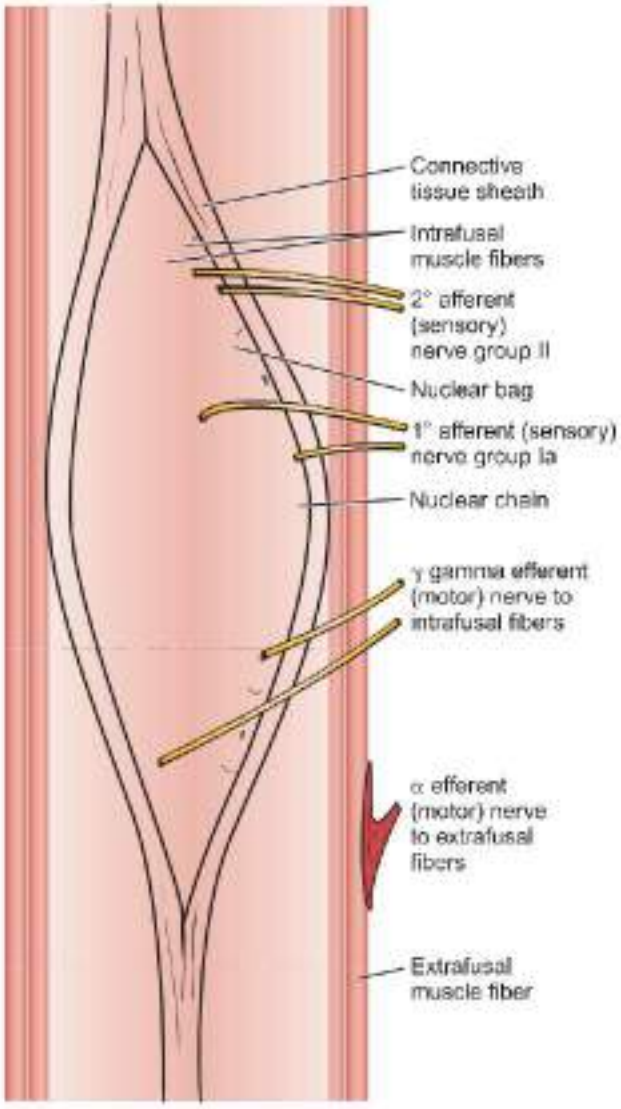

Figure 3.11 The muscle spindle. This diagram shows the anatomic relationships among the major components of a muscle spindle. Although most muscle spindles contain 3 to 10 intrafusal fibers, for simplicity, only 3 sets are shown. Nuclear bag fibers and nuclear chain fibers also are pictured, as well as the afferent and efferent nerve supplies. Extrafusal muscle fibers alongside the muscle spindle and an α motor neuron to the extrafusal fiber also are shown.

the arms and legs (Table 3–3). Muscle spindles are especially abundant in the small muscles of the eye, hand, and foot; all of these muscles have a very high muscle spindle density because of the need for these muscles to be constantly alerted to even small changes.

As a muscle lengthens or shortens, the degree of stretching or relaxation of its intrafusal fibers alters the activity in the Ia and II sensory fibers that innervate them. The Ia afferent fiber detects both the amount of stretch and the velocity of the stretch, thereby exhibiting qualities of both **phasic** (Gr. *Phasis*, an appearance, a distinct stage or phase) and **tonic** (Gr. *tonikos*, continuous tension) activity. On the other hand, the secondary (II) receptor is purely tonic, responding primarily to the amount of stretch. Therefore, the primary and secondary receptors behave differently. As the muscle contracts or stretches to change the length of its extrafusal muscle fibers, its muscle spindles detect this length change and depolarize the Ia afferent sensory nerve wrapped around each muscle spindle. This Ia nerve also has a critical velocity threshold, so it also detects a length change but only if this change exceeds a certain rate or velocity. When this sensory nerve notes a muscle stretch of a sufficient velocity and depolarizes, it sends impulses into the dorsal horn (where all sensory information enters the spinal cord), where it connects with other neurons. Via a monosynaptic reflex, it makes a direct connection to an efferent nerve, an alpha motor neuron (in the anterior horn cell), which then transmits a signal back to the extrafusal muscle fibers in the same muscle as the muscle spindle. This process ceases when the stretch ends. Another pathway the incoming sensory afferent nerve takes is an additional connection through an interneuron (disynaptic) to a different efferent alpha motor neuron that transmits a signal to the antagonist muscle, signaling that muscle to relax.

The monosynaptic component of this example is also known as the **deep tendon reflex** or **stretch (or myotatic) reflex**, a simple reflex arc mediated at the spinal cord level. Afferent nerves (group Ia) from the primary receptor make an immediate synaptic connection with the motor neurons (A, α motor neurons) that control extrafusal muscle fibers in the same muscle. Therefore, an abrupt stretch of a muscle initiates a burst of impulses from the primary stretch receptor in the muscle spindle, which travels to the spinal cord and excites activity in motor units of the same muscle (Fig. 3.12). We have all experienced this reflex connection when a doctor tests it by tapping a reflex hammer on a muscle tendon. When a muscle shortens, the stretch on the muscle and its muscle spindles is relieved, thereby removing the stretch receptor stimulus.

The neural and muscular structures that participate in the stretch reflex are illustrated using a patellar tendon tap in Figure 3.12. The presence of a reflex contraction in the stretched muscle 100 to 200 msec after tapping the tendon demonstrates an intact circuit. In addition, the briskness and relative amplitude of the reflex contraction reflect the general level of excitability of α motor neurons innervating the stretched muscle.

Focus on the Motor Function of the Muscle Spindle

As mentioned earlier, the cell bodies of the gamma (γ) efferent nerves are located in the ventral or anterior horn of the spinal cord. These gamma cells receive synaptic connections and influences from regions throughout the nervous system including the cortex, cerebellum, and

TABLE 3–3 | NUMBER OF MOTOR UNITS, MOTOR FIBERS, AND MUSCLE SPINDLES PER MOTOR UNIT IN HUMAN MUSCLE

		NUMBER OF MUSCLE FIBERS		NUMBER OF MUSCLE SPINDLES	
Muscle	**Number of Motor Axons**	**Per Muscle × 10^2**	**Average Per Motor Unit**	**Per Muscle**	**Per Motor Unit**
Biceps brachii	774	580	750	320	0.4
Brachioradialis	330	130	390	65	0.2
First dorsal interosseus	119	41	340	34	0.3
First lumbrical	98	10	110	53	0.6
Opponens pollicis	133	79	595	44	0.3
Masseter	1020	1000	980	160	0.2
Temporalis	1150	1500	1300	217	0.2
Gastrocnemius medius	580	1000	1720	80	0.1
Tibialis anterior	445	270	610	284	0.6

Source: Adapted from Buchthal, F, Schmalbruch, H. Motor unit of mammalian muscle. *Physiol Rev* 60:95, 1980.[180]

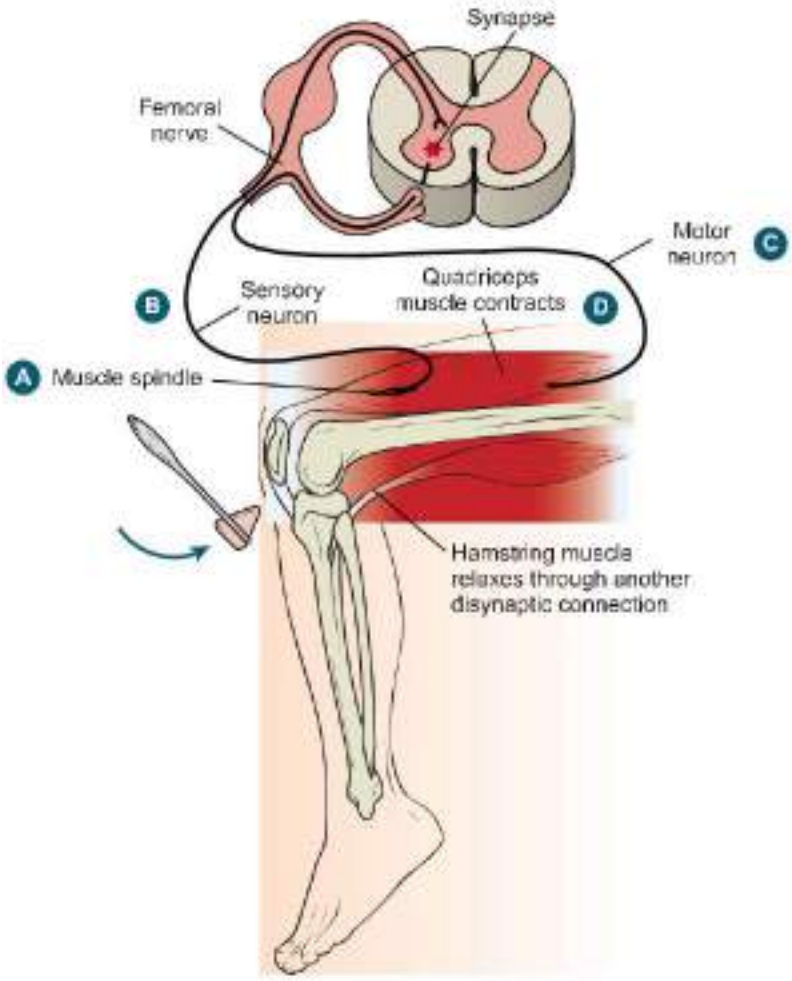

Figure 3.12 The stretch reflex elicited upon a tendon tap. Four fundamental parts of the simple stretch reflex arc are: **A)** A receptor in the muscle generates nerve impulses in proportion to the degree of deformation; **B)** An afferent neuron conducts the burst of sensory impulses from the receptor to the spinal cord; **C)** An efferent neuron conducts motor impulses from the spinal cord to extrafusal muscle fibers; and **D)** An effector, the muscle, responds to the motor impulses.

PRACTICE POINT

Active and passive stretching techniques utilize the neuroanatomical connections described above. These connections provide the basis for the rationale behind active stretching, whereby a patient is asked to actively contract a muscle to shut off its opposing muscle so the "shut off" muscle's stretch is more effective. For example, if a patient actively contracts the quadriceps muscles, an induced relaxation of the hamstrings occurs, allowing a more effective stretch of the hamstrings. In addition to the benefits of improved stretching of a tight muscle resulting from this neural reciprocation is the fact that this agonistic muscle contraction with concomitant antagonistic relaxation provides important functional ramifications. This is known as **reciprocal inhibition** and allows for some of the fluidity seen in movement. For example, when the quadriceps is activated to deliver a forceful kick, the hamstrings receive signals to relax during midrange and are activated again at the end of range to slow the motion and prevent injury (fig. 3.13 and 3.14).

brainstem. The amount of shortening of the contractile portions of the muscle spindle regulates the stretch receptor portion of the muscle spindle. With extrafusal shortening, the length of the spindle is correspondingly adjusted to maintain its sensitivity to additional changes in length or stretch. This relationship is a highly important property of muscle spindles. In essence, as α motor neurons stimulate the contraction of extrafusal fibers, γ motor neurons discharge, causing contraction of the intrafusal (muscle spindle) fibers. The contraction of the intrafusal fibers adjusts the sensitivity range for changing lengths of the muscle.

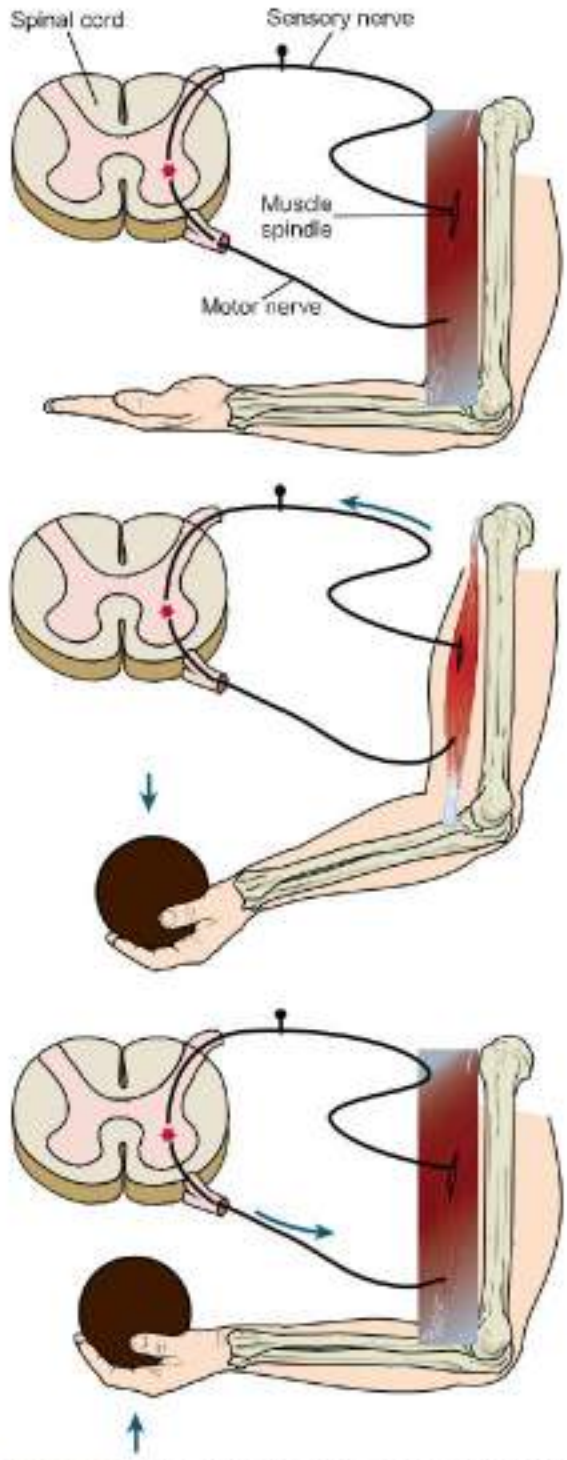

Figure 3.13 Schematic representation of the stretch reflex regulation of muscle length. A muscle is under the influence of the stretch reflex when the muscle is engaged in a steady contraction of a voluntary nature, as when a person's elbow is flexed steadily. A sudden unexpected increase in the load stretches the muscle, causing the sensory ending on the muscle spindle to send nerve impulses to the spinal cord, where the impulses contact a motor nerve cell at the synapse and excite it. As a result, motor impulses are sent back to the muscle, where the impulses cause the muscle to contract. More complicated nerve pathways than the one shown may also be involved in the stretch reflex. Any actual muscle is, of course, supplied with many motor nerve fibers and spindles. In addition, the synaptic connections to even a single motor neuron are multiple.

Gamma motor neurons also are referred to as **fusimotor** (L. *fusus*, a spindle plus *movere*, to move) neurons because the neurons supply motor impulses to the intrafusal muscle spindle fibers. Thus, the middle, noncontractile part of the muscle spindle can be stretched by two different mechanisms. First, when the entire skeletal muscle is stretched, the muscle spindle also is stretched. Second, when the contractile portions at each end of the muscle spindle are activated by impulses arriving over γ motor nerves, the contractile portions shorten, thereby stretching the central "bag" portion of the muscle spindle. In either situation, stretch of the nuclear bag portion of the muscle spindle activates one or both types of sensory receptors residing within the muscle spindle—that is, the primary (Ia) and secondary (II) stretch receptors (Fig. 3.11).

This constant volley of regulatory input onto the muscle spindle's intrafusal fibers sets up a constant state of readiness so that although the muscle is not activated, it is literally on a steady state of alert, ready to act when needed. This constant state of readiness is called **muscle tone**, characterized by an innate amount of muscle stiffness and resting tension. Tone is determined by the level of excitability of the entire pool of motor neurons controlling a muscle, the intrinsic **stiffness** of the muscle itself, and the level of sensitivity of many different reflexes. The contribution of the muscle spindle is only one piece of the puzzle contributing to the phenomenon called muscle tone.

Normal muscles exhibit a firmness to palpation, considered to be typical or "normal" muscle tone. The firmness present in muscles is observed at rest, even in muscles of well-relaxed subjects. The firmness, however, is impaired if the motor nerve supplying the muscle is not intact or the muscle is atrophied. Relaxed muscles exhibit at least a palpable amount of muscle tone, but investigators have failed to detect any muscle action potentials to account for this tone.[45-47] Thus, the tone of relaxed muscles in persons with an intact neuromusculoskeletal system appears to be the result of basic physical properties of muscle, such as elasticity, viscosity, plasticity, and the innate stiffness of the tissue.

Postural tone is a term used to describe the development of muscular tension in specific muscles that maintain body segments in their proper relationships to maintain posture. Postural tone is accompanied by recordable electrical activity from active motor units. Muscles used most often to maintain an erect position of the body are **antigravity muscles**. Muscles of the trunk, flexor muscles of the upper extremities, and extensor muscles of the lower extremities are considered antigravity muscles. Motor centers supply nerve

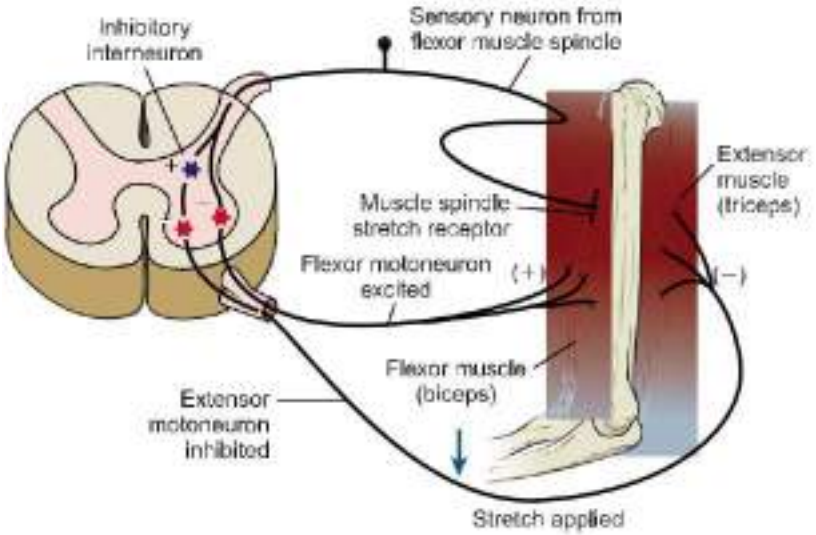

Figure 3.14 Schematic representation of the reciprocal inhibition of motor neurons to the opposing muscle. Impulses from a stretched muscle excite motor units in the same muscle (facilitatory synaptic influence is designated with a plus [+] sign) and inhibit, through an interneuron, motor units in the opposing muscle (inhibitory synaptic influence is designated with a minus [−] sign).

impulses that influence the excitability of lower motor neurons in the spinal cord segments supplying antigravity muscles. These motor centers include the cerebral cortex, basal ganglia, facilitating and inhibiting centers in the midbrain, brainstem reticular formation, and the cerebellum (Fig. 3.15). Postural tone is an automatic (reflexive) phenomenon that is influenced by both afferent impulses from sensory receptors and efferent mechanisms from γ motor neurons.

In functional terms, postural muscle tone in individuals without a pathological condition has been described as "high enough to hold the head, body and extremities against gravity yet low enough to allow for movement."[48] The appropriate amount of muscle tone ensures that the muscle is ready to resist any change in position to maintain posture. In persons with an intact neuromusculoskeletal system, descending motor tracts from the brainstem, particularly from the reticulospinal and vestibulospinal tracts, deliver low frequency trains of impulses to spinal motor neurons, either indirectly through interneurons or directly (Fig. 3.16). Although local postsynaptic depolarizations may not be great enough to provoke complete depolarization and firing of the cell, they maintain the neuron in a slightly oscillating state of high excitability, ready to respond to more concentrated presynaptic input. Muscle tone also ensures that the muscle is ready to contract or relax promptly when appropriate control signals reach the motor neurons. Muscle tone may be influenced by disease or injury affecting various levels of the nervous system resulting in symptoms of insufficient muscle tone (low tone, **hypotonia**) or excessive muscle tone (high tone, **hypertonia**), both to be elaborated upon in later sections of this chapter.

Summary of Muscle Spindle Functions

In essence, muscle spindles function as "thermostats," comparing the length of the muscle spindle with the length of skeletal muscle fibers that surround the muscle spindle (Fig. 3.11 and 3.13). If the length of the surrounding extrafusal muscle fibers is less than that of the muscle spindle, the frequency of muscle spindle's discharged nerve impulses is reduced since they are not being facilitated. However, when the central portion of the muscle spindle is stretched because of γ-efferent activity, its sensory receptors discharge more nerve impulses to excite α motor neurons and activate the extrafusal muscle fibers to contract. The mechanism is particularly important in the regulation and maintenance of postural muscle tone.

Kinesthesia and Proprioception

Under most conditions, a person can be consciously aware of the position of the various parts of his or her body relative to all other parts and whether a particular part is moving or still. This awareness is **kinesthesia** (Gr. *kinen*, to move, plus *aisthesis*, perception) and **position sense**. These two terms are often treated as synonyms and are used frequently to cover all aspects of this awareness, whether static or dynamic. Strictly speaking, however, the term *kinesthesia* regards awareness of dynamic joint motion, and the term *position sense* refers to the awareness of static position. Kinesthetic signals are generated in various sensory receptors residing in muscles, tendons, and joints and respond to body movements and tension within tendons. The impulses produced in the receptors are transmitted predominantly over group II afferent fibers to the spinal cord, the cerebellum, and sensory nuclei. Thus, other sensory and motor centers in the central nervous system are

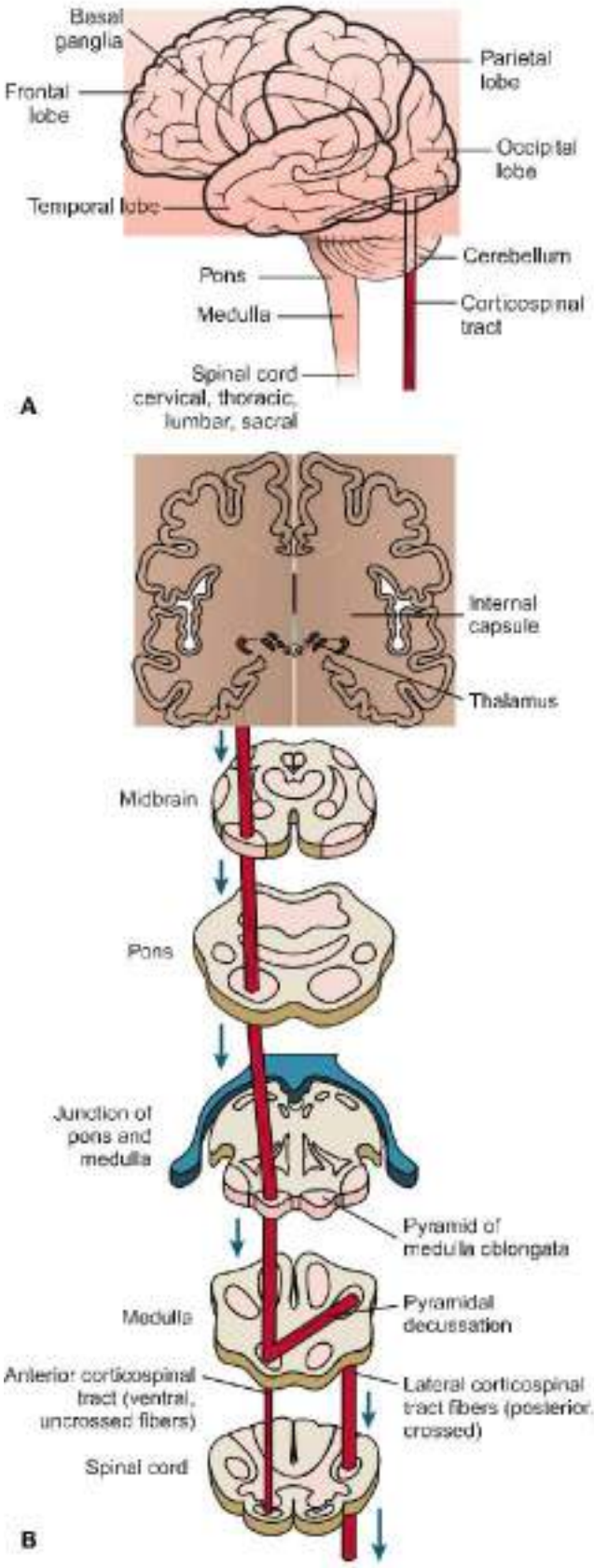

Figure 3.15 **A)** Central nervous system areas primarily involved in the control of movement; **B)** shows a cross-sectional view from the internal capsule and thalamus and inferior structures of the midbrain and spinal cord.

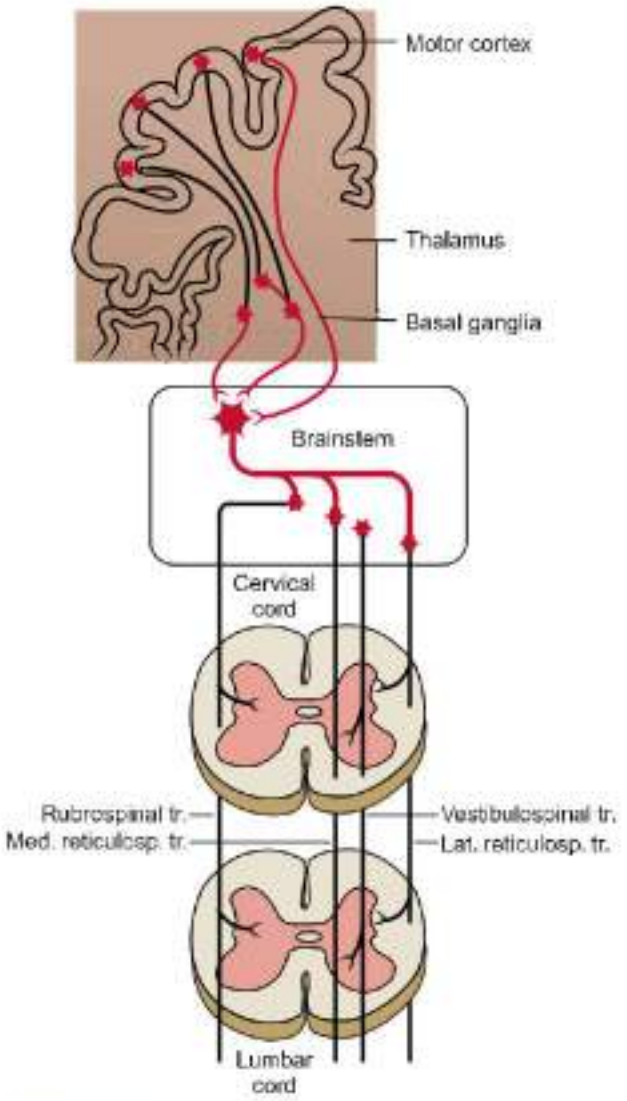

Figure 3.16 Schematic diagram of courses of important tracts that do not cross within the pyramids of the medulla, contributing to supraspinal motor control to the spinal cord. The neuron with a thick axon in the brainstem symbolizes the crossing of most of these extrapyramidal motor fibers to the opposite side at that level and does not imply convergence. Pathways from the motor cortex to the nuclei in the brainstem are partly collaterals of the corticospinal tract and partly separate efferents. Details of connectivity among the brainstem structures involved in motor activity are extremely complicated; this representation is greatly simplified. Note that the structures and tracts exist on both the left and right sides; however, for simplicity, only one side is illustrated.

"informed" of the exact locations of different parts of the body at each instant to assist in controlling posture and movement.

Proprioception (L. *proprio*, one's own, plus *ception*, to receive) is a more inclusive term than kinesthesia and refers to the use of sensory input from receptors in muscle spindles, tendons, and joints to discriminate joint position and joint movement, including direction,

amplitude, and speed, as well as relative tension within tendons. Proprioceptive impulses are transmitted predominantly over group I afferent fibers and are integrated in various sensorimotor centers to automatically regulate postural muscle adjustments and maintain postural equilibrium.

Several types of **somatosensory** (Gr. *soma*, body; plus L. *sensorius*, pertaining to sensation) inputs also are important in maintaining postural equilibrium. For example, pressure sensations from the soles of the feet provide information about the distribution of load between the two feet and whether the weight is more forward or backward on the feet.

Postural equilibrium is crucial for both static position and dynamic motion. Without it, the body is unable to function. Its importance is underscored by the number of systems the body uses to achieve equilibrium during static and dynamic activities. In addition to proprioceptors and somatosensory receptors, the body uses two additional input mechanisms to aide in equilibrium: the vestibular system and the visual system. The vestibular receptors in the inner ears provide awareness of head orientation and movements. Anyone who has had a middle ear infection can testify to the importance of the vestibular system in balance. Vision of where the body and its segments are relative to the surrounding environment also assist in maintaining equilibrium. In fact, visual input sometimes serves as the primary means of maintaining equilibrium when the proprioception system is impaired. The importance of equilibrium is observed not only during daily activities but also when performing various sports or when assessing impairments of equilibrium and suggesting solutions to balance problems. Even static equilibrium is affected by vision. Try standing on one leg with your eyes open and then with your eyes closed, and you will quickly realize how much you use vision for equilibrium.

Movement or "Motor" Control

When considering the control of movement, we must realize that movement and posture are exceedingly intricate and complex, and may be affected by an abundance of factors. For example, several systems must be intact for appropriate regulation of posture and movement. The neuromuscular systems must be intact, including the muscles that experience excitation or inhibition, muscles spindles, GTOs, neuromuscular junctions, peripheral nerves that innervate the muscles, spinal cord ascending and descending pathways, cortical motor centers, and the interconnections of these systems. The skeletal system, including the bones, ligaments, joints, joint capsules, and joint receptors also must be unimpaired. In addition, the respiratory, cardiovascular, and digestive systems must supply energy sources for muscular contractions and for the maintenance of the neuromusculoskeletal systems. Furthermore, accurate sensory input of the internal and external environments must be provided.

In order to perform skilled motor activities, a highly integrated set of motor commands is required to activate or inhibit several muscles in the proper way and in the proper sequence. We *cannot* view movement simply as the action of the various systems which carry out the movement task; rather, there is a highly complex organization and regulation in play that orchestrates our ability to move. **Motor control** refers to this dynamic regulation of posture and movement. Muscle **synergy** (Gr. *synergia*, together) is a term used to describe functional coordinated muscle activation, such as seen during functional movement when muscles typically work together as a group.[49]

Motor control requires the individual to maintain and change posture, and his or her movement response is based on an interaction between the individual, task, and environment. This interaction utilizes the contributions of many systems to orchestrate coordinated movement. These systems are not arranged in a **hierarchy** (Gr. *hierarchia*, rule or power of the high priest), in which one is more important than the other. Rather, they are a functioning **heterarchy** (Gr. *heteros*, other and *archos*, rule), in which the contributing systems work parallel to each other.[1]

Heterarchy recognizes that different levels of motor control exist and that portions of the nervous system interact with each other. In the heterarchy of motor control, cortical centers interact not only with each other but also with brainstem and spinal regions of the central nervous system, with the peripheral nervous system, and with ascending and descending pathways.[50, 51] In this heterarchy, information regarding the environmental milieu both inside and outside the body is provided to the central nervous system, specifically to the cerebral cortex, basal ganglia, and cerebellum, which plan, initiate, execute, coordinate, and regulate movement and posture. These centers also coordinate the timing of specific movements, whether simple or complex, the sequencing and synchronization of movements, as well as the amount of force generated. Which region is considered the "controller" varies, depending on the motor task desired and on the information provided to the central nervous system at a given time. Therefore, no one area is responsible for the control of all movement and posture.[52] The brainstem and spinal cord generate patterns of movement that are referred to as pattern generators, sometimes further clarified as central or stepping pattern generators.[5, 39, 53] Other

systems involved in motor control include ascending and descending pathways that provide feedback and feed-forward information.

Interactions within and between the various neural regions provide the most effective and efficient regulation of posture and movement. This model of heterarchy, therefore, considers both the importance of interactions of multiple areas within the CNS and the effect of an individual's ability to anticipate movements and adapt to changes in the environment. Heterarchy suggests that the flow of information is in more than one direction: The interaction occurs within and between levels of the nervous system, the interaction is reciprocal, and the information may be modified as a result of feedback and feed-forward systems.

Movement, therefore, is made possible by the contributions of many systems. Additionally, no system acts in isolation from the others in order to produce movement. In other words, movement emerges through the interaction and self-organization of many subsystems, and a movement behavior is greater than the sum of its individual parts.[54] Absent or disordered movement may result from a problem in the nervous system (either motor or sensory) or skeletal system or due to a difficulty encountered by the moving individual within the environment. In the rehabilitation setting, the patient's movement that a clinician observes is the end result of all of the possibilities and **constraints** (limitations or restrictions) offered by all of the contributing systems. In a simple example, if the gastrocnemius muscle is tight, the patient may stand with hyperextended knees.

Normal movements, therefore, are coordinated not because of muscle activation patterns prescribed only by sensory and motor pathways, but because the strategies of motion emerge from the interaction of the systems working together in this functional heterarchy. It is important to note that included in this complex of interactive systems are subsystems related to the environment and to the task itself. All of these elements are crucially important to movement execution. Our movements are ideally selected, executed, and modified as the best movement choice for a specific task within the environment in which we are moving. Multiple subsystems interact to produce a given motor behavior within a context appropriate for the environment and the task.[55] The interactions between these subsystems within the individual, the requirements of the task, and the unique aspects of the environment all affect the movement outcome.

Motor behaviors are conceived of as functional synergies (groups of muscles working together) rather than specific muscles or individual muscle groups. The human movement system is viewed as tremendously flexible, extremely dynamic, and capable of adapting to changes within the individual, the task, and/or the environment in an effort to produce the most effective movement possible.[1,56]

Dynamic Systems Approach to Understanding Motor Control

It is important to realize that these contributing systems change over time since they are dynamic in nature. A **dynamic action system** is any system that demonstrates change over time.[57] This **dynamical action system model** views movement not as the unfolding of predetermined or prescribed patterns in the CNS, but as emerging from the dynamic cooperation of many subsystems in a task-specific context.[54] The many systems then self-organize to produce movement.[58] Motor behavior emerges from the dynamic cooperation of all subsystems within the context of a specific task including the central nervous system as well as biomechanical, psychological, and social-emotional components.

Movement can then be expressed from among a wide variety of movement combinations, represented by a sum of all the possible degrees of freedom of the total joints involved in a movement. As you remember from Chapter 1, degrees of freedom represent the potential movements possible at specific joints. It is through a wide variety of movement combinations that human movement can be so varied. For example, the shoulder can move within three planes and therefore has three degrees of freedom, the elbow and the forearm each have one, and the wrist has two degrees of freedom. These comprise seven degrees of freedom for the upper extremity, excluding the fingers and thumb. If one was to add all the degrees of freedom available and all of the different directions of muscle pull across those joints, the total number of possible movement combinations is extremely numerous.[59] Every joint contributes its number of degrees of freedom to a movement, so that any functional movement emerges as the sum of all of the possible combinations that can occur. As mentioned, these functional movement synergies are self-assembled according to the interaction between the individual, the task, and the environment.

Motor Control at the Spinal Region

Neural connections within the spinal cord contribute much to the automatic control of movement. Specifically, the spinal region is the site for reflex motions, muscle synergy activations, and central pattern generators. Reflex motion control includes the stretch reflex, reciprocal inhibition, and autogenic inhibition, all

described earlier in this chapter. Transmission in local spinal circuits involves very little delay and ensures rapid responses. Interneurons within the spinal cord also link motor neurons into functional groups, or muscle synergies.

Reflexes

Spinal reflexes provide movement that is largely generated as a response to information arising from cutaneous, muscle, and joint receptors. These movements are stereotypical and predictable in nature but can be modified by the central nervous system. For example, arousal or alertness will change a person's response to the stretch reflex.

Pattern Generators

Complex muscle activation patterns that produce purposeful movement through neural connections at a spinal cord level are called **pattern generators**. These flexible networks of interneurons produce stepping and walking patterns that can be modified by cortical commands. Adaptable networks of interneurons in the spinal cord activate the lower motor neurons to elicit an alternating activation of flexor and extensor muscles at the hips, knees, and ankles.[60-63] This mechanism allows for efficiency of movement. These patterns are sensitive to changes in the task and the environment and will adapt body responses to these changes. For example, weight gain concomitant with growth in human infants will change the individual's stepping pattern.[49,64]

Motor Control within the Brainstem

As you recall from anatomy, the brainstem consists of the midbrain, pons, and medulla oblongata (Fig. 3.17). The brainstem contains numerous ascending and descending tracts as well as nuclei. These brainstem elements function as an extremely complex prespinal integrating system. These brainstem efferent tracts are largely responsible for automatic postural control. Most supraspinal postural control and proximal movement are regulated from brainstem centers.[49] Since these neurons are within the central nervous system, they are upper motor neurons. Bundles of axons that originate from neurons within the reticular formation of the brainstem and terminate within the spinal cord form the *reticulospinal tract*. Likewise, bundles of axons originating within the vestibular nuclei and terminating within the spinal cord comprise the *vestibulospinal tract* (Fig. 3.16). The reticulospinal tract provides excitatory input to extensor muscles of the arms and flexor muscles of the legs and trunk. In contrast, the vestibulospinal tract carries excitatory input destined for flexor muscles of the arms and extensor muscles of the legs and trunk.[5] Movement requires sufficient postural support from the limbs and body as a whole. Movement is the end product of a number of control systems that interact extensively together. The postural muscles are chiefly responsible for this control as they respond to efferent input from the reticulospinal tract.

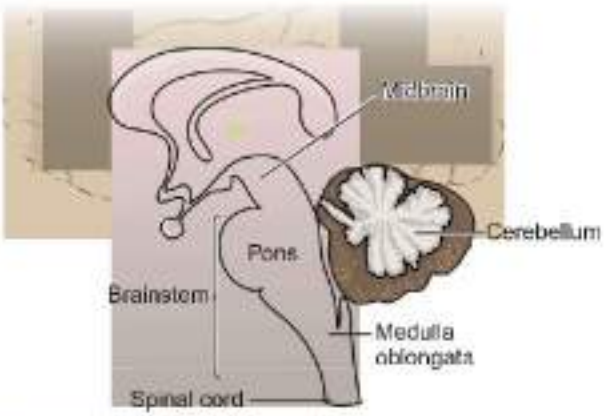

Figure 3.17 Sagittal view illustrating the positions of the main subdivisions of the brainstem.

Cerebral Motor Centers

When considering the motor functions of the nervous system, keep in mind that the motor centers can function appropriately only if an uninterrupted stream of afferent or sensory information about the status of the environment is received from all parts of the body. To emphasize the role of the sense organs in the control of posture and movement, the term *sensorimotor system* is sometimes used to denote the combined afferent and efferent processes required to produce coordinated movement.

Nerve fibers that descend from the motor cortex collectively form the *corticospinal tract* (Figs. 3.2 and 3.15). As the name implies, most of the axons arise from cell bodies in the motor area of the cerebral cortex and route downward in the spinal cord, where synaptic contact is made with motor neurons in the anterior horn gray matter of the spinal cord. The corticospinal tract also is referred to as the **pyramidal tract** because many of the cell bodies located in the motor cortex have a triangular shape and appear as small pyramids when a section of cortex is stained and viewed under a light microscope. Most of the corticospinal axons cross to the opposite side in the brainstem and descend in the lateral corticospinal tract of the spinal cord (Figs. 3.2, 3.15, 3.18). The crossing fibers from the right and left motor cortex also form a pyramid in the brainstem. At the spinal segmental level, axons of the corticospinal tract terminate

predominantly on interneurons. The interneurons terminate on α motor neurons. The organization of the corticospinal tract suggests that its design provides precise control of individual muscle groups. Other cortical neurons originating in the same areas of the motor cortex have shorter axons that synapse with second order motor neurons lying in the basal ganglia or brainstem (Fig. 3.18).

Motor Cortex

The frontal lobe is responsible for voluntary control of complex motor activities and cognitive functions, such as judgment, attention, mood, abstract thinking, and aggression. The frontal lobe, frequently referred to as the motor cortex, is further subdivided into the primary motor cortex, the premotor cortex, and the supplementary motor area. All three of these areas have their own somatotopic maps of the body, so that if different cortex areas are stimulated, different muscles and body parts move. However, all three subdivisions offer their own unique yet collaborative contribution to the cerebral control of movement, highlighting once again the beauty of the heterarchical organization of motor control.

The primary motor cortex is responsible for **contralateral** (opposite side of the body) voluntary control of the upper extremity and facial movements. The premotor cortex controls the muscles of the trunk and muscles used in anticipatory postural adjustments, such as required in establishing the correct "postural set" in preparation for standing up from a chair.[1] The supplemental motor cortex controls the initiation of movement, orientation of the head and eyes, and bilateral (involving both body sides) movements.[65,66] The supplemental area also controls the sequencing of movement and plays a role in the pre-programming of movement sequences that are familiar and part of an individual's memory repertoire.[49]

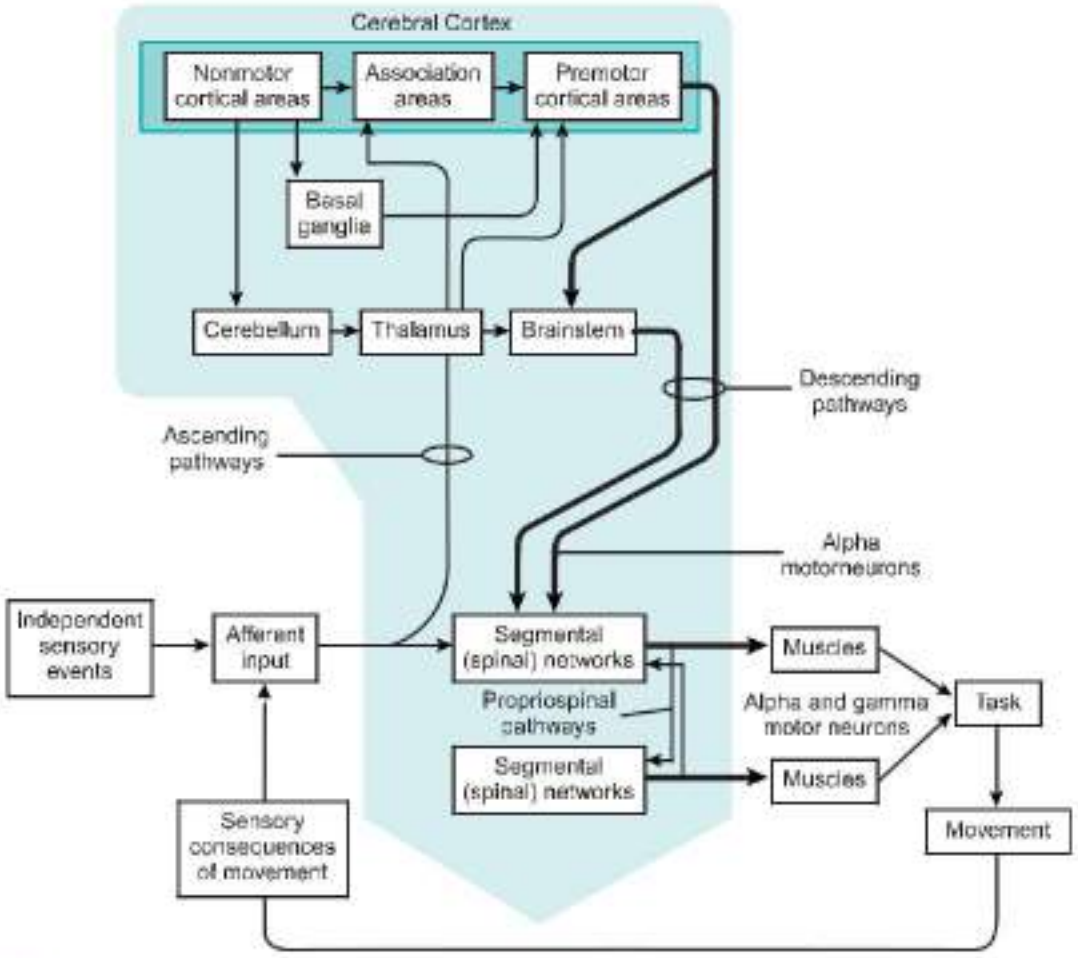

Figure 3.18 Schematic diagram of the course of the lateral and medial corticospinal tracts from the motor cortex to the spinal cord. For simplicity, collaterals to the basal ganglia, the cerebellum, and the motor centers of the brainstem have been omitted. Note that the structures and tracts exist on both the left and right sides; however, only one side is illustrated. For further description, see text.

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Lesions of the primary motor cortex (as seen following a stroke) usually give rise to contralateral weakness or paresis, typically seen as a period of initial **flaccidity**, in which muscle tone is absent. Clinically, this type of damage will produce reduced postural reactions and diminished stretch reflexes in the acute phase. Oftentimes, this event is followed by a slow recovery and eventually overactive neural responses, producing hyperactive stretch reflexes. Recovery is typically gradual but incomplete.

Lesions of the supplemental motor cortex result in complex motor dysfunction, including severe **akinesia** (lack of movement) and difficulty with tasks requiring the cooperative use of both hands. Patients also have difficulty performing self-initiated tasks but are able to benefit from therapeutic approaches that teach them to use additional sensory cues to start a movement.

Lesions of the premotor cortex result in nonspecific motor disturbances or **apraxia**, in which the patient's movements are slow and clumsy with mild proximal weakness and loss of coordination around the proximal joints. Rhythmic movements such as typing or tapping are disrupted, and **perseveration** (stammering or repeating) may occur. Unlike patients with lesions of the supplemental motor cortex, these patients are able to perform self-initiated tasks, but have difficulty with sensory-triggered tasks. Previously acquired sequential tasks deteriorate, even though individual components of the sequenced task can be performed.[1, 97]

Cerebellar lesions cause distinctive motor symptoms. Cerebellar symptomology usually includes balance and coordination deficits. These deficits may produce **ataxia** (wide based movements), intention tremor (tremor accompanying purposeful movement), and **dysmetria** (inability to gauge distance or properly scale the required force in reaching or stepping).[98] Cerebellar damage can cause any number of errors in the kinematic parameters of movement control, including difficulties with timing, accuracy, coordination, and regulation of intensity.[1]

The cerebellum and the basal ganglia serve different but related functions in programming cortically initiated movement patterns, and both act as important motor control centers, contributing vital regulatory functions in movement control.

Cerebellum

The cerebellum is interconnected with all levels of the central nervous system and functions as an overall "coordinator" of motor activities. The cerebellum is responsible primarily for programming rapid movements, correcting the course of rapid movements, and correlating posture and movement. The cerebellum regulates balance and coordination. It is responsible for regulating and adjusting the accuracy, intensity, and timing of movement as required by the specific movement task. It sequences the order of muscle firing when a group of muscles work together to perform a complex task such as stepping or reaching.[56] The cerebellar pathways control balance, coordination, and movement accuracy on the **ipsilateral** (same) body side, as opposed to the **contralateral** control feature associated with the cerebral cortex. The cerebellum is often called the "great comparator," because it constantly monitors and compares the movement requested to the actual output, making adjustments as necessary.[1] The cerebellum has the ability to receive sensory feedback from receptors about a movement as the movement is occurring, a property called **reafference**.

Basal Ganglia

At the base of the cerebrum (hence the name "basal") are several nuclei, including the caudate, putamen, globus pallidus, substantia nigra, and subthalamic nuclei. Although all of their functions are unknown, the basal ganglia play a vital role in the regulation of posture and muscle tone. The basal ganglia have no input from the spinal cord, but they do have direct input from the cortex. They have an important role in the control of both automatic and voluntary movement, exerting effects on the motor planning areas of the motor cortex.

The basal ganglia can either inhibit or facilitate cortex output to alter conscious motion. Nuclei of the basal ganglia are particularly significant with respect to the initiation and execution of slow movements (Fig. 3.18).

Integration of Motor Control to Produce Functional Movement

The entire chapter thus far has presented physiological and anatomical information on the integration of the nervous system with the musculoskeleral system to provide motion. Hopefully, you now realize that movement is the result of complex interactions of many systems and subsystems. For example, sensory impulses from muscles are not restricted to influencing only their own motor neurons. Afferent input also spreads through collateral branches of primary sensory neurons and through interneuron circuits to reach the motor neurons of closely related muscles and, to some extent, those of more "distant" muscles. Therefore, stretch or contraction of one muscle affects its own motor neurons most strongly and, to a lesser extent, also affects motor neurons of muscles performing an opposite action. The effect on the muscles that perform the opposite action is to inhibit activity of those muscles (Fig. 3.14). Motor neurons of other muscles that assist in the motion are also affected but to an even lesser extent. The effect on muscles that assist in the movement is to facilitate action. Thus, every primary loop or pathway is part of a larger feedback network serving a group of muscles.

While an immediate response is occurring, the same sensory signals are being transmitted to higher centers by way of collaterals, projection tracts, and secondary relays to widely separated parts of the nervous system for more elaborate analyses of the information. The ascending tracts allow information integration regarding the status of the body and the status of the environment. Many types of receptors in tendons, joints, and skin, as well as from visual, auditory, and vestibular receptors, simultaneously also provide sensory information regarding the body and the environment. Subsequently, motor signals are relayed back to numerous segmental levels to adjust posture and perform other actions.

It is important to recognize that not only is the precise functioning of the musculoskeletal and nervous systems essential for motor control, but cognition also plays an important role. Cognitive factors are necessary to assimilate sensory information, process and integrate the information, and determine appropriate movements and postures at any given instant. Furthermore, memory of movement and the ability to recall movement information are integral components to the regulation of posture and movement. The execution and efficiency of movement are further influenced by factors such as the ability to concentrate; the presence or absence of visual, auditory, mental or emotional distractions; one's level of proficiency; and one's motivation. Likewise, cognitive strategies influence motor control.[70-74]

The more often a motor pattern is learned, the less input from the cortical level is required until the motor pattern becomes subcortical, not requiring conscious thought to produce accuracy. We see this result when we walk. As toddlers, we used our cortex a great deal to provide us with feedback to balance our body weight over our feet, put one foot in front of the other, change the position of our center of mass, and move forward. As we perfected our gait, we were able to walk without conscious correction, relying on our subcortical nervous innervation and corrections to move safely and securely in our environment. **Motor learning** is concerned with how motor skills are acquired and how they are made proficient, transferred, and retained to allow consistent, accurate, and automatic motion. The reader is referred to other sources a complete discussion of motor learning.[75,76]

Health care professionals work to enhance motor control and motor performance in patients as well as healthy individuals engaged in competitive or leisure sports activities and wellness programs. Intervention programs are designed to stretch and strengthen muscles, to increase endurance, to improve balance and

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The most common clinical condition that results from pathology in the basal ganglia is Parkinson's disease in which patients exhibit a resting tremor, difficulty initiating movement (**akinesia**), slowness of movement (**bradykinesia**), muscular rigidity, and a stooped posture. This progressive disease, with a mean onset age of 56, is caused by a gradual loss of neurons that produce dopamine (a neurotransmitter) in the basal ganglia.[69]

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If we take an example of a motor skill, all of these factors may be more easily appreciated. Kicking a ball towards a target is a complex motor skill. Many cognitive components are first assimilated. The weight of the ball, the distance of the target, the wind speed and direction are all factors that the individual consciously considers before kicking a ball. In the early days of this skill acquisition, the individual also had to rely on his or her coordination and balance input to be able to stand securely on one foot while moving the contralateral leg from hip extension to hip flexion. Additionally, hip abduction to adduction and medial to lateral rotation motions were likely included in the performance. Other joint motions the nervous system had to control included the knee and the ankle. Cocontraction of trunk muscles was necessary for posture control during the kick. Sensory input regarding muscle length, tension, and changes in length as the individual approached the ball were continually being fed, received, and responded to by the neuromuscular system. Once the ball was kicked, the neural system obtained the feedback regarding the performance: Was the ball kicked far enough? Where did the ball land in relation to the target? What part of the foot came in contact with the ball? Where in relation to the body was the foot and leg when contact with the ball was made? The nervous system collects all this data, the subsystems self-organize and alter the next attempt based on this input.

muscle tone, and to improve the ability to regulate posture and movement. These professionals also realize the importance of practice to enhance skill and recognize that more than just the neuromusculoskeletal system must be enhanced to augment performance. It is important to acknowledge that cognitive strategies such as mental practice and imagery, as well as positive self-talk, are important for the client's success.[75-77] Principles of cognition used to enhance motor performance may be applied clinically so that an individual's mindset for rehabilitation is productive to facilitate recovery.

Functional Applications and Clinical Considerations

Neuromuscular impairments encompass a diverse group of problems that constitute a major constraint on functional movement. Impairments of motor control may result from many diseases, injuries, or developmental disabilities and can result from pathology to any part of the movement system. Pathological conditions that affect any part of the neuromuscular system, including motor, sensory, perceptual, and cognitive elements, will result in associated signs, symptoms, and impairments.[78] Other factors may be involved in motor control dysfunction, including the skeletal, cognitive, visual or vestibular systems. Since this chapter deals with movement control and primarily the neuromuscular aspects involved in the control of movement, we will continue to focus on impairments that affect these structures and contribute to alterations in movement.

Impairments are the typical consequences of the disease or pathological process, further defined as the loss or abnormality of function, at the tissue, organ, or system level, resulting in constrained movement. Examples of primary motor impairments include weakness, abnormalities in muscle tone, and motor coordination problems.[1, 68] In addition to primary impairments, secondary impairments also contribute to movement problems. These secondary impairments do not result from the pathology directly, but rather develop as a result of the consequences of the primary impairment and may be preventable. Examples of secondary impairments include loss of range of motion or contracture.[78]

The ability to produce and coordinate an appropriate movement response requires production of muscular force, activation and sustenance of muscle activity, and the coordination and timing of muscle activation patterns. The primary motor system impairments that interfere with functional movement are muscle weakness, abnormalities of muscle tone, and coordination problems.[68]

Muscle Weakness

Muscle weakness is defined as an inability to generate normal levels of muscular force and is a major impairment of motor function in patients with nervous and/or muscular system damage.[68] Lesions within the CNS, PNS, or muscular system can produce weakness. It is important to differentiate the weakness from where in the movement system the damage is located. By definition, damage to the descending motor control systems in the CNS is associated with lesions affecting upper motor neurons, anywhere from the spinal cord superiorly.[68] This damage will produce signs of upper motor

neuron (UMN) damage. Upper motor neuron lesions are associated with hypertonicity, or hypotonicity, depending on the site of the lesion and the time of onset (acute vs. chronic). Depending on the extent of the lesion, weakness in the patient with an upper motor neuron lesion can vary in severity from total loss of muscle activity (paralysis or plegia) to a mild or partial loss of muscle activity (paresis).[68] Paresis results from damage to the descending motor pathways, which interferes with the brain's excitatory drive to the motor units, thereby resulting in a loss of descending control of the lower motor neurons.[79] The end result is an inability to recruit and modulate the motor neurons, leading to a loss of movement.

Upper motor neuron lesions are accompanied by secondary abnormal muscle tone and altered motor control.[68, 80] The range of muscle tone abnormalities found within patients who have UMN covers a broad spectrum, ranging from complete flaccidity (loss of tone), to spasticity (hypertonicity).[68] Changes in muscle tone will vary depending on the specific lesion. Following an upper motor neuron lesion, weakness occurs due to loss of motor unit recruitment, changes in recruitment patterns, and changes in firing rates. Additionally, changes occur in the properties of the motor units and in the morphological and mechanical properties of the muscle itself. These secondary changes happen as adaptations to loss of innervation, immobility, and disuse. In upper motor neuron lesions, reduced numbers of motor units and reduced firing rates of motor units have been reported.[14, 81] Within two months of the insult, patients with hemiparesis resulting from a stroke show up to a 50% reduction in motor units on the affected side. Individuals who have had a stroke display atrophy in motor units on the hemiparetic side. The remaining motor units require more time to contract, and they fatigue more rapidly. Altered recruitment and decreased motor unit firing accounts for this apparent weakness.[82] The degree of weakness may differ for different muscle groups. Given that the pyramidal tract is the primary pathway for voluntary goal-directed movement, it has been suggested that interruption of this pathway produces a greater impairment in prime mover muscles.[83] Prolonged paresis, a primary neuromuscular impairment, also produces secondary musculoskeletal impairments. Changes in muscle tissue resulting from damage to upper motor neurons suggest that muscle may not be as "strong" due to changes in the properties of the muscle and the presence of denervated muscle fibers.[82] Specific changes at the motor neuron secondary to the upper motor neuron damage can decrease a patient's ability to produce force.

Muscle weakness most often results from direct injury to the muscle. A wide continuum of injuries, from contusions to ruptures, produce weakness, initially from the injury itself, and secondarily from inactivity and disuse following the injury during the recovery phase. Pain, whether in an injured muscle or in a joint on which the muscle acts, reduces the individual's willingness to move the muscle. When a muscle is not used at its normal functional level, weakness ensues.

Regardless of the underlying etiology or pathology, when a muscle is not used or exercised, muscle weakness and atrophy occur. When a muscle does not function for long periods of time, the quantity of actin and myosin myofilaments in the muscle's fibers actually decrease. This change is reflected in reduced diameters of individual fibers, and diminished overall muscle cross-sectional area.[91] Muscle wasting is due, at least in part, to a decrease in protein synthesis coupled with increased protein degradation; these changes cause alterations in contractile properties and a resultant loss in the muscle's ability to develop and hold tension.[31, 92] In response to decreased use, skeletal muscle also undergoes an adaptive remodeling; this process includes a transition from slow to fast myosin fiber types, a fuel shift toward glycolysis, decreased capacity for fat oxidation, and energy substrate

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Clinicians formerly believed that prescribing strength training was not appropriate for patients with UMN pathology. Research has demonstrated that improvements in strength not only contribute to an improvement in functional performance, but that there is also no indication of any associated increase in spasticity.[79-83] Strength training is thought to not only improve voluntary motor control, but it also appears to prevent or slow down some of the mechanical changes and denervation changes seen in muscle tissue following UMN damage.[67, 10] The shift in emphasis to the functional significance of weakness in patients with CNS lesions has led to increased attention on strengthening programs for both adults and children with CNS disorders.

accumulation in the atrophied muscle.[93] A loss of peak muscle force and functional muscle strength results from these changes.[51] **Disuse atrophy** is a term used to specifically describe this atrophy that occurs when a person or limb is immobile, such as during bed rest, or when a limb is restricted in a sling, brace, or cast.[94, 95] Current research demonstrates that this disuse atrophy begins within 4 hours of the start of bed rest![97]

Abnormal Muscle Tone

As described briefly earlier, typically muscle tone is characterized by a state of readiness of muscle to perform the task demands to be placed on it. The level of excitability of the pool of motor neurons controlling a muscle, the intrinsic muscle stiffness, the absence of neuropathology, and the level of reflex sensitivity determine this state of readiness. A hallmark of central nervous system pathology is the presence of abnormal muscle tone. Abnormally high (hypertonia) or abnormally low (hypotonia) muscle tone are universally recognized clinical signs of nervous system pathology. Flaccidity and hypotonia are states of muscle hypotonicity, while spasticity and rigidity are states of hypertonicity. Typically, upper motor neuron lesions oftentimes results in hypertonia and lower motor neuron lesions in hypotonia. Terms related to abnormal muscle tone are found in Table 3–4.

Coordination Problems

Coordinated movement involves multiple joints and muscles that are activated at the appropriate time and with the correct amount of force so that smooth, efficient, and accurate movement occurs.[68] The essence of coordinated movement, therefore, is the synergistic organization of multiple muscles for purposeful motion, not just the capacity to fire an isolated muscle contraction. Incoordination can result from pathology in a wide variety of neural structures, including the motor cortex, basal ganglia, and cerebellum. Uncoordinated movement may be displayed through the manifestation of abnormal synergies, inappropriate coactivation patterns, and timing problems.

As mentioned earlier, **synergy** is a group of muscles that often act together as if in a bound unit. Nicolai Bernstein[100] used the term synergy to aptly describe the functional muscle groups that produce motor behavior. Lesions to corticospinal centers can also lead to the ability to recruit only a limited number of muscles controlling a movement. The result is the emergence of mass patterns of movement, referred to as abnormal synergies. Abnormal synergies reflect an inability to move a single joint without simultaneously generating movement in other joints. Abnormal synergies are stereotypical patterns of movement that don't change or adapt to environmental or task demands.[1,68]

Coordination problems can also be manifested as abnormalities with muscle activation patterns and difficulties with muscle sequencing. Inappropriate coactivation of muscles is an example of a sequencing problem. Coactivation, which means that the agonist and antagonist both fire, is normally present in the early stages of learning a skilled movement. Coactivation is commonplace in young children just learning to balance and during early walking patterns. Adults also frequently demonstrate coactivation when attempting to learn a new task. In the neurologically intact adult, coactivation is atypical unless during the early stages of learning a new skill. Coactivation requires unnecessary energy expenditure and results in inefficient movement. Inappropriate coactivation occurs in central nervous system disorders in both children and adults. This inappropriate and ungraded coactivation of agonist and antagonist contributes to functional limitations in force generation. Coactivation has been demonstrated in adults following a stroke and in children with cerebral palsy during walking and the performance of common functional skills.[101,102]

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It is important to recognize how quickly skeletal muscle atrophy occurs in response to disuse. Disuse atrophy can be delayed and decreased in severity by intermittently contracting the muscle isometrically during any period of immobilization or relative inactivity.[96] Exercise and proper nutrition have both resulted in protein synthesis stimulation in muscle and tendon with obvious implications for rehabilitation management.[99] On the other hand, clinicians are cautioned that if atrophy has already occurred, strenuous exercise of atrophied muscle can lead to muscle damage, including sarcolemma disruption and distortion of the myofibrils' contractile components.[97] Prevention with early intervention is truly the best practice.

Uncoordinated movement can also be manifested as an inability to appropriately time the action of muscles, to activate muscles in the appropriate sequence, or to scale or grade the force needed. There can be many facets to timing errors including problems initiating the movement, slowed movement execution, and problems terminating a movement. All of these timing errors have been observed in individuals with neurological damage. Coordination problems, characterized by problems in muscle activation, sequencing, timing, and scaling, can create a tremendous obstacle to efficient functional movement.

Since coordination requires adequate strength and ROM, uncoordinated movement is often characterized by some degree of weakness, fatigue, or instability. Likewise, body segments weakened by injury or disuse may suffer inadequate coordination and sequencing. Even as a patient fatigues during rehabilitation exercises, coordination becomes more difficult. As previously mentioned, muscle recruitment occurs in normal muscles from single joint muscles to multiple joint muscles. Proper muscle sequencing is also important in daily activities, but if correct sequencing is not present, the individual is at risk of injury at the most and inefficient movement at the least. Such consequences place additional stresses on other body segments. For example, studies have demonstrated that muscle recruitment and sequencing vary between back patients and normal groups.[103, 104] It is unclear, however, if the changes in recruitment were the cause of pain or the result of pain. In either situation, an individual is not able to function optimally if proper muscle activation and recruitment sequencing is dysfunctional.

Involuntary Movements

Involuntary movements are a common motor sign of neurological damage and can take many forms. **Dystonia** is a syndrome dominated by sustained muscle contractions, frequently causing abnormal postures, twisting or writhing movements, and repetitive abnormal postures. Dystonic movements usually result from basal ganglia disturbances.[1]

Tremor is defined as a rhythmic, involuntary, oscillatory movement of a body part.[106] A tremor results from damage to the CNS. A **resting tremor** is a tremor occurring in a body part that is not voluntarily activated and is supported against gravity. Resting tremors is a symptom of Parkinson's disease and is secondary to basal ganglia dysfunction. An **intention tremor** occurs when the individual attempts purposeful movement of an extremity. Intention tremors often accompany cerebellar lesions.

Common Pathological Conditions Affecting Movement System Function

The movement system can be impacted by numerous pathological conditions that affect any contributing

TABLE 3–4 | ABNORMAL MUSCLE TONE TERMINOLOGY

Term	Origin of Term	Definition	Clinical Examples
Flaccid	L. *flaccidus*, weak, soft, lax	complete loss of muscle tone	Flaccidity is often seen in the acute stage of injury, immediately following a CNS injury, but it can also be secondary to a lower motor neuron lesion. In patients with flaccidity, deep tendon reflexes (DTRs) are absent.
Hypotonia	Gr. *hypo*, under and *tonos*, tension	reduction in muscle stiffness	Characterized by low muscle tone, weak neck and trunk control, poor muscular co-contraction, and limited stability. Patients with hypotonia present with weakness, a decreased ability to sustain muscle activation, a decreased ability to coactivate muscle groups, abnormal joint mobility patterns, and a delayed or ineffective exhibition of normal postural responses.
Hypertonia	Gr. *hyper*, over, above and *tonus*, tension	excessive muscle tone	See spasticity

TABLE 3-4 | ABNORMAL MUSCLE TONE TERMINOLOGY—cont'd

Term	Origin of Term	Definition	Clinical Examples
Spasticity	Gr. *spastikos*, to tug or draw	motor disorder characterized by a velocity-dependent increase in the stretch reflex with exaggerated tendon jerks, resulting from hyperexcitability	Typically seen as part of the upper motor neuron clinical presentation. As a result, there will be increased alpha motor neuron excitability with a resultant increase in muscle tone and exaggerated stretch reflexes, secondary to this damage in the descending motor systems. Clinically, the term spasticity is used to describe a wide range of abnormal motor behaviors including: 1) Hyperactive stretch reflexes, 2) Abnormal posturing of the limbs, 3) Excessive coactivation of the antagonist muscles, 4) Associated movements, 5) Clonus, and 6) Stereotypical movement synergies
Rigidity	L. *Rigidus*, inflexible, rigid	heightened resistance to passive movement, but independent of the velocity of that stretch or movement.	Rigidity is associated with lesions of the basal ganglia, and appears to be the result of excessive supraspinal drive acting upon a normal spinal reflex mechanism. Rigidity tends to be predominant in the flexor muscles of the trunk and limbs and results in severe functional limitations. There are two types of rigidity, lead pipe and cogwheel. A constant resistance to movement throughout the range characterizes **lead pipe rigidity,** whereas **cogwheel rigidity** is characterized by alternate episodes of resistance and relaxation. Rigidity is frequently associated with lesions of the basal ganglia, commonly seen in Parkinson's disease

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Because the stretch reflex is velocity dependent, and due to increased muscular stiffness, spasticity limits a patient's ability to move quickly. Regardless of its complex neural basis, it is important to remember that spasticity is simply one of several symptoms of neurological damage and should be treated as it interferes with function. Functional treatment approaches should focus primarily on improving active muscle control in addition to spasticity symptom reduction when it limits movement.

component of the nervous, muscular, or skeletal systems, arising at any phase of the life span. A few commonly encountered pathological conditions with their associated impairments and functional limitations are briefly described in the following section. This section identifies only a few selected conditions to illustrate the functional implications associated with impaired movement.

Peripheral nerves (Figs. 3.2 and 3.3) may be damaged by disease or trauma. Acute injury includes lacerations or other causes of partial or complete severance of the nerve. Other acute or repetitive injuries may occur from pressure or compression of the peripheral nerve. If the damage is complete, flaccid paralysis of muscle fibers supplied by the damaged lower motor axons will result when the muscles no longer receive efferent signals.

A common peripheral nerve lesion in the upper extremity affects the median nerve. The median nerve is susceptible to damage at the wrist, where it may be compressed within the carpal tunnel. Remember from your study of anatomy that the tendons of the long finger flexors and the median nerve pass under the flexor retinaculum (L. *retinaculum*, a rope or cable). In instances of essential narrowing of the carpal tunnel through anatomical constraints, enlargement of soft tissue structures, or swelling of structures within the tunnel, compression of the median nerve within the carpal tunnel often results in carpal tunnel syndrome. Symptoms associated with the compression of the median nerve include decreased sensation in the area innervated by the nerve, pain and, if the condition progresses, atrophy with weakness of muscles innervated by the median nerve. Peripheral nerves in the more proximal upper extremity also suffer injury secondary to fractures. For example, a fracture of the humerus may cause a lesion of the radial nerve, resulting in weakness or total loss of function of the elbow and wrist extensors. In the lower extremities, the sciatic nerve is a frequent site of pathology.

Peripheral nerve injuries may result in muscular imbalance. Muscle imbalance occurs when one group of muscles is opposed by an impaired muscle group. This condition can then lead to secondary deformities. For example, following a lesion of the ulnar nerve, the individual is predisposed to developing a "claw hand" deformity. In this case, the long flexors and extensors of the fingers are not affected by the ulnar nerve deficiency. Their pull, however, is opposed by nonfunctioning intrinsic muscles in the hand so the balance between the long finger flexors and long finger extensors is lacking. Without occasional movement, adhesions can form between tendons and the sheaths that surround them, as well as between adjacent bundles of muscle fibers. When tissues crossing a joint remain in the same position for prolonged periods, a contracture forms, whereby the tissues adapt to the shortened position and exhibit a decrease from normal joint range of motion. These complications may be prevented by using passive physical activity to maintain full range of movement and increase flow of blood and lymph through the area. Splints also may assist in preventing contractures.

Cerebral Palsy

Cerebral palsy (L. *cerebrum*, brain; *palsy*, paralysis) is a general term used to describe a group of motor disorders that generally result from damage to the developing brain. As one of the most common developmental disabilities, cerebral palsy results from a lesion to the brain during prenatal (L. *prae*, before, plus L. *natal*, birth), perinatal, or early postnatal stages of life. The brain lesion causes a nonprogressive but permanent damage to one or more areas of the brain. Although cerebral palsy is defined as a neurologically static condition, it can be considered orthopedically progressive in nature. Depending on the sites of the neurologic lesion, an individual with cerebral palsy may show a variety of motor or other impairments. Because of the close relationship of motor functions with other neural functions and because of the potential diffuse nature of the lesion, the individual with cerebral palsy also may demonstrate sensory, communicative, perceptual, and/or cognitive impairments.

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Loss of sensation can be a more serious problem than loss of muscle strength for a person with a peripheral nerve lesion. Individuals with impaired sensory function may exhibit a loss of awareness of location or position of certain body segments, reduced pressure sensation, deficient temperature detection, and/or loss of pain sensation. If such sensory deficiencies exist, the person may not detect when blood flow is occluded by external pressure or when the part is in contact with excessively hot or cold objects. Various sensory losses place the affected body segment at risk for traumatic injuries, ischemia (Gr. *ischein*, to suppress, plus *haima*, blood), burns, pressure sores, and subsequent infections.

The central nervous system is very vulnerable to reduction of its blood supply. Cerebrovascular accidents (CVAs), or strokes (from the Greek term *streich*, meaning "to strike"), occur when the blood supply to an area in the CNS is disrupted. Residual problems following a stroke vary greatly depending on numerous factors such as the cause of the CVA, the affected CNS area, the extent of the damage, and the functions of the damaged area(s). The clinical deficits may include weakness or paralysis of the muscles of the face, trunk, and/or extremities; impairment of sensation and proprioception; visual deficits; cognitive difficulties; language impairments; and perceptual problems. Impairment of motor and sensory impulse conduction is likely to produce paralysis of muscles on the side contralateral (opposite) to the lesion, causing the clinical presentation referred to as hemiplegia.

Basal Ganglia Disorders

The basal ganglia are generally responsible for the regulation of posture and muscle tone. They convert plans for movement into programs for movement by affecting the motor planning areas of the motor cortex, particularly with respect to the initiation and execution of movements. The most common complex of symptoms resulting from disturbance of basal ganglia connections is Parkinson's disease. Individuals with Parkinson's disease demonstrate movement characterized by slowness of movement; rigidity of facial expressions; decreased or absent communicative gestures; a hesitant, shuffling gait with small steps; and resting tremor of the hands.

Athetosis is another movement disorder involving the basal ganglia. Athetosis, however, results in slow, writhing movements that are exhibited especially in the upper extremities. Basal ganglia disorders also include chorea, a complex disorder in which the individual has involuntary, sudden, nonpurposeful movements.

Cerebellar Disorders

The cerebellum regulates balance and coordination. It is responsible for regulating and adjusting the accuracy, intensity, and timing of movement as required by the specific movement task. It sequences the order of muscle firing when a group of muscles work together to perform a complex task such as ambulation or reaching.[56] The cerebellar pathways control balance, coordination, and movement accuracy on the ipsilateral body side, as opposed to the contralateral-control feature associated with the cerebral cortex. Cerebellar lesions cause distinctive motor symptoms. Cerebellar damage can cause any number of errors in the kinematic parameters of movement control, including difficulties with timing, accuracy, coordination, and regulation of intensity.

Summary

This chapter gave an overview of the human movement system and its main structural components. The anatomy and physiology of muscle tissue was reviewed and an organizational framework for studying the human nervous system was described. Motor control, as a dynamic and heterarchical system controlling functional human movement, was discussed. Movement impairments and their functional consequences were defined and described. Common primary impairments that affect human movement were described. For the purposes of illustration, a few commonly encountered pathological conditions that cause disordered movement were introduced with a focus on the functional consequences to movement.

CLINICAL SCENARIO SOLUTION

Joseph has cerebral palsy, and so the weakness that he demonstrates in his lower extremities is caused by a lack of movement control secondary to the developmental nature of that disability. Spasticity is a symptom of upper motor neuron brain damage, secondary to the pathological condition which caused his cerebral palsy. The ulnar nerve injury sustained in Joseph's left upper extremity will result in motor and sensory loss of function to the muscles supplied by the ulnar nerve below the injury, functionally resulting in lost innervation to many of the muscles required for a full grasp. Because the ulnar nerve injury is a lower motor neuron lesion, it will regenerate and function will return over a period of a few months. The transient nature of the ulnar nerve injury is in contrast to the more permanent weakness and overlying spasticity seen in his lower extremities due to the cerebral palsy.

Discussion Questions

1. Think about muscle fiber types in discussing the following: Why is the breast meat of a domestic chicken white as opposed to the breast meat of a pheasant or duck? Along the same lines, why is the leg meat of domestic fowl (chicken and turkeys) dark?
2. What do you think would be the major fiber composition in each of the following muscles, based on their primary function as primarily tonic or phasic: 1) back extensors; 2) biceps brachii; 3) soleus; and 4) finger flexors?
3. What is the overall organization of the nervous system, both anatomically and physiologically?
4. How does the nervous system send signals, utilizing the following physiological processes in order to transduce and communicate a signal: action potential, receptor potential, threshold, excitatory and inhibitory postsynaptic potential, summation?
5. What is motor control? What is the dynamic nature of the systems involved in motor control?

Lab Activities

1. **Stretch Reflex (Materials needed—reflex hammer):**

   Working in pairs, one partner is seated at the edge of a plinth so that the foot is off the ground, hip and knee flexed comfortably. This subject should be sitting and relaxed, with his or her eyes closed. The other partner uses the reflex hammer to elicit the stretch reflex in the quadriceps muscle. Explain Figure 3.12. The most important part of this lesson is for one partner to be able to explain to the other the mechanism involved, including the monosynaptic stretch reflex, reciprocal innervation, and a beginning understanding of the basis for normal muscle tone. Note the length of time between the tap and the response. Observe the "crispness" of the response. Observe variations among different subjects.

2. **Proprioception:**

   One partner is instructed to close his or her eyes. The other partner passively moves the partner's arm into a new position and asks the person to hold it there momentarily. Once the partner has moved the extremity back to a position of rest, the partner whose eyes were closed is instructed to duplicate the arm position that the arm had been placed in by the other partner. Discussion can then follow about the value of proprioception and intact kinesthesia.

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