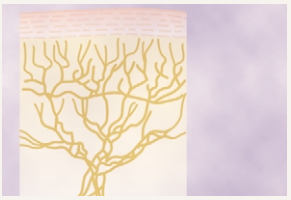
C H A P T E R 4 6

Sensory Receptors, Neuronal Circuits for Processing Information



Input to the nervous system is provided by sensory receptors that detect such sensory stimuli as touch, sound, light, pain, cold, and warmth. The purpose of this chapter is to discuss the basic mechanisms by which these receptors change sensory stimuli into nerve signals that are then conveyed to and processed in the central nervous system.

Types of Sensory Receptors and the Sensory Stimuli They Detect

Table 46–1 lists and classifies most of the body's sensory receptors. This table shows that there are five basic types of sensory receptors: (1) *mechanoreceptors*, which detect mechanical compression or stretching of the receptor or of tissues adjacent to the receptor; (2) *thermoreceptors*, which detect changes in temperature, some receptors detecting cold and others warmth; (3) *nociceptors* (pain receptors), which detect damage occurring in the tissues, whether physical damage or chemical damage; (4) *electromagnetic receptors*, which detect light on the retina of the eye; and (5) *chemoreceptors*, which detect taste in the mouth, smell in the nose, oxygen level in the arterial blood, osmolality of the body fluids, carbon dioxide concentration, and perhaps other factors that make up the chemistry of the body.

In this chapter, we discuss the function of a few specific types of receptors, primarily peripheral mechanoreceptors, to illustrate some of the principles by which receptors operate. Other receptors are discussed in other chapters in relation to the sensory systems that they subserve. Figure 46–1 shows some of the types of mechanoreceptors found in the skin or in deep tissues of the body.

Differential Sensitivity of Receptors

The first question that must be answered is, how do two types of sensory receptors detect different types of sensory stimuli? The answer is, by "differential sensitivities." That is, each type of receptor is highly sensitive to one type of stimulus for which it is designed and yet is almost nonresponsive to other types of sensory stimuli. Thus, the rods and cones of the eyes are highly responsive to light but are almost completely nonresponsive to normal ranges of heat, cold, pressure on the eyeballs, or chemical changes in the blood. The osmoreceptors of the supraoptic nuclei in the hypothalamus detect minute changes in the osmolality of the body fluids but have never been known to respond to sound. Finally, pain receptors in the skin are almost never stimulated by usual touch or pressure stimuli but do become highly active the moment tactile stimuli become severe enough to damage the tissues.

Modality of Sensation—The "Labeled Line" Principle

Each of the principal types of sensation that we can experience—pain, touch, sight, sound, and so forth—is called a *modality* of sensation. Yet despite the fact that we experience these different modalities of sensation, nerve fibers

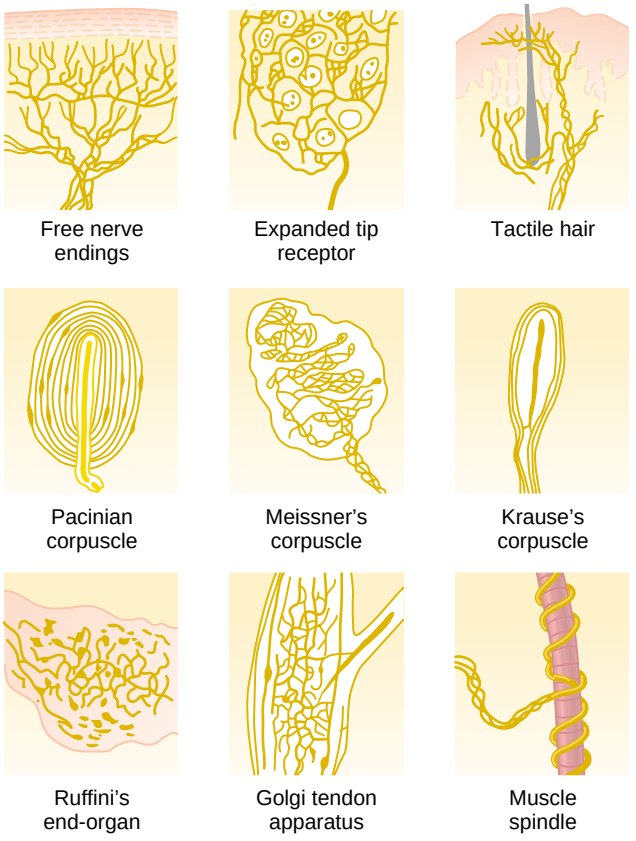


Figure 46–1

Several types of somatic sensory nerve endings.

transmit only impulses. Therefore, how is it that different nerve fibers transmit different modalities of sensation?

The answer is that each nerve tract terminates at a specific point in the central nervous system, and the type of sensation felt when a nerve fiber is stimulated is determined by the point in the nervous system to which the fiber leads. For instance, if a pain fiber is stimulated, the person perceives pain regardless of what type of stimulus excites the fiber. The stimulus can be electricity, overheating of the fiber, crushing of the fiber, or stimulation of the pain nerve ending by damage to the tissue cells. In all these instances, the person perceives pain. Likewise, if a touch fiber is stimulated by electrical excitation of a touch receptor or in any other way, the person perceives touch because touch fibers lead to specific touch areas in the brain. Similarly, fibers from the retina of the eye terminate in the vision areas of the brain, fibers from the ear terminate in the auditory areas of the brain, and temperature fibers terminate in the temperature areas.

This specificity of nerve fibers for transmitting only one modality of sensation is called the *labeled line principle*.

Table 46–1

Classification of Sensory Receptors

- **I. Mechanoreceptors**
  - Skin tactile sensibilities (epidermis and dermis)
    - Free nerve endings
    - Expanded tip endings
      - Merkel's discs
      - Plus several other variants
    - Spray endings
    - Ruffini's endings
    - Encapsulated endings
      - Meissner's corpuscles
      - Krause's corpuscles
    - Hair end-organs
  - Deep tissue sensibilities
    - Free nerve endings
    - Expanded tip endings
    - Spray endings
      - Ruffini's endings
    - Encapsulated endings
      - Pacinian corpuscles
      - Plus a few other variants
    - Muscle endings
      - Muscle spindles
      - Golgi tendon receptors
  - Hearing
    - Sound receptors of cochlea
  - Equilibrium
    - Vestibular receptors
  - Arterial pressure
    - Baroreceptors of carotid sinuses and aorta
- **II. Thermoreceptors**
  - Cold
    - Cold receptors
  - Warmth
    - Warm receptors
- **III. Nociceptors**
  - Pain
    - Free nerve endings
- **IV. Electromagnetic receptors**
  - Vision
    - Rods
    - Cones
- **V. Chemoreceptors**
  - Taste
    - Receptors of taste buds
  - Smell
    - Receptors of olfactory epithelium
  - Arterial oxygen
    - Receptors of aortic and carotid bodies
  - Osmolality
    - Neurons in or near supraoptic nuclei
  - Blood CO_2
    - Receptors in or on surface of medulla and in aortic and carotid bodies
  - Blood glucose, amino acids, fatty acids
    - Receptors in hypothalamus

Transduction of Sensory Stimuli into Nerve Impulses

Local Electrical Currents at Nerve Endings—Receptor Potentials

All sensory receptors have one feature in common. Whatever the type of stimulus that excites the receptor, its immediate effect is to change the membrane

electrical potential of the receptor. This change in potential is called a *receptor potential*.

Mechanisms of Receptor Potentials. Different receptors can be excited in one of several ways to cause receptor potentials: (1) by mechanical deformation of the receptor, which stretches the receptor membrane and opens ion channels; (2) by application of a chemical to the membrane, which also opens ion channels; (3) by change of the temperature of the membrane, which alters the permeability of the membrane; or (4) by the effects of electromagnetic radiation, such as light on a retinal visual receptor, which either directly or indirectly changes the receptor membrane characteristics and allows ions to flow through membrane channels. It will be recognized that these four means of exciting receptors correspond in general with the different types of known sensory receptors. In all instances, the basic cause of the change in membrane potential is a change in membrane permeability of the receptor, which allows ions to diffuse more or less readily through the membrane and thereby to change the *transmembrane potential*.

Maximum Receptor Potential Amplitude. The maximum amplitude of most sensory receptor potentials is about 100 millivolts, but this level occurs only at an extremely high intensity of sensory stimulus. This is about the same maximum voltage recorded in action potentials and is also the change in voltage when the membrane becomes maximally permeable to sodium ions.

Relation of the Receptor Potential to Action Potentials. When the receptor potential rises above the *threshold* for eliciting action potentials in the nerve fiber attached to the receptor, then action potentials occur, as illustrated in Figure 46–2. Note also that the more the receptor potential rises above the threshold level, the greater becomes the *action potential frequency*.

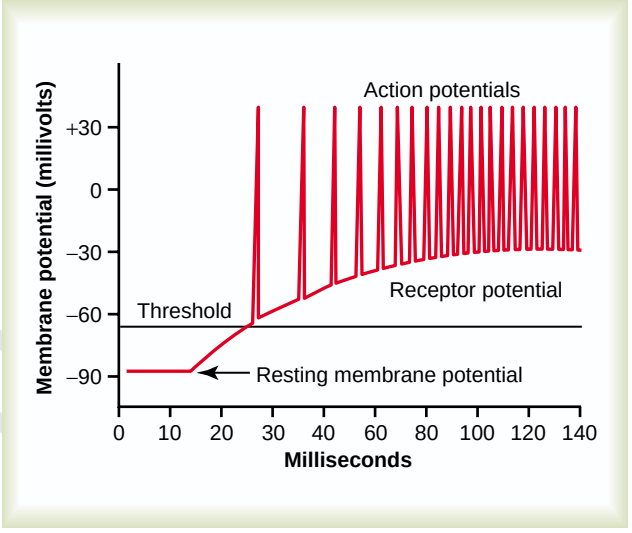


Figure 46–2

Typical relation between receptor potential and action potentials when the receptor potential rises above threshold level.

Receptor Potential of the Pacinian Corpuscle—An Example of Receptor Function

The student should at this point restudy the anatomical structure of the pacinian corpuscle shown in Figure 46–1. Note that the corpuscle has a central nerve fiber extending through its core. Surrounding this are multiple concentric capsule layers, so that compression anywhere on the outside of the corpuscle will elongate, indent, or otherwise deform the central fiber.

Now study Figure 46–3, which shows only the central fiber of the pacinian corpuscle after all capsule layers but one have been removed. The tip of the central fiber inside the capsule is unmyelinated, but the fiber does become myelinated (the blue sheath shown in the figure) shortly before leaving the corpuscle to enter a peripheral sensory nerve.

The figure also shows the mechanism by which a receptor potential is produced in the pacinian corpuscle. Observe the small area of the terminal fiber that has been deformed by compression of the corpuscle, and note that ion channels have opened in the membrane, allowing positively charged sodium ions to diffuse to the interior of the fiber. This creates increased positivity inside the fiber, which is the "receptor potential." The receptor potential in turn induces a *local circuit* of current flow, shown by the arrows, that spreads along the nerve fiber. At the first node of Ranvier, which itself lies inside the capsule of the pacinian corpuscle, the local current flow depolarizes the fiber membrane at this node, which then sets off typical action potentials that are transmitted along the nerve fiber toward the central nervous system.

Relation Between Stimulus Intensity and the Receptor Potential. Figure 46–4 shows the changing amplitude of the receptor potential caused by progressively stronger mechanical compression (increasing "stimulus strength") applied experimentally to the central core of a pacinian corpuscle. Note that the amplitude

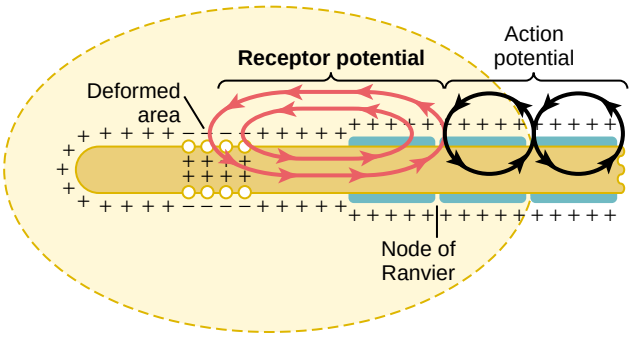


Figure 46–3

Excitation of a sensory nerve fiber by a receptor potential produced in a pacinian corpuscle. (Modified from Loëwenstein WR: Excitation and inactivation in a receptor membrane. Ann N Y Acad Sci 94:510, 1961.)

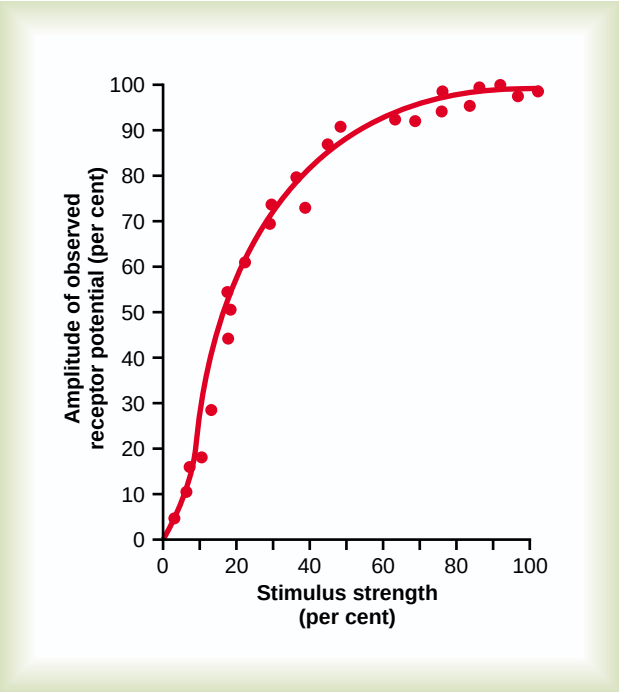


Figure 46–4

Relation of amplitude of receptor potential to strength of a mechanical stimulus applied to a pacinian corpuscle. (Data from Loëwenstein WR: Excitation and inactivation in a receptor membrane. Ann N Y Acad Sci 94:510, 1961.)

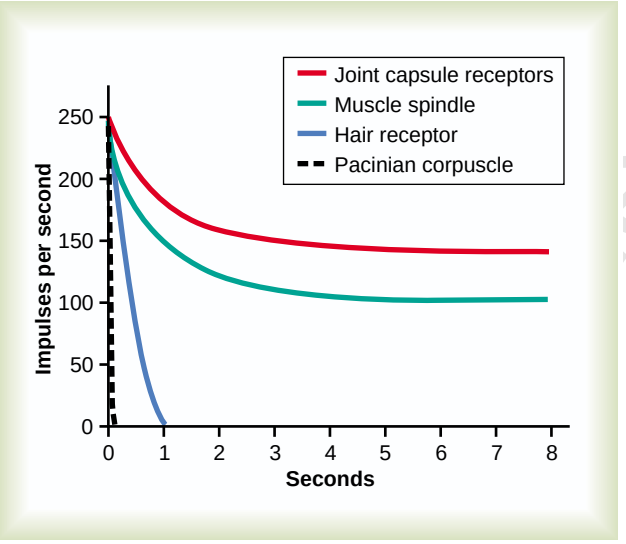


Figure 46–5

Adaptation of different types of receptors, showing rapid adaptation of some receptors and slow adaptation of others.

increases rapidly at first but then progressively less rapidly at high stimulus strength.

In turn, the *frequency of repetitive action potentials* transmitted from sensory receptors increases approximately in proportion to the increase in receptor potential. Putting this principle together with the data in Figure 46–4, one can see that very intense stimulation of the receptor causes progressively less and less additional increase in numbers of action potentials. This is an exceedingly important principle that is applicable to almost all sensory receptors. It allows the receptor to be sensitive to very weak sensory experience and yet not reach a maximum firing rate until the sensory experience is extreme. This allows the receptor to have an extreme range of response, from very weak to very intense.

Adaptation of Receptors

Another characteristic of all sensory receptors is that they *adapt* either partially or completely to any constant stimulus after a period of time. That is, when a continuous sensory stimulus is applied, the receptor responds at a high impulse rate at first and then at a progressively slower rate until finally the rate of action potentials decreases to very few or often to none at all.

Figure 46–5 shows typical adaptation of certain types of receptors. Note that the pacinian corpuscle adapts extremely rapidly and hair receptors adapt within a second or so, whereas some joint capsule and muscle spindle receptors adapt slowly.

Furthermore, some sensory receptors adapt to a far greater extent than others. For example, the pacinian corpuscles adapt to "extinction" within a few hundredths of a second, and the receptors at the bases of the hairs adapt to extinction within a second or more. It is probable that all other *mechanoreceptors* eventually adapt almost completely, but some require hours or days to do so, for which reason they are called "nonadapting" receptors. The longest measured time for complete adaptation of a mechanoreceptor is about 2 days, which is the adaptation time for many carotid and aortic baroreceptors. Conversely, some of the nonmechanoreceptors—the chemoreceptors and pain receptors, for instance—probably never adapt completely.

Mechanisms by Which Receptors Adapt. The mechanism of receptor adaptation is different for each type of receptor, in much the same way that development of a receptor potential is an individual property. For instance, in the eye, the rods and cones adapt by changing the concentrations of their light-sensitive chemicals (which is discussed in Chapter 50).

In the case of the mechanoreceptors, the receptor that has been studied in greatest detail is the pacinian corpuscle. Adaptation occurs in this receptor in two ways. First, the pacinian corpuscle is a viscoelastic structure so that when a distorting force is suddenly applied to one side of the corpuscle, this force is instantly transmitted by the viscous component of the corpuscle directly to the same side of the central nerve fiber, thus eliciting a receptor potential. However, within a few hundredths of a second, the fluid within the corpuscle redistributes, so that the receptor potential is no longer elicited. Thus, the receptor potential

appears at the onset of compression but disappears within a small fraction of a second even though the compression continues.

The second mechanism of adaptation of the pacinian corpuscle, but a much slower one, results from a process called *accommodation*, which occurs in the nerve fiber itself. That is, even if by chance the central core fiber should continue to be distorted, the tip of the nerve fiber itself gradually becomes "accommodated" to the stimulus. This probably results from progressive "inactivation" of the sodium channels in the nerve fiber membrane, which means that sodium current flow through the channels causes them gradually to close, an effect that seems to occur for all or most cell membrane sodium channels, as was explained in Chapter 5.

Presumably, these same two general mechanisms of adaptation apply also to the other types of mechanoreceptors. That is, part of the adaptation results from readjustments in the structure of the receptor itself, and part from an electrical type of accommodation in the terminal nerve fibril.

Slowly Adapting Receptors Detect Continuous Stimulus Strength—The "Tonic" Receptors. Slowly adapting receptors continue to transmit impulses to the brain as long as the stimulus is present (or at least for many minutes or hours). Therefore, they keep the brain constantly apprised of the status of the body and its relation to its surroundings. For instance, impulses from the muscle spindles and Golgi tendon apparatuses allow the nervous system to know the status of muscle contraction and load on the muscle tendon at each instant.

Other slowly adapting receptors include (1) receptors of the macula in the vestibular apparatus, (2) pain receptors, (3) baroreceptors of the arterial tree, and (4) chemoreceptors of the carotid and aortic bodies.

Because the slowly adapting receptors can continue to transmit information for many hours, they are called *tonic* receptors.

Rapidly Adapting Receptors Detect Change in Stimulus Strength—The "Rate Receptors," "Movement Receptors," or "Phasic Receptors." Receptors that adapt rapidly cannot be used to transmit a continuous signal because these receptors are stimulated only when the stimulus strength changes. Yet they react strongly *while a change is actually taking place*. Therefore, these receptors are called *rate* receptors, *movement* receptors, or *phasic* receptors. Thus, in the case of the pacinian corpuscle, sudden pressure applied to the tissue excites this receptor for a few milliseconds, and then its excitation is over even though the pressure continues. But later, it transmits a signal again when the pressure is released. In other words, the pacinian corpuscle is exceedingly important in apprising the nervous system of rapid tissue deformations, but it is useless for transmitting information about constant conditions in the body.

Importance of the Rate Receptors—Their Predictive Function. If one knows the rate at which some change in bodily status is taking place, one can predict in one's mind the state of the body a few seconds or even a few minutes later. For instance, the receptors of the semicircular canals in the vestibular apparatus of the ear detect the rate at which the head begins to turn when one runs around a curve. Using this information, a person can predict how much he or she will turn within the next 2 seconds and can adjust the motion of the legs *ahead of time* to keep from losing balance. Likewise, receptors located in or near the joints help detect the rates of movement of the different parts of the body. For instance, when one is running, information from the joint rate receptors allows the nervous system to predict where the feet will be during any precise fraction of the next second. Therefore, appropriate motor signals can be transmitted to the muscles of the legs to make any necessary anticipatory corrections in position so that the person will not fall. Loss of this predictive function makes it impossible for the person to run.

Nerve Fibers That Transmit Different Types of Signals, and Their Physiologic Classification

Some signals need to be transmitted to or from the central nervous system extremely rapidly; otherwise, the information would be useless. An example of this is the sensory signals that apprise the brain of the momentary positions of the legs at each fraction of a second during running. At the other extreme, some types of sensory information, such as that depicting prolonged, aching pain, do not need to be transmitted rapidly, so that slowly conducting fibers will suffice. As shown in Figure 46–6, nerve fibers come in all sizes between 0.5 and 20 micrometers in diameter—the larger the diameter, the greater the conducting velocity. The range of conducting velocities is between 0.5 and 120 m/sec.

General Classification of Nerve Fibers. Shown in Figure 46–6 is a "general classification" and a "sensory nerve classification" of the different types of nerve fibers. In the general classification, the fibers are divided into types A and C, and the type A fibers are further subdivided into α, β, γ, and δ fibers.

Type A fibers are the typical large and medium-sized *myelinated* fibers of spinal nerves. Type C fibers are the small *unmyelinated* nerve fibers that conduct impulses at low velocities. The C fibers constitute more than one half of the sensory fibers in most peripheral nerves as well as all the postganglionic autonomic fibers.

The sizes, velocities of conduction, and functions of the different nerve fiber types are also given in Figure 46–6. Note that a few large myelinated fibers can transmit impulses at velocities as great as 120 m/sec, a distance in 1 second that is longer than a football field. Conversely, the smallest fibers transmit impulses as slowly as 0.5 m/sec, requiring about 2 seconds to go from the big toe to the spinal cord.

Alternative Classification Used by Sensory Physiologists. Certain recording techniques have made it possible to separate the type Aα fibers into two subgroups; yet

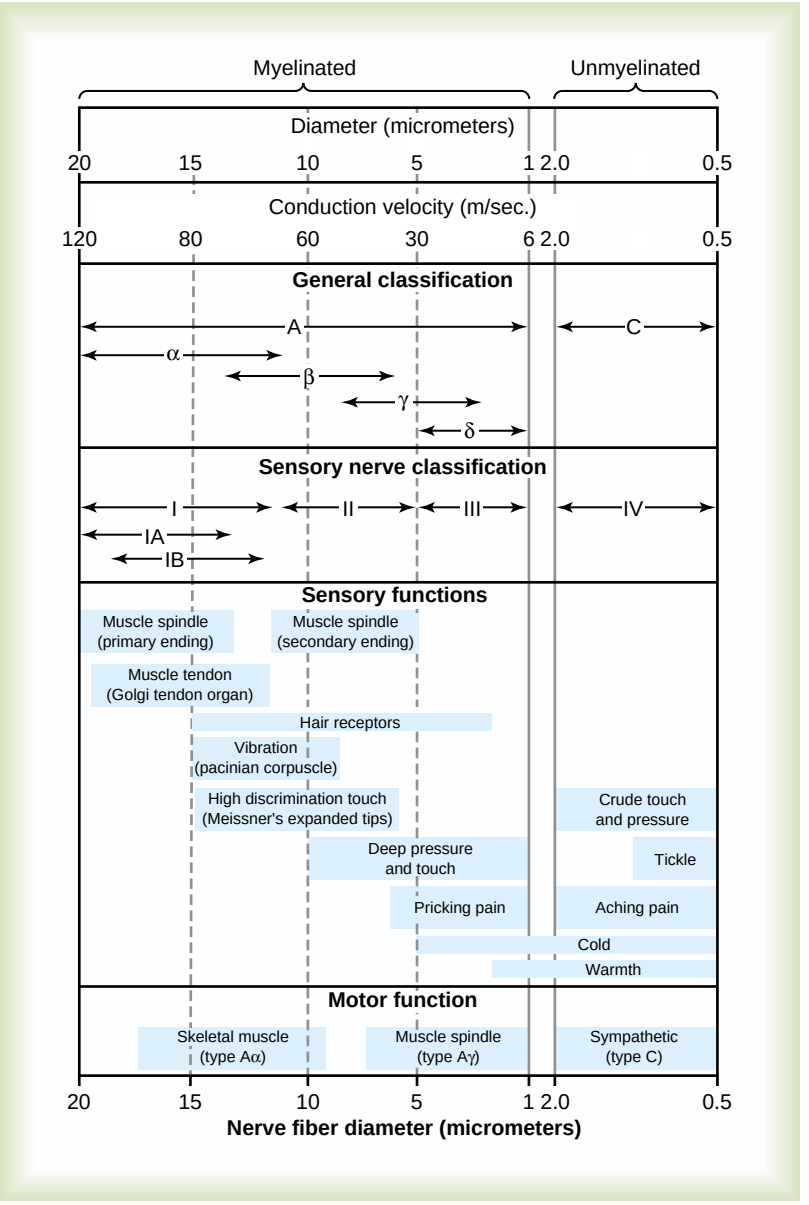


Figure 46–6

Physiologic classifications and functions of nerve fibers.

these same recording techniques cannot distinguish easily between Aβ and Aγ fibers. Therefore, the following classification is frequently used by sensory physiologists:

Group Ia
Fibers from the annulospiral endings of muscle spindles (average about 17 microns in diameter; these are α-type A fibers in the general classification).

Group Ib
Fibers from the Golgi tendon organs (average about 16 micrometers in diameter; these also are α-type A fibers).

Group II
Fibers from most discrete cutaneous tactile receptors and from the flower-spray endings of the muscle spindles (average about 8 micrometers in diameter; these are β- and γ-type A fibers in the general classification).

Group III
Fibers carrying temperature, crude touch, and pricking pain sensations (average about 3 micrometers in diameter; they are δ-type A fibers in the general classification).

Group IV
Unmyelinated fibers carrying pain, itch, temperature, and crude touch sensations (0.5 to 2 micrometers in diameter; they are type C fibers in the general classification).

Transmission of Signals of Different Intensity in Nerve Tracts—Spatial and Temporal Summation

One of the characteristics of each signal that always must be conveyed is signal intensity—for instance, the intensity of pain. The different gradations of intensity

can be transmitted either by using increasing numbers of parallel fibers or by sending more action potentials along a single fiber. These two mechanisms are called, respectively, spatial summation and temporal summation.

Spatial Summation. Figure 46–7 shows the phenomenon of *spatial summation*, whereby increasing signal strength is transmitted by using progressively greater numbers of fibers. This figure shows a section of skin innervated by a large number of parallel pain fibers. Each of these arborizes into hundreds of minute *free nerve endings* that serve as pain receptors. The entire cluster of fibers from one pain fiber frequently covers an area of skin as large as 5 centimeters in diameter. This area is called the *receptor field* of that fiber. The number of endings is large in the center of the field but diminishes toward the periphery. One can also see from the figure that the arborizing fibrils overlap those from other pain fibers. Therefore, a pinprick of the skin usually stimulates endings from many different pain fibers simultaneously. When the pinprick is in the center of the receptive field of a particular pain fiber, the degree of stimulation of that fiber is far greater than when it is in the periphery of the field, because the number of free nerve endings in the middle of the field is much greater than at the periphery.

Thus, the lower part of Figure 46–7 shows three views of the cross section of the nerve bundle leading from the skin area. To the left is the effect of a weak stimulus, with only a single nerve fiber in the middle of the bundle stimulated strongly (represented by the red-colored fiber), whereas several adjacent fibers are stimulated weakly (half-red fibers). The other two views of the nerve cross section show the effect of a moderate stimulus and a strong stimulus, with progressively more fibers being stimulated. Thus, the stronger signals spread to more and more fibers. This is the phenomenon of *spatial summation*.

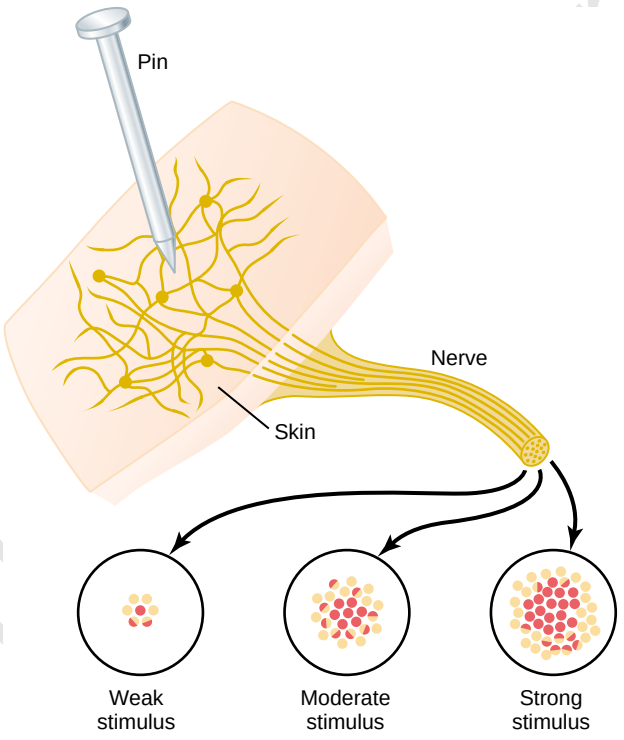


Figure 46–7

Pattern of stimulation of pain fibers in a nerve leading from an area of skin pricked by a pin. This is an example of *spatial summation*.

Temporal Summation. A second means for transmitting signals of increasing strength is by increasing the *frequency* of nerve impulses in each fiber, which is called *temporal summation*. Figure 46–8 demonstrates this, showing in the upper part a changing strength of signal and in the lower part the actual impulses transmitted by the nerve fiber.

Transmission and Processing of Signals in Neuronal Pools

The central nervous system is composed of thousands to millions of neuronal pools; some of these contain few neurons, while others have vast numbers. For instance, the entire cerebral cortex could be considered to be a single large neuronal pool. Other neuronal pools include the different basal ganglia and the specific nuclei in the thalamus, cerebellum, mesencephalon, pons, and medulla. Also, the entire dorsal gray matter of the spinal cord could be considered one long pool of neurons.

Each neuronal pool has its own special organization that causes it to process signals in its own unique way,

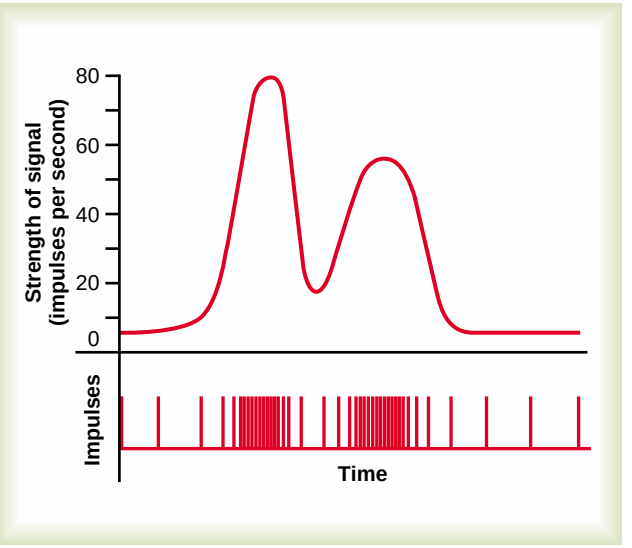


Figure 46–8

Translation of signal strength into a frequency-modulated series of nerve impulses, showing the strength of signal (*above*) and the separate nerve impulses (*below*). This is an example of *temporal summation*.

thus allowing the total consortium of pools to achieve the multitude of functions of the nervous system. Yet despite their differences in function, the pools also have many similar principles of function, described in the following pages.

Relaying of Signals Through Neuronal Pools

Organization of Neurons for Relaying Signals. Figure 46–9 is a schematic diagram of several neurons in a neuronal pool, showing "input" fibers to the left and "output" fibers to the right. Each input fiber divides hundreds to thousands of times, providing a thousand or more terminal fibrils that spread into a large area in the pool to synapse with dendrites or cell bodies of the neurons in the pool. The dendrites usually also arborize and spread hundreds to thousands of micrometers in the pool.

The neuronal area stimulated by each incoming nerve fiber is called its *stimulatory field*. Note in Figure 46–9 that large numbers of the terminals from each input fiber lie on the nearest neuron in its "field," but progressively fewer terminals lie on the neurons farther away.

Threshold and Subthreshold Stimuli—Excitation or Facilitation. From the discussion of synaptic function in Chapter 45, it will be recalled that discharge of a single excitatory presynaptic terminal almost never causes an action potential in a postsynaptic neuron. Instead, large numbers of input terminals must discharge on the same neuron either simultaneously or in rapid succession to cause excitation. For instance, in Figure 46–9, let us assume that six terminals must discharge almost simultaneously to excite any one of the neurons. If the student counts the number of terminals on each one of the neurons from each input fiber, he or she will see that *input fiber 1* has more than enough terminals to cause *neuron a* to discharge. The stimulus from input fiber 1 to this neuron is said to be an *excitatory stimulus*; it is also called a *suprathreshold stimulus* because it is above the threshold required for excitation.

Input fiber 1 also contributes terminals to neurons b and c, but not enough to cause excitation. Nevertheless, discharge of these terminals makes both these neurons more likely to be excited by signals arriving through other incoming nerve fibers. Therefore, the stimuli to these neurons are said to be *subthreshold*, and the neurons are said to be *facilitated*.

Similarly, for *input fiber 2*, the stimulus to *neuron d* is a suprathreshold stimulus, and the stimuli to *neurons b* and *c* are subthreshold, but facilitating, stimuli.

Figure 46–9 represents a highly condensed version of a neuronal pool because each input nerve fiber usually provides massive numbers of branching terminals to hundreds or thousands of neurons in its distribution "field," as shown in Figure 46–10. In the central portion of the field in this figure, designated by the circled area, all the neurons are stimulated by the incoming fiber. Therefore, this is said to be the *discharge zone* of the incoming fiber, also called the *excited zone* or *liminal zone*. To each side, the neurons are facilitated but not excited, and these areas are called the *facilitated zone,* also called the *subthreshold zone* or *subliminal zone*.

Inhibition of a Neuronal Pool. We must also remember that some incoming fibers inhibit neurons, rather than exciting them. This is the opposite of facilitation, and the entire field of the inhibitory branches is called the *inhibitory zone.* The degree of inhibition in the center of this zone is great because of large numbers of endings in the center; it becomes progressively less toward its edges.

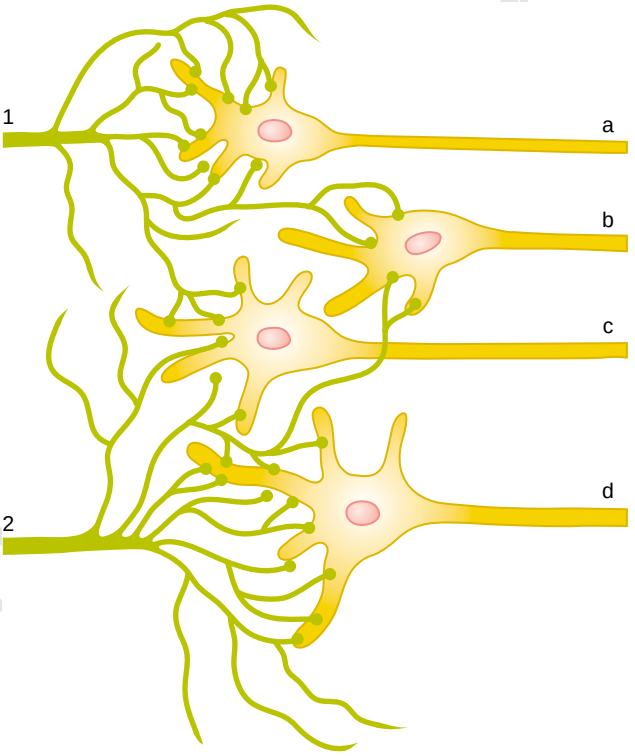


Figure 46–9

Basic organization of a neuronal pool.

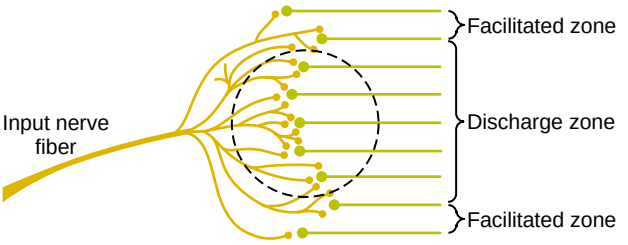


Figure 46–10

"Discharge" and "facilitated" zones of a neuronal pool.

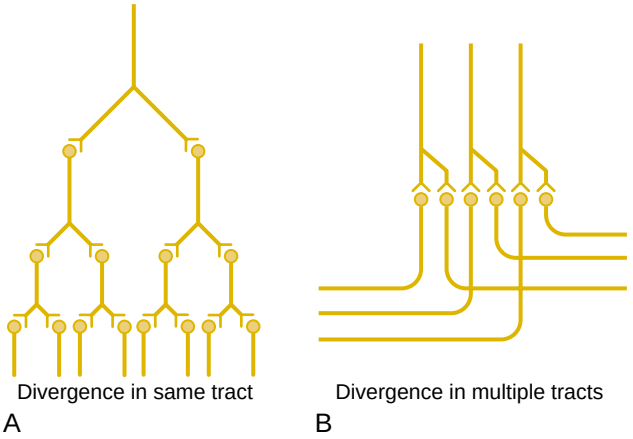


Figure 46–11

"Divergence" in neuronal pathways. *A,* Divergence within a pathway to cause "amplification" of the signal. *B,* Divergence into multiple tracts to transmit the signal to separate areas.

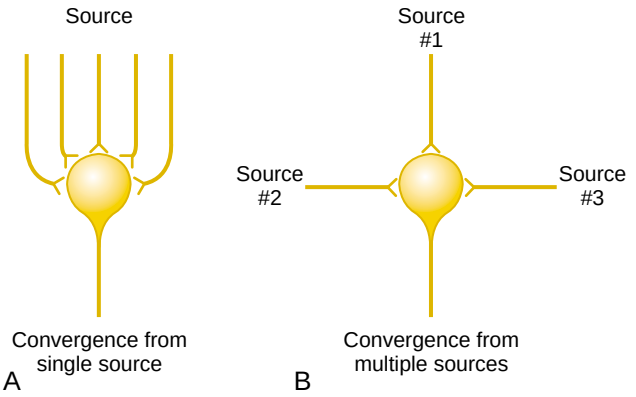


Figure 46–12

"Convergence" of multiple input fibers onto a single neuron. *A,* Multiple input fibers from a single source. *B,* Input fibers from multiple separate sources.

Divergence of Signals Passing Through Neuronal Pools

Often it is important for weak signals entering a neuronal pool to excite far greater numbers of nerve fibers leaving the pool. This phenomenon is called *divergence.* Two major types of divergence occur and have entirely different purposes.

An *amplifying* type of divergence is shown in Figure 46–11*A*. This means simply that an input signal spreads to an increasing number of neurons as it passes through successive orders of neurons in its path. This type of divergence is characteristic of the corticospinal pathway in its control of skeletal muscles, with a single large pyramidal cell in the motor cortex capable, under highly facilitated conditions, of exciting as many as 10,000 muscle fibers.

The second type of divergence, shown in Figure 46–11*B,* is *divergence into multiple tracts*. In this case, the signal is transmitted in two directions from the pool. For instance, information transmitted up the dorsal columns of the spinal cord takes two courses in the lower part of the brain: (1) into the cerebellum and (2) on through the lower regions of the brain to the thalamus and cerebral cortex. Likewise, in the thalamus, almost all sensory information is relayed both into still deeper structures of the thalamus and at the same time to discrete regions of the cerebral cortex.

Convergence of Signals

Convergence means signals from multiple inputs uniting to excite a single neuron. Figure 46–12*A* shows *convergence from a single source*. That is, multiple terminals from a single incoming fiber tract terminate on the same neuron. The importance of this is that neurons are almost never excited by an action potential from a single input terminal. But action potentials converging on the neuron from multiple terminals provide enough spatial summation to bring the neuron to the threshold required for discharge.

Convergence can also result from input signals (excitatory or inhibitory) *from multiple sources*, as shown in Figure 46–12*B*. For instance, the interneurons of the spinal cord receive converging signals from (1) peripheral nerve fibers entering the cord, (2) propriospinal fibers passing from one segment of the cord to another, (3) corticospinal fibers from the cerebral cortex, and (4) several other long pathways descending from the brain into the spinal cord. Then the signals from the interneurons converge on the anterior motor neurons to control muscle function.

Such convergence allows *summation* of information from different sources, and the resulting response is a summated effect of all the different types of information. Convergence is one of the important means by which the central nervous system correlates, summates, and sorts different types of information.

Neuronal Circuit with Both Excitatory and Inhibitory Output Signals

Sometimes an incoming signal to a neuronal pool causes an output excitatory signal going in one direction and at the same time an inhibitory signal going elsewhere. For instance, at the same time that an excitatory signal is transmitted by one set of neurons in the spinal cord to cause forward movement of a leg, an inhibitory signal is transmitted through a separate set of neurons to inhibit the muscles on the back of the leg so that they will not oppose the forward movement. This type of circuit is characteristic for controlling all antagonistic pairs of muscles, and it is called the *reciprocal inhibition circuit*.

Figure 46–13 shows the means by which the inhibition is achieved. The input fiber directly excites the excitatory output pathway, but it stimulates an intermediate *inhibitory neuron* (neuron 2), which secretes a different type of transmitter substance to inhibit the second output pathway from the pool. This type of circuit is also important in preventing overactivity in many parts of the brain.

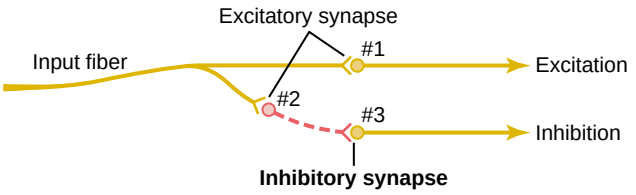


Figure 46–13

Inhibitory circuit. Neuron 2 is an inhibitory neuron.

Prolongation of a Signal by a Neuronal Pool—"Afterdischarge"

Thus far, we have considered signals that are merely relayed through neuronal pools. However, in many instances, a signal entering a pool causes a prolonged output discharge, called *afterdischarge*, lasting a few milliseconds to as long as many minutes after the incoming signal is over. The most important mechanisms by which afterdischarge occurs are the following.

Synaptic Afterdischarge. When excitatory synapses discharge on the surfaces of dendrites or soma of a neuron, a postsynaptic electrical potential develops in the neuron and lasts for many milliseconds, especially when some of the long-acting synaptic transmitter substances are involved. As long as this potential lasts, it can continue to excite the neuron, causing it to transmit a continuous train of output impulses, as was explained in Chapter 45. Thus, as a result of this synaptic "afterdischarge" mechanism alone, it is possible for a single instantaneous input signal to cause a sustained signal output (a series of repetitive discharges) lasting for many milliseconds.

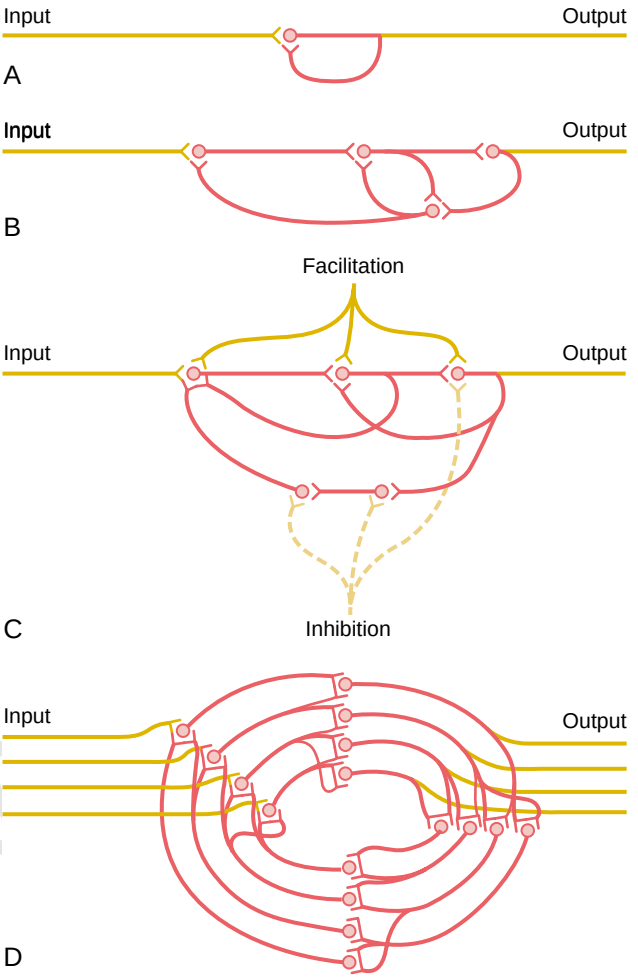


Figure 46–14

Reverberatory circuits of increasing complexity.

Reverberatory (Oscillatory) Circuit as a Cause of Signal Prolongation. One of the most important of all circuits in the entire nervous system is the *reverberatory*, or *oscillatory*, *circuit.* Such circuits are caused by positive feedback within the neuronal circuit that feeds back to re-excite the input of the same circuit. Consequently, once stimulated, the circuit may discharge repetitively for a long time.

Several possible varieties of reverberatory circuits are shown in Figure 46–14. The simplest, shown in Figure 46–14*A,* involves only a single neuron. In this case, the output neuron simply sends a collateral nerve fiber back to its own dendrites or soma to restimulate itself. Although this type of circuit probably is not an important one, theoretically, once the neuron discharges, the feedback stimuli could keep the neuron discharging for a protracted time thereafter.

Figure 46–14*B* shows a few additional neurons in the feedback circuit, which causes a longer delay between initial discharge and the feedback signal. Figure 46–14*C* shows a still more complex system in which both facilitatory and inhibitory fibers impinge on the reverberating circuit. A facilitatory signal enhances the intensity and frequency of reverberation, whereas an inhibitory signal depresses or stops the reverberation.

Figure 46–14*D* shows that most reverberating pathways are constituted of many parallel fibers. At each cell station, the terminal fibrils spread widely. In such a system, the total reverberating signal can be either weak or strong, depending on how many parallel nerve fibers are momentarily involved in the reverberation.

Characteristics of Signal Prolongation from a Reverberatory Circuit. Figure 46–15 shows output signals from a typical reverberatory circuit. The input stimulus may last only 1 millisecond or so, and yet the output can last for many milliseconds or even minutes. The figure demonstrates that the intensity of the output signal usually increases to a high value early in reverberation and then decreases to a critical point, at which it suddenly ceases entirely. The cause of this sudden cessation of reverberation is fatigue of synaptic junctions in the circuit. Fatigue beyond a certain critical level lowers the stimulation of the next neuron in the circuit below threshold level so that the circuit feedback is suddenly broken.

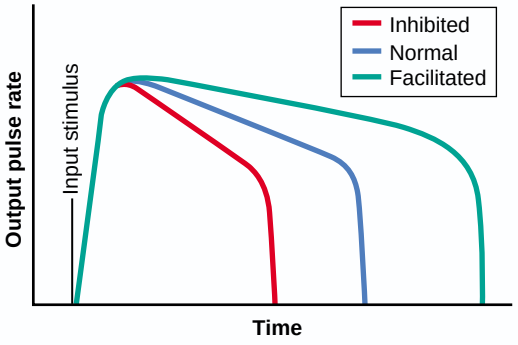


Figure 46–15

Typical pattern of the output signal from a reverberatory circuit after a single input stimulus, showing the effects of facilitation and inhibition.

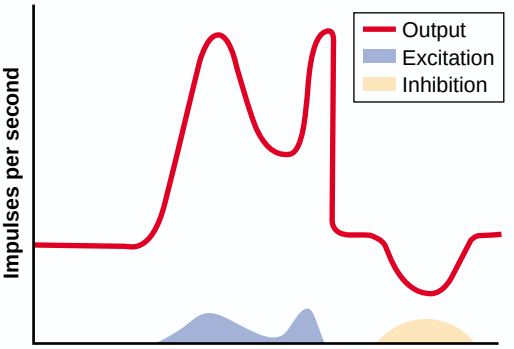


Figure 46–16

Continuous output from either a reverberating circuit or a pool of intrinsically discharging neurons. This figure also shows the effect of excitatory or inhibitory input signals.

The duration of the total signal before cessation can also be controlled by signals from other parts of the brain that inhibit or facilitate the circuit. Almost these exact patterns of output signals are recorded from the motor nerves exciting a muscle involved in a flexor reflex after pain stimulation of the foot (as shown later in Figure 46–18).

Continuous Signal Output from Some Neuronal Circuits

Some neuronal circuits emit output signals continuously, even without excitatory input signals. At least two mechanisms can cause this effect: (1) continuous intrinsic neuronal discharge and (2) continuous reverberatory signals.

Continuous Discharge Caused by Intrinsic Neuronal Excitability. Neurons, like other excitable tissues, discharge repetitively if their level of excitatory membrane potential rises above a certain threshold level. The membrane potentials of many neurons even normally are high enough to cause them to emit impulses continually. This occurs especially in many of the neurons of the cerebellum, as well as in most of the interneurons of the spinal cord. The rates at which these cells emit impulses can be increased by excitatory signals or decreased by inhibitory signals; inhibitory signals often can decrease the rate of firing to zero.

Continuous Signals Emitted from Reverberating Circuits as a Means for Transmitting Information. A reverberating circuit that does not fatigue enough to stop reverberation is a source of continuous impulses. And excitatory impulses entering the reverberating pool can increase the output signal, whereas inhibition can decrease or even extinguish the signal.

Figure 46–16 shows a continuous output signal from a pool of neurons. The pool may be emitting impulses because of intrinsic neuronal excitability or as a result of reverberation. Note that an excitatory input signal greatly increases the output signal, whereas an inhibitory input signal greatly decreases the output. Those students who are familiar with radio transmitters will recognize this to be a *carrier wave* type of information transmission. That is, the excitatory and inhibitory control signals are not the *cause* of the output signal, but they do *control* its changing level of intensity. Note that this carrier wave system allows a *decrease* in signal intensity as well as an increase, whereas up to this point, the types of information transmission we have discussed have been mainly positive information rather than negative information. This type of information transmission is used by the autonomic nervous system to control such functions as vascular tone, gut tone, degree of constriction of the iris in the eye, and heart rate. That is, the nerve excitatory signal to each of these can be either increased or decreased by accessory input signals into the reverberating neuronal pathway.

Rhythmical Signal Output

Many neuronal circuits emit rhythmical output signals—for instance, a rhythmical respiratory signal originates in the respiratory centers of the medulla and pons. This respiratory rhythmical signal continues throughout life. Other rhythmical signals, such as those that cause scratching movements by the hind leg of a dog or the walking movements of any animal, require input stimuli into the respective circuits to initiate the rhythmical signals.

All or almost all rhythmical signals that have been studied experimentally have been found to result from reverberating circuits or a succession of sequential reverberating circuits that feed excitatory or inhibitory signals in a circular pathway from one neuronal pool to the next.

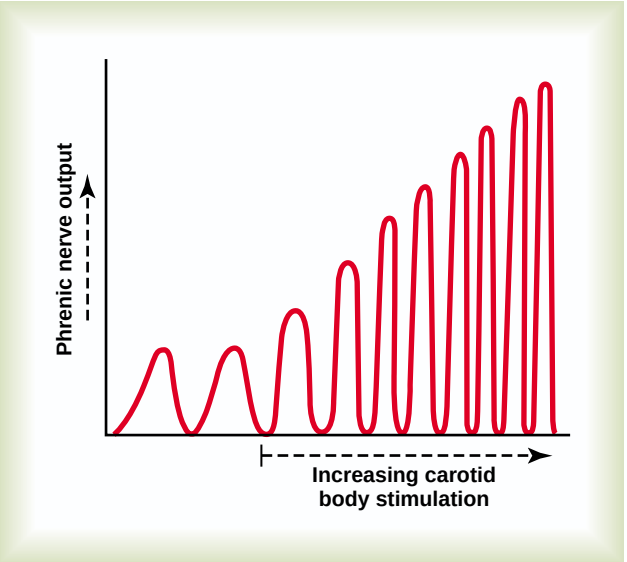


Figure 46–17

The rhythmical output of summated nerve impulses from the respiratory center, showing that progressively increasing stimulation of the carotid body increases both the intensity and the frequency of the phrenic nerve signal to the diaphragm to increase respiration.

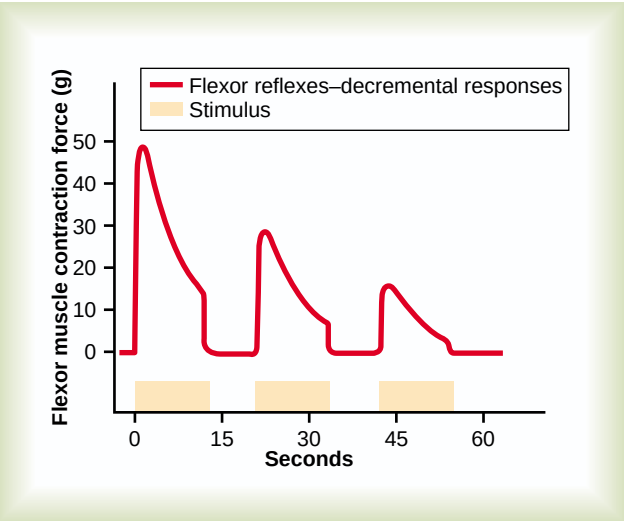


Figure 46–18

Successive flexor reflexes showing fatigue of conduction through the reflex pathway.

Excitatory or inhibitory signals can also increase or decrease the amplitude of the rhythmical signal output. Figure 46–17, for instance, shows changes in the respiratory signal output in the phrenic nerve. When the carotid body is stimulated by arterial oxygen deficiency, both the frequency and the amplitude of the respiratory rhythmical output signal increase progressively.

Instability and Stability of Neuronal Circuits

Almost every part of the brain connects either directly or indirectly with every other part, and this creates a serious problem. If the first part excites the second, the second the third, the third the fourth, and so on until finally the signal re-excites the first part, it is clear that an excitatory signal entering any part of the brain would set off a continuous cycle of re-excitation of all parts. If this should occur, the brain would be inundated by a mass of uncontrolled reverberating signals—signals that would be transmitting no information but, nevertheless, would be consuming the circuits of the brain so that none of the informational signals could be transmitted. Such an effect occurs in widespread areas of the brain during *epileptic seizures*. How does the central nervous system prevent this from happening all the time? The answer lies mainly in two basic mechanisms that function throughout the central nervous system: (1) inhibitory circuits and (2) fatigue of synapses.

Inhibitory Circuits as a Mechanism for Stabilizing Nervous System Function

Two types of inhibitory circuits in widespread areas of the brain help prevent excessive spread of signals: (1) inhibitory feedback circuits that return from the termini of pathways back to the initial excitatory neurons of the same pathways—these circuits occur in virtually all sensory nervous pathways and inhibit either the input neurons or the intermediate neurons in the sensory pathway when the termini become overly excited; and (2) some neuronal pools that exert gross inhibitory control over widespread areas of the brain—for instance, many of the basal ganglia exert inhibitory influences throughout the muscle control system.

Synaptic Fatigue as a Means for Stabilizing the Nervous System

Synaptic fatigue means simply that synaptic transmission becomes progressively weaker the more prolonged and more intense the period of excitation. Figure 46–18 shows three successive records of a flexor reflex elicited in an animal caused by inflicting pain in the footpad of the paw. Note in each record that the strength of contraction progressively "decrements"—that is, its strength diminishes; much of this effect is caused by *fatigue* of synapses in the flexor reflex circuit. Furthermore, the shorter the interval between successive flexor reflexes, the less the intensity of the subsequent reflex response.

Automatic Short-Term Adjustment of Pathway Sensitivity by the Fatigue Mechanism. Now let us apply this phenomenon of fatigue to other pathways in the brain. Those that

are overused usually become fatigued, so that their sensitivities decrease. Conversely, those that are underused become rested, and their sensitivities increase. Thus, fatigue and recovery from fatigue constitute an important short-term means of moderating the sensitivities of the different nervous system circuits. These help to keep the circuits operating in a range of sensitivity that allows effective function.

Long-Term Changes in Synaptic Sensitivity Caused by Automatic Downregulation or Upregulation of Synaptic Receptors. The long-term sensitivities of synapses can be changed tremendously by upregulating the number of receptor proteins at the synaptic sites when there is underactivity, and downregulating the receptors when there is overactivity. The mechanism for this is the following: Receptor proteins are being formed constantly by the endoplasmic reticular–Golgi apparatus system and are constantly being inserted into the receptor neuron synaptic membrane. However, when the synapses are overused so that excesses of transmitter substance combine with the receptor proteins, many of these receptors are inactivated and removed from the synaptic membrane.

It is indeed fortunate that upregulation and downregulation of receptors, as well as other control mechanisms for adjusting synaptic sensitivity, continually adjust the sensitivity in each circuit to almost the exact level required for proper function. Think for a moment how serious it would be if the sensitivities of only a few of these circuits were abnormally high; one might then expect almost continual muscle cramps, seizures, psychotic disturbances, hallucinations, mental tension, or other nervous disorders. But fortunately, the automatic controls normally readjust the sensitivities of the circuits back to controllable ranges of reactivity any time the circuits begin to be too active or too depressed.

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