TABLE 1.5 Physical Agents for the Treatment of Tone Abnormalities

Tone Abnormality	Goals of Treatment	Effective Agents	Contraindicated Agents
Hypertonicity	Decrease tone	Neutral warmth, prolonged cryotherapy, or EMG biofeedback to hypertonic muscles Motor ES or quick ice of antagonists	Quick ice of agonists
Hypotonicity	Increase tone	Quick ice, motor ES, or EMG biofeedback to agonists	Thermotherapy
Fluctuating tone	Normalize tone	Functional ES	

EMG, Electromyographic; *ES,* electrical stimulation.

recommended because of concern that this would further increase muscle tone; however, reports indicate that ES of hypertonic muscles improves patient function, likely by increasing strength and voluntary control of these muscles.[48,49]

In patients with muscle hypotonicity, in which the goal of intervention is to increase tone, quick icing or motor-level ES of hypotonic muscles may be beneficial. In contrast, applying heat to these muscles should usually be avoided because this may further reduce muscle tone. In patients with fluctuating tone, for whom the goal of treatment is to normalize tone, functional ES may be applied to cause a muscle or muscles to contract at the appropriate time during functional activities. For example, if a patient cannot maintain a functional grasp because they cannot contract the wrist extensors while contracting the finger flexors, ES can induce the wrist extensors to contract at the appropriate time during active grasping.

General Contraindications and Precautions for Physical Agent Use

Restrictions on the use of particular treatment interventions are categorized as contraindications or precautions. Contraindications are conditions under which a particular treatment should not be applied, and precautions are conditions under which a particular form of treatment should be applied with special care or limitations. The terms *absolute contraindications* and *relative contraindications* can be used in place of contraindications and precautions, respectively.

Although contraindications and precautions for the application of specific physical agents vary, several conditions are contraindications or precautions for the use of most physical agents. Therefore caution should be used when applying a physical agent to a patient having any of these conditions. In patients with such conditions, the nature of the restriction, the nature and distribution of the physiological effects of the physical agent, and the distribution of energy produced by the physical agent must be considered.

✶ CONTRAINDICATIONS for Application of a Physical Agent

- Pregnancy
- Malignancy
- Pacemaker or other implanted electronic device
- Impaired sensation
- Impaired mentation

PREGNANCY

Pregnancy is generally a contraindication or precaution for the application of a physical agent if the energy produced by that agent or its physiological effects may reach the fetus. These restrictions apply because the influences of these types of energy on fetal development usually are unknown and because fetal development is adversely affected by many influences, some of which are subtle.

MALIGNANCY

Malignancy is a contraindication or precaution for the application of physical agents if the energy produced by the agent or its physiological effects may reach malignant tissue or alter the circulation to such tissue. Some physical agents are known to accelerate the growth, or metastasis, of malignant tissue. These effects are thought to result from increased circulation or altered cellular function. Care must be taken when considering treatment on any area of the body that currently has or previously had cancer cells because malignant tissue can metastasize and therefore may be present in areas where it has not yet been detected.

PACEMAKER OR OTHER IMPLANTED ELECTRONIC DEVICE

The use of a physical agent is generally contraindicated when the energy of the agent can reach a pacemaker or any other implanted electronic device (e.g., deep brain stimulator, spinal cord stimulator, implanted cardioverter defibrillator) because the energy produced by some of these agents may alter the functioning of the device.

IMPAIRED SENSATION AND MENTATION

Impaired sensation and mentation are contraindications or precautions for the use of many physical agents because the limit for application of these agents is the patient's report of how they feel. For example, for most thermal agents, the patient's report of the sensation of heat as comfortable or painful is used to guide the intensity of treatment. If the patient cannot feel heat or pain because of impaired sensation or cannot report this sensation accurately and consistently because of impaired mentation or other factors affecting their ability to communicate, applying the treatment is not safe and therefore is contraindicated.

Although these conditions indicate the need for caution with the use of most physical agents, the specific contraindications and precautions for the agent being considered and the patient's situation must be evaluated before an intervention may be used or should be rejected. For example, although applying ultrasound to a pregnant patient is contraindicated in any area where the ultrasound may reach the fetus, this physical agent may be applied to the distal extremities of a pregnant patient because ultrasound penetration is shallow and limited to the area close to the applicator. In contrast, it is recommended that diathermy not be applied to any part of

a pregnant patient because the electromagnetic radiation it produces reaches areas distant from the applicator. Specific contraindications and precautions, including questions to ask the patient and features to assess before the application of each physical agent, are provided in Part II of this book.

Evaluation and Planning for the Use of Physical Agents

Physical agents have direct effects primarily at the level of impairment. These effects can improve activity and participation. For example, for a patient with pain that impairs motion, electrical currents can be used to stimulate sensory nerves to control pain and allow the patient to increase motion and thus increase activity, such as lifting objects, and participation, such as returning to work. Physical agents can also increase the effectiveness of other interventions and should generally be used to facilitate an active treatment program.[50] For example, a hot pack may be applied before stretching to increase the extensibility of superficial soft tissues and promote a safer and more effective increase in soft tissue length when the patient stretches.

When considering the application of a physical agent, one should first check the physician's referral, if one is required, for a medical diagnosis of the patient's condition and any necessary **precautions.** Precautions are conditions under which a particular treatment should be applied with special care or limitations. The therapist's examination should include, but should not be limited to, the patient's history, which would include information about the history of the current complaint, relevant medical history, and information about current and expected levels of activity and participation; a review of systems; and specific tests and measures. Examination findings and a survey of available evidence in the published literature should be considered in tandem to establish a prognosis and select the interventions and a plan of care, including anticipated goals. This plan may be modified as indicated through ongoing reexamination and reevaluation. The process of staying abreast of the latest clinical evidence is discussed in more detail in Chapter 2, and the sequence of examination, evaluation, and intervention follows in the case studies described in Part II of this book.

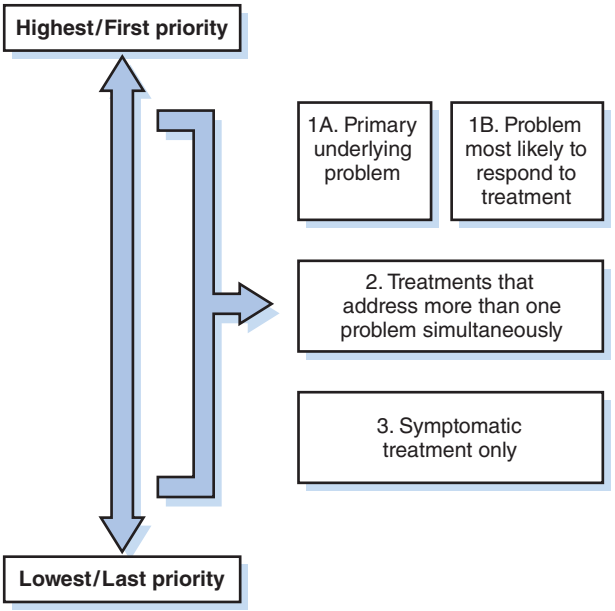

FIGURE 1.2 Prioritizing goals and effects of treatment.

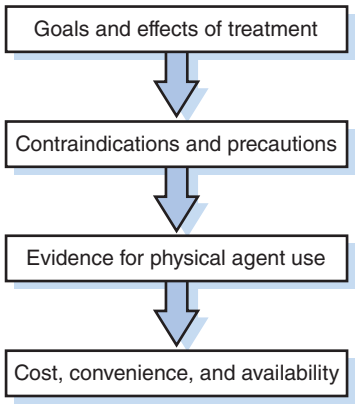

FIGURE 1.3 Attributes to be considered in the selection of physical agents.

CHOOSING A PHYSICAL AGENT

Physical agents generally assist in rehabilitation by reducing inflammation, pain, and motion restrictions; healing tissue; and improving muscle tone. Guidelines for selecting appropriate interventions based on the direct effects of physical agents are presented here in narrative form and are summarized in Tables 1.2 through 1.5. If the patient presents with more than one problem and so has numerous goals for treatment, only a limited number of goals should be addressed at any one time. It is generally recommended that the primary problems and problems most likely to respond to available interventions should be addressed first; however, the ideal intervention will facilitate progress in a number of areas (Fig. 1.2). For example, if a patient has knee pain caused by acute joint inflammation, treatment should first be directed at resolving the inflammation; however, the ideal intervention would also help to relieve pain. When the primary underlying problem, such as arthritis, cannot benefit directly from intervention with a physical agent, treatment with physical agents may still be used to help alleviate sequelae of these problems, such as pain or swelling.

ATTRIBUTES TO CONSIDER IN THE SELECTION OF PHYSICAL AGENTS

Given the variety of available physical agents and the unique characteristics of each patient, it is helpful to take a systematic approach to selecting the physical agents so that the ideal agent will be applied in each situation (Fig. 1.3).

Clinical Pearl

Because of the variety of available physical agents and the unique characteristics of each patient, it is important to take a systematic selection approach so that the ideal agent will be applied in each situation.

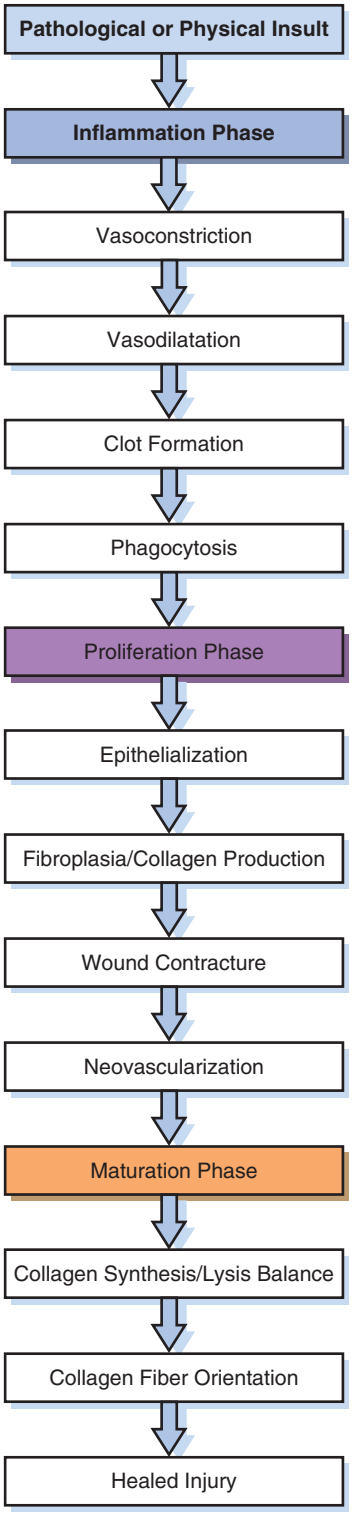

FIGURE 3.1 Flow diagram of the normal phases of inflammation and repair.

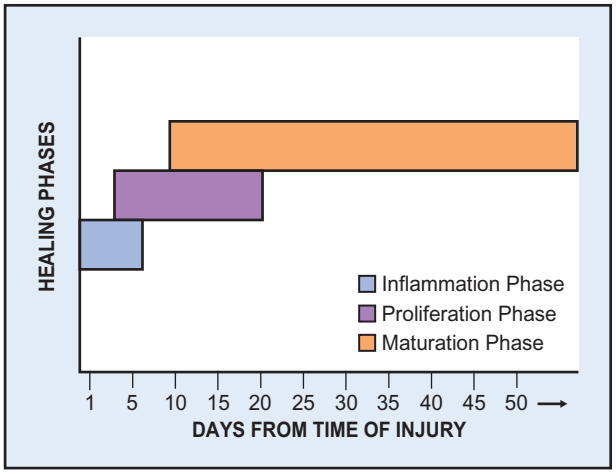

FIGURE 3.2 Timeline of the phases of inflammation and repair.

TABLE 3.1 Cardinal Signs of Inflammation

Sign (English)	Sign (Latin)	Cause
Heat	Calor	Increased vascularity
Redness	Rubor	Increased vascularity
Swelling	Tumor	Blockage of lymphatic drainage
Pain	Dolor	Physical pressure or chemical irritation of pain-sensitive structures
Loss of function	Functio laesa	Pain and swelling

by neurogenic and chemical mediators.[2] Local swelling results from increased permeability and vasodilation of local blood vessels and infiltration of fluid into interstitial spaces of the injured area. Pain results from the pressure of swelling and from irritation of pain-sensitive structures by chemicals released from damaged cells.[2] Both pain and swelling may result in loss of function.

There is some disagreement in the literature about the duration of the inflammation phase. Some investigators state that it is relatively short, lasting for less than 4 days[3,4]; others believe it may last for up to 6 days.[5,6] This discrepancy may be the result of individual and injury-specific variation or it may reflect the overlapping nature of phases of inflammation and tissue healing.

The inflammatory phase involves a complex sequence of interactive and overlapping events, including vascular, cellular, hemostatic, and immune processes. **Humoral mediators** and **neural mediators** act to control the inflammatory phase. Evidence indicates that immediately after injury, **platelets** and **neutrophils** predominate and release a number of factors that amplify the platelet aggregation response, initiate a coagulation cascade, or act as chemoattractants for cells involved in the inflammatory phase.[7] Neutrophil infiltration ceases after a few days, and neutrophils are replaced by **macrophages** starting 2 days after injury.[8] This shift in cell type at the site of injury correlates with a shift from the inflammation phase to the proliferation phase of healing.

Vascular Response

Alterations in anatomy and function of the microvasculature, including capillaries, postcapillary venules, and lymphatic vessels, are among the earliest responses noted in the inflammatory phase.[9] Trauma such as a laceration, a sprain, or a contusion physically disrupts these structures and may produce

bleeding, fluid loss, cell injury, and exposure of tissues to foreign material, including bacteria. Damaged vessels respond rapidly with transient constriction in an attempt to minimize blood loss. This response, which is mediated by norepinephrine, generally lasts for 5 to 10 minutes but can be prolonged in small vessels by serotonin released from mast cells and platelets.

After the transient vasoconstriction of injured vessels, noninjured vessels near the injured area dilate. Capillary permeability is also increased by injury to the capillary walls and in response to chemicals released from injured tissues (Fig. 3.3). The vasodilation and increase in capillary permeability are initiated by histamine, Hageman factor, bradykinin, prostaglandins, and complement fractions. Vasodilation and increased capillary permeability last for up to 1 hour after tissue damage.

Histamine is released primarily by mast cells, as well as by platelets and basophils at the injury site.[10] Histamine causes vasodilation and increased vascular permeability in venules, which contribute to local **edema** (swelling). Histamine also attracts **leukocytes** (white blood cells) to the damaged tissue area.[11] The ability of a chemical to attract cells is known as **chemotaxis.** Histamine is one of the first inflammatory mediators released after tissue injury and is active for approximately 1 hour after injury (Fig. 3.4).[12]

Hageman factor (also known as clotting factor XII), an enzyme found in the blood, is activated by contact with negatively charged surfaces of the endothelial lining of vessels that are exposed when vessels are damaged. The role of Hageman factor is twofold. First, it activates the coagulation system to stop local bleeding. Second, it causes vasoconstriction and increased vascular permeability by activating other **plasma** proteins. It converts plasminogen to plasmin and prekallikrein to kallikrein, and it activates the alternative complement pathway (Fig. 3.5).[13]

Plasmin augments vascular permeability in both skin and lungs by inducing breakdown of fibrin and by cleaving components of the **complement system.** Plasmin also activates Hageman factor, which initiates the cascade that generates bradykinin.

Plasma kallikrein attracts neutrophils and cleaves kininogen to generate several kinins such as bradykinin. Kinins are biologically active peptides that are potent inflammatory substances derived from plasma. Kinins, particularly bradykinin, function similarly to histamine, causing a marked increase in permeability of the microcirculation. They are most prevalent

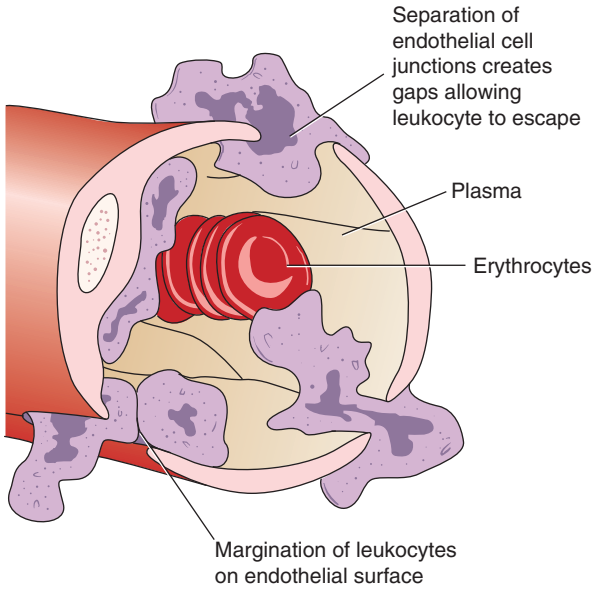

FIGURE 3.3 Vascular response to wound healing.

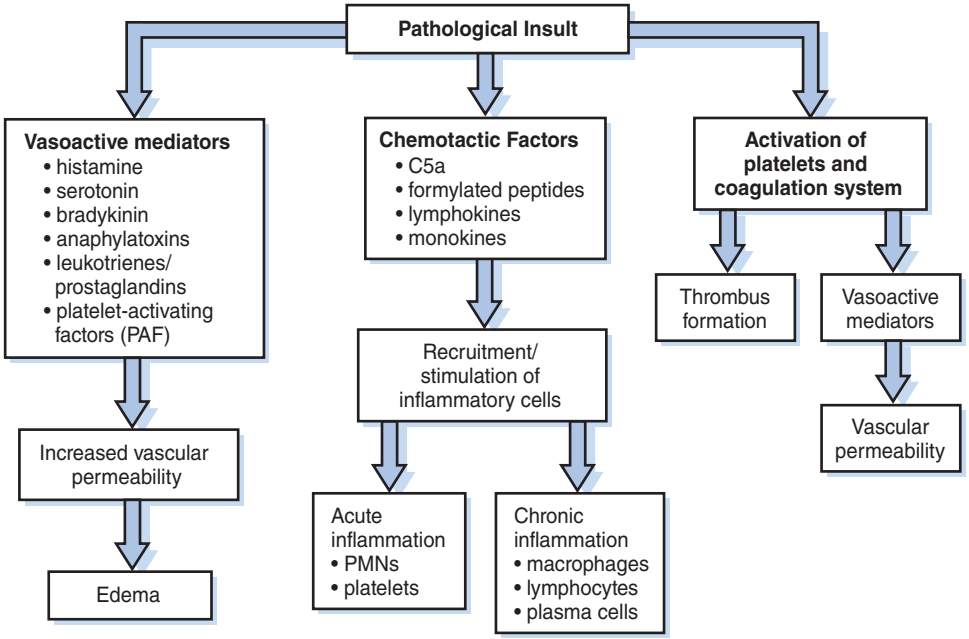

FIGURE 3.4 Mediators of the inflammatory response. *PMN,* Polymorphonucleocytes.

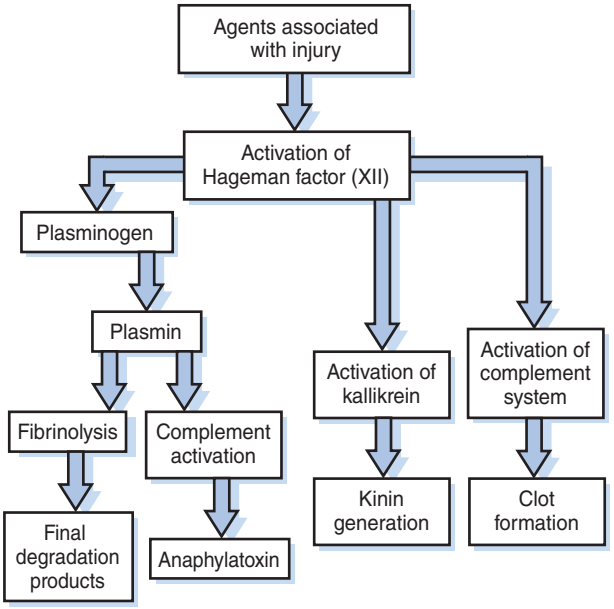

FIGURE 3.5 Hageman factor activation and inflammatory mediator production.

TABLE 3.2 Mediators of the Inflammatory Response

Response	Mediators
Vasodilation	Histamine Prostaglandins Serotonin
Increased vascular permeability	Bradykinin C3a, C5a PAF Histamine Serotonin Prostaglandins
Chemotaxis	Histamine C5a Monokines Kallikrein Lymphokines
Fever	Prostaglandins
Pain	Prostaglandins Hageman factor Bradykinin

PAF, Platelet-activating factor.

in the early phases of inflammation, after which they are rapidly destroyed by tissue proteases or kininases.[14]

Prostaglandins are produced by nearly all cells in the body and are released when the cell membrane is damaged. Two prostaglandins affect the inflammatory phase: prostaglandin E_1 (PGE_1) and PGE_2. PGE_1 increases vascular permeability by antagonizing vasoconstriction, and PGE_2 attracts leukocytes and synergizes the effects of other inflammatory mediators such as bradykinin. Proinflammatory prostaglandins are also thought to be responsible for sensitizing pain receptors and **hyperalgesia.** In the early stages of the healing response, prostaglandins may regulate the repair process; they are also responsible for the later stages of inflammation.[15] Nonsteroidal antiinflammatory drugs (NSAIDs) specifically work by inhibiting prostaglandin synthesis, whereas **corticosteroids** inhibit inflammation through this and other mechanisms. Because prostaglandins are responsible for febrile states, these medications are also effective in reducing fever. More recent studies suggest that proinflammatory growth factors including fibroblast growth factor and platelet-activating factor also contribute to hyperalgesia.[16,17]

The anaphylatoxins C3a, C4a, and C5a are important products of the complement system. These complement fractions cause increased vascular permeability and induce mast cell and basophil degranulation, causing further release of histamine and further increasing vascular permeability.

Aside from chemically mediated vascular changes (Table 3.2), changes in physical attraction between blood vessel walls also alter blood flow. During the initial vasoconstriction, the opposing walls of the small vessels become approximated, causing the linings of blood vessels to stick together. Under normal physiological conditions, the cell membranes of inflammatory cells and the basement membranes have mutually repulsive negative charges; however, after injury, this repulsion decreases, and polarity may be reversed. This results in decreased repulsion between circulating inflammatory cells and vessel walls and contributes to adherence of inflammatory cells to blood vessel linings.

As vasoconstriction of the postcapillary venules and increased permeability of the microvasculature cause blood flow to slow, an increase in cellular concentration occurs in the vessels, resulting in increased viscosity. Blood viscosity also increases as blood velocity slows because blood has shear-thinning properties.[18] In the normal physiological state, cellular components of blood within the microvasculature are confined to a central axial column, and the blood in contact with the vessel wall is relatively cell-free plasma.

Early in the inflammatory response, neutrophils, a type of leukocyte in the circulating blood, begin to migrate to the injured area. Within a few hours of injury, the bulk of neutrophils in the wound transmigrate across the capillary endothelial cell walls. The sequence of events in the journey of these cells from inside the blood vessel to the tissue outside the blood vessel is known as **extravasation.** Neutrophils break away from the central cellular column of blood and start to roll along the blood vessel lining (the endothelium) and adhere. They line the walls of the vessels in a process known as **margination.** Within 1 hour, the endothelial lining of the vessels can be completely covered with neutrophils. As these cells accumulate, they lay down in layers in a process known as **pavementing.** Certain mediators control the adherence of leukocytes to the endothelium, enhancing or inhibiting this process. For example, fibronectin, a glycoprotein present in plasma and basement membranes, has an important role in the modulation of cellular adherence to vessel walls. After injury to the vessels, increased amounts of fibronectin are deposited at the injury site. Adherence of leukocytes to the endothelium or the vascular basement membrane is critical for their recruitment to the site of injury.

After margination, neutrophils begin to squeeze through the vessel walls in a process known as **diapedesis.** Endothelial

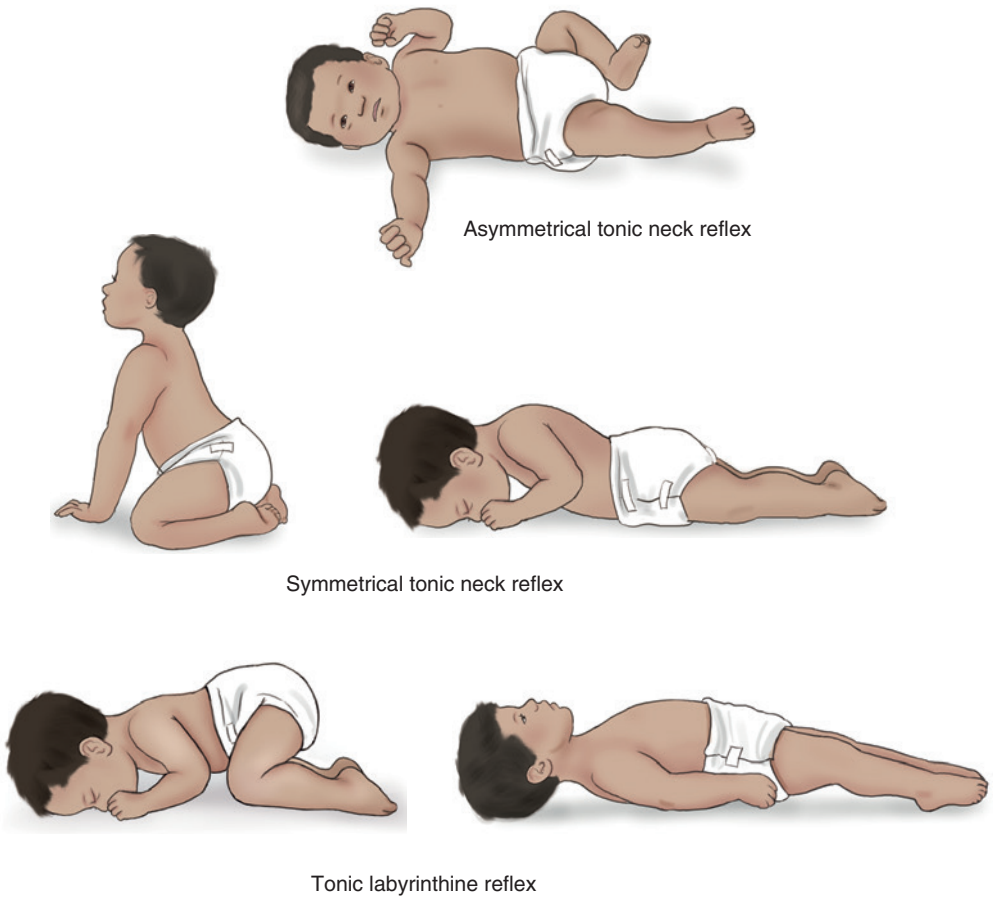

FIGURE 5.7 Reflex responses to head or neck position.

tone. For example, asymmetrical and symmetrical tonic neck reflexes (ATNR and STNR, respectively) are known to influence the tone of flexors and extensors of the arms and legs, depending on the position of the head (Fig. 5.7), both in infants and in patients with central nervous system disorders.[35] Even in subjects with mature and intact nervous systems, subtle differences in muscle tone can be detected by palpation when the head position changes and initiates one of these reflexes. Likewise, the pull of gravity on a limb to stretch muscles or on the **vestibular system** to trigger responses to keep the head upright will change muscle tone according to the position of the head and the body. Therefore the testing position must be reported for accurate interpretation and replication of any measurement of muscle tone.

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When documenting muscle tone, note the testing position.

Additional general guidelines for measuring muscle tone include standardization of touch and consideration of the muscle length at which a group of muscles is tested. The examiner must be aware that touching the patient's skin with a hand or with an instrument can influence muscle tone. For instance, a cold hand or stethoscope can change muscle tone when the touch is unexpected. Handholds and instrument temperature and placement must be consistent for accurate interpretation and replication. The length at which the tone of a specific muscle is tested must also be standardized. Because resistance to stretch differs with passive biomechanical differences at the extremes of range, and because ROM can be altered as a result of long-term changes in tone, the most consistent length to measure muscle tone is at the midrange of the available length of the muscle tested.

Clinical Pearl

Muscle tone is measured most accurately at the midrange of the muscle's length.

Anatomical Bases of Muscle Tone and Activation

Muscle tone and muscle activation originate from interactions between nervous system input and the biomechanical and biochemical properties of the muscle and its surrounding connective tissue. The practitioner must understand the anatomical basis for tone and activation to determine which physical agents to apply when either is dysfunctional. Anatomical contributions to muscle tone and activation are reviewed in this section.

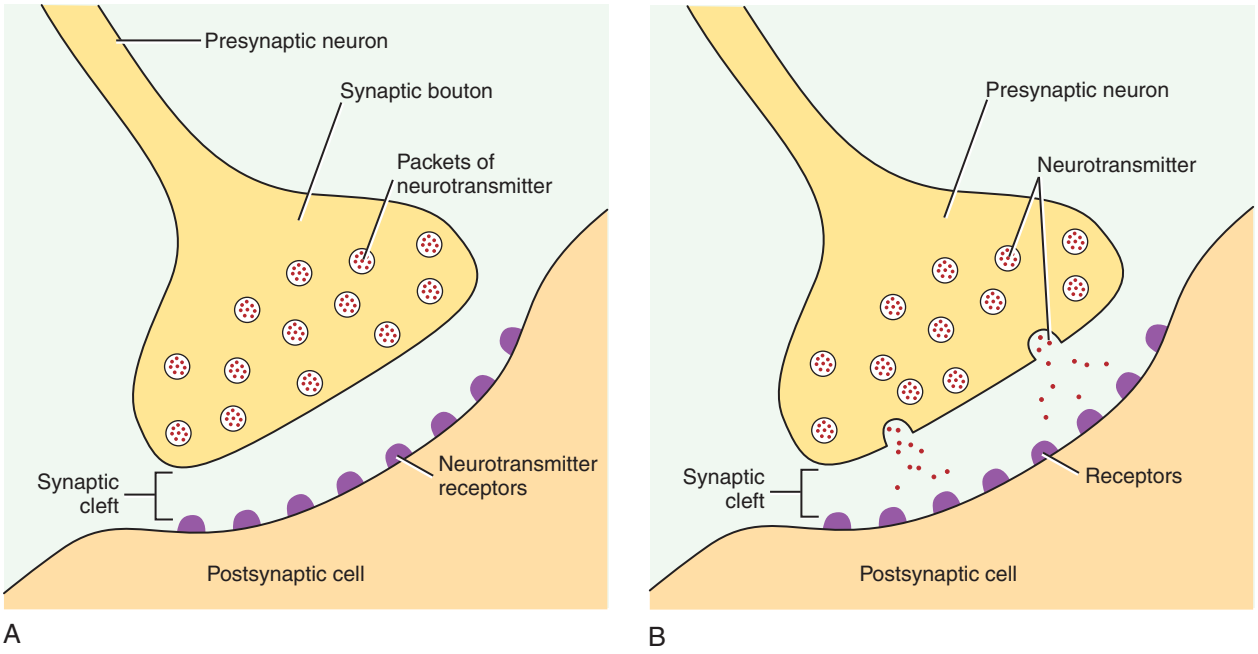

FIGURE 5.13 (A) Synapse between presynaptic and postsynaptic neurons at rest. (B) Synapse between presynaptic and postsynaptic neurons when activated.

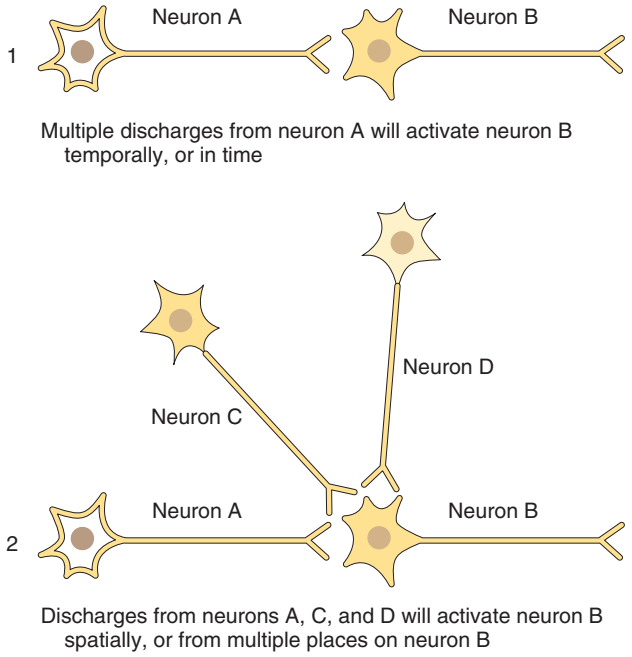

FIGURE 5.14 Temporal and spatial summation of input to a neuron.

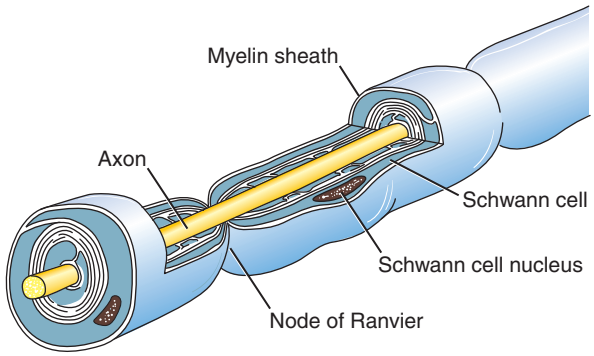

FIGURE 5.15 Myelin formed by Schwann cells on a peripheral neuron.

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Small-diameter axons and axons with little or no myelin conduct more slowly than large-diameter axons and highly myelinated axons.

Insulation speeds the transmission of a depolarizing wave by increasing the speed at which ions move across the membrane. A fatty tissue called **myelin,** provided by Schwann cells in the **peripheral nervous system (PNS)** and oligodendrocytes in the **central nervous system (CNS),** is the source of insulation for neurons. Myelin wraps around the axons of neurons, leaving gaps, known as nodes of Ranvier, at regular intervals (Fig. 5.15). When a depolarizing wave travels down an axon, it moves quickly down sections that have myelin and slows at the nodes of Ranvier. Because the signal slows at the nodes and travels very quickly between nodes, the signal appears to jump from one node to the next in rapid succession all the way to the end of all the axonal branches.[40] This jumping is referred to as **saltatory conduction** (Fig. 5.16).

The fastest nerve conduction velocities recorded in human nerves are 70 to 80 m/second.[41] Temperature changes can alter these velocities. When axons are cooled, as with the application of ice packs, nerve conduction velocity slows by approximately 2 m/second for every 1°C decrease in temperature.[42]

Clinical Pearl

In general, cold slows nerve conduction velocity, and heat accelerates nerve conduction velocity.

Once the signal reaches the synaptic boutons and neurotransmitters are released, a slight delay occurs as the molecules move across the synaptic cleft. Even at 200 Ångström units (200×10^{-10} m), it takes time for diffusion and then reception by the next neuron or target tissue. In addition, the receiving neuron must sum all its excitatory and inhibitory inputs before an action potential can develop. Therefore, if a signal is traveling the same distance on neurons of identical size, the pathway that contains more neurons (and therefore more synapses) will take longer to transmit the signal than the pathway with fewer neurons (and synapses). The shortest connection known is the single monosynaptic connection of the muscle stretch reflex, observable when certain tendons are tapped (Fig. 5.17). It is called *monosynaptic* because there is only one synapse between the sensory neuron receiving the stretch stimulus and the motor neuron transmitting the signal to the muscle fibers to contract.

Monosynaptic transmission, as recorded from muscle stretch (tap) to initiation of the muscle stretch reflex contraction, has been recorded in as little as 25 ms at the arm.[43] The time between stimulus and response is longer when multiple synapses are involved. For example, when the arm is reaching to catch a ball and visual input indicates a sudden change in the direction of the ball, it takes approximately 300 ms for the arm muscles to respond to that input.[43] If a person unexpectedly sees a ball begin to drop off a shelf 1 m overhead, the ball would fall approximately 44 cm before they could start the move to catch it.

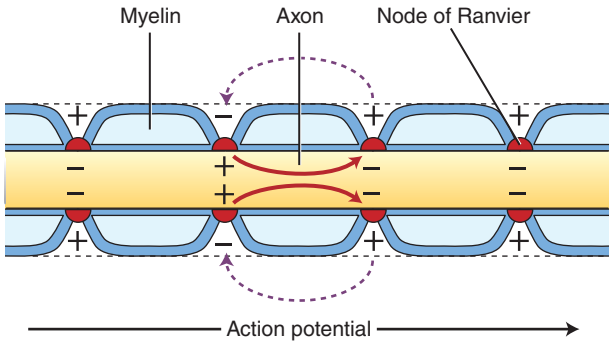

FIGURE 5.16 Saltatory conduction along a myelin-wrapped axon.

SOURCES OF NEURAL STIMULATION OF MUSCLE

Alpha Motor Neuron

Muscle tone and activation depend on alpha motor neurons for neural stimulation. An alpha motor neuron, sometimes called a **lower motor neuron,** transmits signals from the CNS to muscles. The lower motor neuron cell body is in the ventral horn of the spinal cord (see Fig. 5.17), and its axon exits the spinal cord and thus the CNS through the ventral nerve root. Each axon eventually reaches muscle, where it branches and innervates between 5 (in the eye muscles) and more than 1900 (in the gastrocnemius muscle) muscle fibers at motor end plates.[44] Muscle fibers innervated by a single axon with its branches, which constitute one **motor unit** (Fig. 5.18), all contract at once whenever an action potential is transmitted down that axon. A single action potential generated by the alpha motor neuron cannot provide its motor unit with a graded signal; each action potential is "all or none." When sufficient motor units are recruited, the muscle visibly contracts. More forceful contraction of the muscle requires an increased number or rate of action potentials down the same axons or recruitment of additional motor units.

Activation of a particular motor unit depends on the sum of excitatory and inhibitory input to that alpha motor neuron (Fig. 5.19). Excitation or inhibition depends on sources and amounts of input from the thousands of neurons that synapse on that one particular alpha motor neuron. Understanding the sources of input to alpha motor neurons is essential for understanding the control of motor unit activation and thus alteration of muscle tone by physical agents or other means (Table 5.3).

Input From the Periphery

The PNS includes all the neurons that project outside of the CNS, even if the cell bodies are located within the CNS. The PNS is composed of alpha motor neurons, **gamma motor**

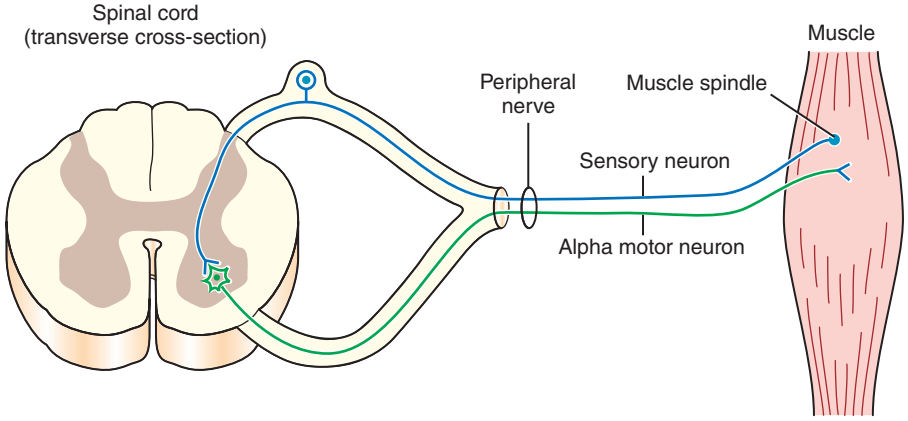

FIGURE 5.17 Monosynaptic muscle stretch reflex.