FIFTH EDITION

TEXTBOOK OF MEDICAL PHYSIOLOGY

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one mechanism substituting for the other when the second is missing.

Another important value of the adrenal medullae is the capability of epinephrine and norepinephrine to stimulate structures of the body that are not innervated by direct sympathetic fibers. For instance, the metabolic rate of every cell of the body is increased by these hormones, especially by epinephrine, even though only a small proportion of all the cells in the body are innervated by sympathetic fibers.

RELATIONSHIP OF STIMULUS RATE TO DEGREE OF SYMPATHETIC AND PARASYMPATHETIC EFFECT

A special difference between the autonomic nervous system and the skeletal nervous system is the low frequency of stimulation required for full activation of autonomic effectors. In general, only one impulse every second or so suffices to maintain normal sympathetic or parasympathetic effect, and full activation occurs when the nerve fibers discharge 10 to 30 times per second. This compares with full activation in the skeletal nervous system at about 75 to 200 impulses per second.

SYMPATHETIC AND PARASYMPATHETIC "TONE"

The sympathetic and parasympathetic systems are continually active, and the basal rates of activity are known, respectively, as *sympathetic tone* or *parasympathetic tone*.

The value of tone is that it allows a single nervous system to increase or to decrease the activity of a stimulated organ. For instance, sympathetic tone normally keeps almost all the blood vessels of the body constricted to approximately half their maximum diameter. By increasing the degree of sympathetic stimulation, the vessels can be constricted even more; but, on the other hand, by inhibiting the normal tone, the vessels can be dilated. If it were not for the continual sympathetic tone, the sympathetic system could cause only vasoconstriction, never vasodilatation.

Another interesting example of tone is that of the parasympathetics in the gastrointestinal tract. Surgical removal of the parasympathetic supply to the gut by cutting the vagi can cause serious and prolonged gastric and intestinal "atony," thus illustrating that in normal function the parasympathetic tone to the gut is strong. This tone can be decreased by the brain, thereby inhibiting gastrointestinal motility; on the other hand, it can be increased, then promoting increased gastrointestinal activ

Tone Caused by Basal Secretion of Norepinephrine and Epinephrine by the Adrenal Medullae. The normal resting rate of secretion by the adrenal medullae is about μgm./kg./min. of epinephrine and about μgm./kg./min. of norepinephrine. These quantities are considerable—indeed, enough to maintain the blood pressure almost up to normal value even if all direct sympathetic pathways to the cardiovascular system are removed. Therefore, it is obvious that much of the overall tone of the sympathetic nervous system results from basal secretion of epinephrine and norepinephrine in addition to that which results from direct sympathetic stimulation.

Effect of Loss of Sympathetic or Parasympathetic Tone Following Denervation. Immediately after a sympathetic or parasympathetic nerve is cut, the innervated organ loses its sympathetic or parasympathetic tone. In the case of the blood vessels, for instance, cutting the sympathetic nerves results immediately in almost maximal vasodilatation. However, over several days or weeks, the *intrinsic tone* in the smooth muscle of the vessels increases, usually restoring almost normal vasoconstriction.

Essentially the same events occur in most effector organs whenever sympathetic or parasympathetic tone is lost. That is, intrinsic compensation soon develops to return the function of the organ almost to its normal basal level. However, in the parasympathetic system the compensation sometimes requires many months. For instance, loss of parasympathetic tone to the heart increases the heart rate from 90 to 160 beats per minute in a dog, and this will still be about 120 six months later.

DENERVATION SUPERSENSITIVITY OF SYMPATHETIC AND PARASYMPATHETIC ORGANS FOLLOWING DENERVATION

During the first week or so after a sympathetic or parasympathetic nerve is destroyed, the innervated organ becomes more and more sensitive to injected norepinephrine or acetylcholine, respectively. This effect is illustrated in Figure 57–4; the blood flow in the forearm before removal of the sympathetics was 200 ml. per minute, and a test dose of norepinephrine caused only a slight depression in flow. Then the stellate ganglion was removed, and normal sympathetic tone was lost. At first, the blood flow rose marked

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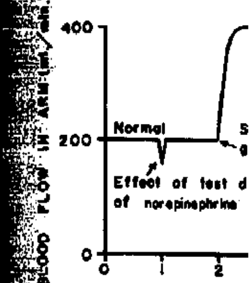

Figure 57–4. Effect of s the arm, and the effect c before and after sympathe the vasculature to norepi

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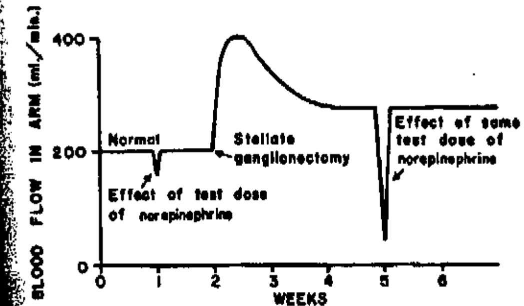

igure 57–4. Effect of sympathectomy on blood flow in he arm, and the effect of a test dose of norepinephrine efore and after sympathectomy, showing sensitization of he vasculature to norepinephrine.

ecause of the lost vascular tone, but over a period of ys to weeks the blood flow returned almost to rmal because of progressive increase in intrinsic ne of the vascular musculature itself, thus compen- ting for the loss of sympathetic tone. Another test se of norepinephrine was then administered and blood flow decreased much more than before, strating that the blood vessels had become about to four times as responsive to norepinephrine as viously. This phenomenon is called *denervation ersensitivity.* It occurs in both sympathetic and asympathetic organs and to a far greater extent in e organs than in others, often increasing the re- se as much as 10-fold.

Mechansim of Denervation Supersensitiv- The precise cause of denervation supersensitiv- is not known, although several suggestions have n made. In the case of the sympathetic nervous em, it is supposed that destruction of the nerve ings prevents removal of norepinephrine or ephrine by the process of re-uptake into the e endings. Likewise, the monamine oxidase that und in the nerve endings is not available to help roy the norepinephrine or epinephrine. There- these two hormones can act strongly and for onged periods of time on the receptor organs. the case of acetylcholine supersensitivity, it is ible that loss of cholinesterase, particularly that h is attached to the nerve endings themselves, es part of the supersensitivity. On the other , it is also possible that some functional system e receptor cells themselves increases in activity n the cells are not continually bombarded by e stimuli.

E AUTONOMIC REFLEXES

is mainly by means of autonomic reflexes the autonomic nervous system regulates eral functions. Throughout this text the functions of these reflexes are discussed in detail in relation to individual organs, but, to illustrate their importance, a few are presented here briefly.

Cardiovascular Autonomic Reflexes. Several reflexes in the cardiovascular system help to control the arterial blood pressure, cardiac output, and heart rate. One of these is the *baroreceptor reflex,* which was described in Chapter 22 along with other cardiovascular reflexes. Briefly, stretch receptors called *baroreceptors* are located in the walls of the major arteries, including the carotid arteries and the aorta. When these become stretched by high pressure, signals are transmitted to the brain stem, where they inhibit the sympathetic centers. This results in decreased sympathetic impulses to the heart and blood vessels, which allows the arterial pressure to fall back toward normal.

The Gastrointestinal Autonomic Reflexes. The uppermost part of the gastrointestinal tract and the rectum are controlled principally by autonomic reflexes. For instance, the smell of appetizing food initiates signals from the nose to the vagal, glossopharyngeal, and salivary nuclei of the brain stem. These in turn transmit signals through the parasympathetic nerves to the secretory glands of the mouth and stomach, causing secretion of digestive juices even before food enters the mouth. And when fecal matter fills the rectum at the other end of the alimentary canal, sensory impulses initiated by stretching the rectum are sent to the sacral portion of the spinal cord, and a reflex signal is retransmitted through the parasympathetics to the distal parts of the colon; these result in strong peristaltic contractions that empty the bowel.

Other Autonomic Reflexes. Emptying of the bladder is also controlled in the same way as emptying of the rectum; stretching the bladder sends impulses to the sacral cord, and this in turn causes contraction of the bladder as well as relaxation of the urinary sphincters, thereby promoting micturition.

Also important are the sexual reflexes which are initiated both by psychic stimuli from the brain and stimuli from the sexual organs. Impulses from these sources converge on the sacral cord and, in the male, result, first, in erection, mainly a parasympathetic function, and then in ejaculation, a sympathetic function.

Other autonomic reflexes include reflex contributions to the regulation of pancreatic secretion, gallbladder emptying, urinary excretion,

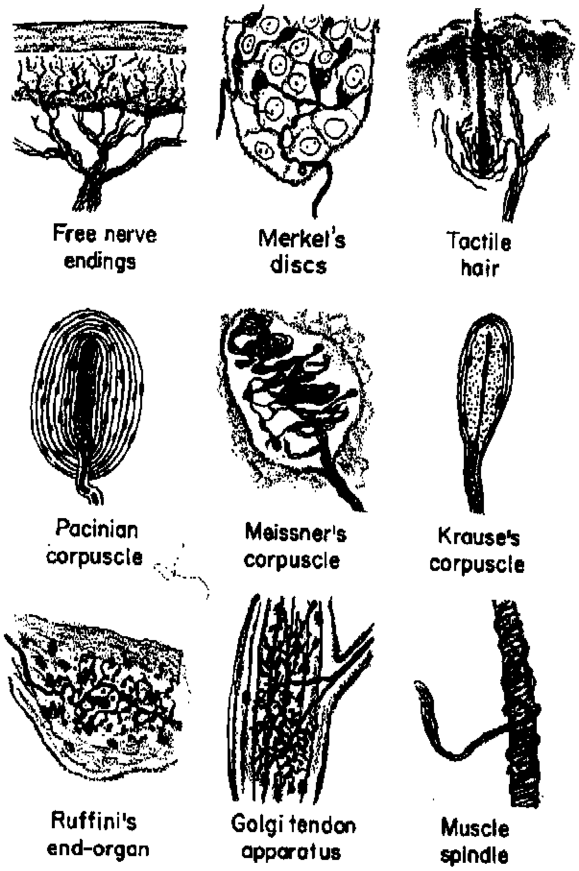

Figure 48–1. Several types of somatic sensory nerve endings. (Modified from Ramon y Cajal: Histology. William Wood and Co.)

TRANSDUCTION OF SENSORY STIMULI INTO NERVE IMPULSES

LOCAL CURRENTS AT NERVE ENDINGS—RECEPTOR POTENTIALS

All sensory receptors studied thus far have one feature in common. Whatever the type of stimulus that excites the ending, it first causes a local potential called a *receptor potential* in the neighborhood of the nerve endings, and it is *local flow of current* caused by the receptor potential that in turn excites action potentials in the nerve fiber.

There are two different ways in which receptor potentials can be elicited. One of these is to deform or chemically alter the terminal nerve ending itself. This causes ions to diffuse through the nerve membrane, thereby setting up the receptor potential.

The second method for causing receptor po- tentials involves specialized receptor cells that lie adjacent to the nerve endings. For instance, when sound enters the cochlea of the ear, specialized *receptor cells* called *hair cells* that lie on the basilar membrane develop local po- tentials which are receptor potentials that stimulate the terminal nerve fibrils entwining the hair cells.

(Some physiologists prefer to use the term *generator potential* to designate the receptor poten- tials elicited in terminal nerve endings because the nerve fibers themselves actually "generate" the po- tentials, and they reserve the term "receptor poten- tials" only for those potentials that arise in specialized receptor cells of non-nervous tissue ori- gin such as taste cells, hair cells of the ear, and so forth. However, because both of these potentials subserve the same function and because of the con- fusion that has developed by use of two separate terms, it is probably best to use the single term "re- ceptor potentials" as we shall do here.)

The Receptor Potential of the Pacinian Corpuscle. The pacinian corpuscle is a ver[y] large and easily dissected sensory receptor. Fo[r] this reason, one can study in detail th[e] mechanism by which tactile stimuli excite it an[d] by which it causes action potentials in the sen- sory fiber leading from it. Note in Figure 48- that the pacinian corpuscle has a central no[n]- myelinated tip of a nerve fiber extending through its core. Surrounding this fiber a[re] many concentric capsule layers so that com- pression on the outside of the corpuscle tends t[o] elongate, shorten, indent, or otherwise defor[m] the central core of the fiber, depending on ho[w] the compression is applied. The deformatio[n] causes a sudden change in membrane potential as illustrated in Figure 48–2. This perhaps re- sults from stretching the nerve fiber membrane, thus increasing its permeability and allowin[g]

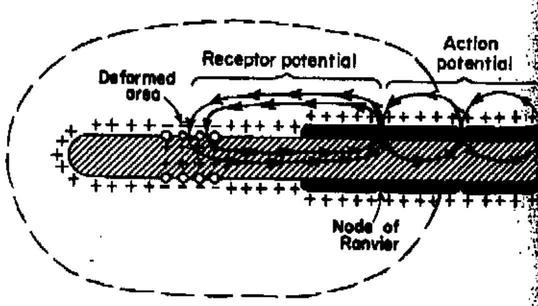

Figure 48–2. Excitation of a sensory nerve fiber by a generator potential produced in a pacinian corpuscle. (Modified from Loewenstein: Ann. N.Y. Acad. Sci. 94:510, 1961.)

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positively charged sodium ions to leak to the interior of the fiber. This change in local potential causes a local circuit of current flow that spreads along the nerve fiber to its myelinated portion. At the first node of Ranvier, which itself lies inside the capsule of the pacinian corpuscle, the local current flow initiates action potentials in the nerve fiber. That is, the current flow through the node depolarizes it, and this then sets off a typical saltatory transmission of an action potential along the nerve fiber toward the central nervous system, as was explained in Chapter 10.

Electrotonic Nature of the Receptor Potential. Note especially that the receptor potential has a different electrical character from the action potential. It will be recalled from Chapter 10 and also from Chapter 46 that the action potential is a self-regenerative cyclic event that begins with a resting negative potential, then changes to a positive potential and finally returns back to a negative potential. On the other hand, the receptor potential is an "electrotonic" potential that causes "tonic" flow of current without proceeding through the regenerative events of an action potential. It is a local potential just as are the end-plate potential of muscle fibers and the postsynaptic potential of neurons. If the receptor potential is great enough, it will elicit one or more action potentials at the first node of Ranvier. On the other hand, if the potential does not reach *threshold* level for excitation of an action potential, it will simply exist locally and will spread only a short distance along the fiber; the spreading will be by the process of electrotonic conduction, not by means of a self-regenerative action potential.

Relationship Between Receptor Potential and Stimulus Strength. Figure 48–3 illustrates the effect on the amplitude of the receptor potential caused by progressively stronger stimuli applied to the central core of the pacinian corpuscle. Note that the amplitude increases rapidly at first but then progressively less rapidly at high stimulus strengths. The maximum amplitude that can be achieved by receptor potentials is around 100 millivolts. That is, a receptor potential can have almost as high a voltage as an action potential.

Receptor Potentials Recorded from Other Sensory Receptors. Receptor potentials have been recorded from many other sensory receptors, including most notably the muscle spindles, the hair cells of the ear, and the rods and cones of the eyes and many others. In all of these, the amplitude of the potential increases as the strength of stimulus increases, but the additional response usually becomes progressively less as the strength of stimulus becomes great.

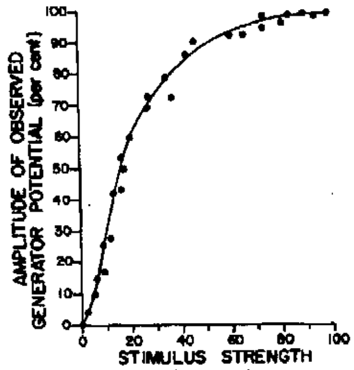

Figure 48–3. Relationship of amplitude of receptor (generator) potential to strength of a stimulus applied to a pacinian corpuscle. (From Loewenstein: *Ann. N.Y. Acad. Sci.*, 94:510, 1961.)

Yet, the mechanism for causing the receptor potential is not the same in different receptors. For instance, in the rods and cones of the eye changes in certain intracellular chemicals caused by exposure to light alter the membrane potential, resulting in the receptor potential. In this case, the basic mechanism eliciting the receptor potential is a chemical one in contrast to mechanical deformation that causes the receptor potential in the pacinian corpuscle. In the case of thermal receptors, it is believed that changes in rates of chemical reaction at or near the membrane alter the membrane potential and thereby create a receptor potential. In the case of the hair cells of the ear, bending of cilia protruding from the hairs probably causes the receptor potentials. Thus, the mechanisms for eliciting receptor potentials are individualized for each type of receptor.

Relationship of Amplitude of Receptor Potential to Nerve Impulse Rate. Referring once again to Figure 48–2, we see that the receptor potential generated in the core of the pacinian corpuscle causes a local circuit of current flow through the first node of Ranvier. When an action potential occurs at the node, this does not affect the receptor potential being emitted by the core of the pacinian corpuscle. Instead, the core continues to emit its current as long as an effective mechanical stimulus is applied. As a result, when the node of Ranvier repolarizes after its first action potential is over, it discharges once again, and action potentials continue as long as the receptor potential persists, which, in the case of the pacinian corpuscle, is only a few thousandths or hundredths of a second.

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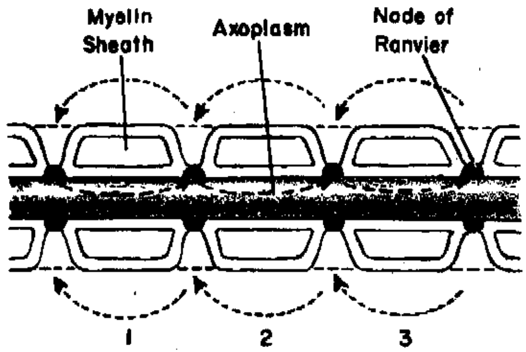

Figure 10–14. Saltatory conduction along a myelinated axon.

VELOCITY OF CONDUCTION IN NERVE FIBERS

The velocity of conduction in nerve fibers varies from as little as 0.5 meter per second in very small unmyelinated fibers up to as high as 130 meters per second (the length of a football field) in very large myelinated fibers. The velocity increases approximately with the fiber diameter in myelinated nerve fibers and approximately with the square root of fiber diameter in unmyelinated fibers.

EXCITATION—THE PROCESS OF ELICITING THE ACTION POTENTIAL

Chemical Stimulation. Basically, any factor that causes sodium ions to begin to diffuse inward through the membrane in sufficient numbers will set off the automatic, regenerative "activation" mechanism, noted earlier in the chapter, that eventuates in the action potential. Thus, certain chemicals can stimulate a nerve fiber by increasing the membrane permeability. Such chemicals include acids, bases, almost any salt solution of very strong concentration, and, most importantly, the substance *acetylcholine*. Many nerve fibers, when stimulated, secrete acetylcholine at their endings where they synapse with other neurons or where they end on muscle fibers. The acetylcholine in turn stimulates the successive neuron or muscle fiber. This is discussed in much greater detail in Chapter 12, and it is one of the most important means by which nerve and muscle fibers are stimulated. Likewise, *norepinephrine* secreted by sympathetic nerve endings can stimulate cardiac muscle fibers and some smooth muscle fibers, and still other hormonal substances can stimulate successive neurons in the central nervous system.

Mechanical Stimulation. Crushing, pinching, or pricking a nerve fiber can cause a sudden surge of sodium influx and, for obvious reasons, can elicit an action potential. Even slight pressure on some specialized nerve endings can stimulate these; this will be discussed in Chapter 48 in relation to sensory perception.

Electrical Stimulation. Electrical stimulation also can initiate an action potential. An electrical charge artificially induced across the membrane causes excess flow of ions through the membrane; this in turn can initiate an action potential. However, not all methods of applying electrical stimuli result in excitation, and, since this is the usual means by which nerve fibers are excited when they are studied in the laboratory, the process of electrical excitation deserves more detailed comment.

Cathodal versus Anodal Currents. Figure 10–15 illustrates a battery connected to two electrodes on the surface of a nerve fiber. At the cathode or negative electrode, the potential outside the membrane is negative with respect to that on the inside, and the current that flows outward through the membrane at this point is called *cathodal current*. At the anode, the electrode is positive with respect to the potential immediately inside the membrane, and the inward current flow at this point is called *anodal current*. *A cathodal current excites the fiber whereas an anodal current actually makes the fiber more resistant to excitation than normal.* Though the cause of this difference between the two types of current cannot be explained completely, it is known that the normal impermeability of the membrane to sodium results partially from the high resting membrane potential across the membrane, and any condition that lessens this potential causes the membrane to become progressively more permeable to sodium. Obviously, at the cathode the applied voltage is opposite to the resting potential of the membrane, and this reduces the net potential. As a result, the membrane becomes far more permeable than usual to sodium followed by subsequent development of an action potential.

On the other hand, at the anode, the applied potential actually enhances the membrane potential. This makes the membrane less permeable to sodium than ever, resulting in increased resistance of the membrane to stimulation by other means.

Threshold for Excitation and "Acute Subthreshold Potential." A very weak cathodal potential cannot excite the fiber. But, when this potential is progressively increased, there comes a point at which excitation takes place. Figure 10–16 illustrates the effects of successively applied cathodal stimuli of progressing strength. A very weak stimulus at point A causes the membrane potential to change from −85 to −80 millivolts, but this is not a sufficient change for the automatic regenerative processes of the action potential to develop. At point B the stimulus is greater, but, here again, the intensity still is not enough to set off the automatic action potential. Nevertheless, the membrane voltage is disturbed for the cell stroma are ingested by the reticuloendothelial cells of the spleen.

Reticuloendothelial Cells of the Spleen. The pulp of the spleen contains many large phagocytic reticuloendothelial cells, and the venous sinuses are lined with similar cells. These cells act as a cleansing system for the blood, similar to that in the venous sinuses of the liver. When the blood is invaded by infectious agents, the reticuloendothelial cells of the spleen rapidly remove debris, bacteria, parasites, etc. Also, in many infectious processes the spleen enlarges in the same manner that lymph glands enlarge and performs its cleansing function even more adequately.

Much of the spleen is filled with *white pulp,* which is in reality a large quantity of lymphocytes and plasma cells. These function in exactly the same way in the spleen as in the lymph glands to cause either humoral or lymphocytic immunity against toxins, bacteria, and so forth, as described in Chapter 7.

The Spleen as a Hemopoietic Organ. During fetal life, the splenic pulp produces blood cells in exactly the same manner that the red bone marrow in the adult produces cells. As the normal fetus approaches birth, the spleen normally loses this ability to produce cells, but, in some diseases, the spleen continues to produce cells even after birth. For instance, in the disease *erythroblastosis fetalis,* which results from excessive destruction of red blood cells by abnormal antibodies in the plasma, as discussed in Chapter 5, the fetus must produce 10 or more times as many red blood cells as normally. As a result, the hemopoietic function of the spleen persists for several weeks after birth.

CIRCULATION IN THE SKIN

PHYSIOLOGIC ANATOMY OF THE CUTANEOUS CIRCULATION

Circulation through the skin subserves two major functions: first, *nutrition of the skin tissues* and, second, *conduction of heat* from the internal structures of the body to the skin so that the heat can be removed from the body. To perform these two functions the circulatory apparatus of the skin is characterized by two major types of vessels, illustrated diagrammatically in Figure 29–7: (1) the usual nutritive arteries, capillaries, and veins and (2) vascular structures concerned with heating the skin, consisting principally of (a) an extensive *subcutaneous venous plexus,* which holds large quantities of blood that can heat the surface of the skin, and (b) in some skin areas, *arteriovenous anastomoses,* which are large vascular communications directly between the arteries and the venous plexuses. The walls of these anastomoses have strong muscular coats inner-

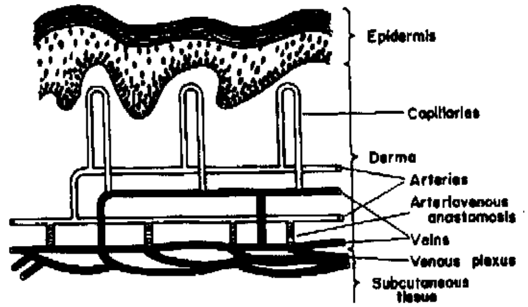

Figure 29–7. Diagrammatic representation of the skin circulation.

vated by sympathetic vasoconstrictor nerve fibers that secrete norepinephrine. When constricted, they reduce the flow of blood into the venous plexuses to almost nothing; or when maximally dilated, they allow extremely rapid flow of warm blood into the plexuses. The arteriovenous anastomoses are found principally in the volar surfaces of the hands and feet, the lips, the nose, and the ears, which are areas of the body most often exposed to maximal cooling.

Rate of Blood Flow Through the Skin. The rate of blood flow through the skin is among the most variable of any part of the body, because the flow required to regulate body temperature changes markedly in response to, first, the rate of metabolic activity of the body, and, second, the temperature of the surroundings. This will be discussed in detail in Chapter 72. The blood flow required for nutrition is slight, so that this plays almost no role in controlling normal skin blood flow. At ordinary skin temperatures, the amount of blood flowing through the skin vessels to subserve heat regulation is about 10 times as much as that needed to supply the nutritive needs of the tissues. But, when the skin is exposed to extreme cold, the blood flow may become so slight that nutrition begins to suffer—even to the extent, for instance, that the fingernails grow considerably more slowly in arctic climates than in temperate climates.

Under ordinary cool conditions the blood flow to the skin is about 0.25 liter/sq. meter of body surface area, or a total of about 400 ml. per minute, in the average adult. On the other hand, when the skin is heated until maximal vasodilatation has resulted, the blood flow can be as much as 7 times this value, or a total of about 2.8 liters per minute, thus illustrating both the extreme variability of skin blood flow and the great drain on cardiac output that can occur

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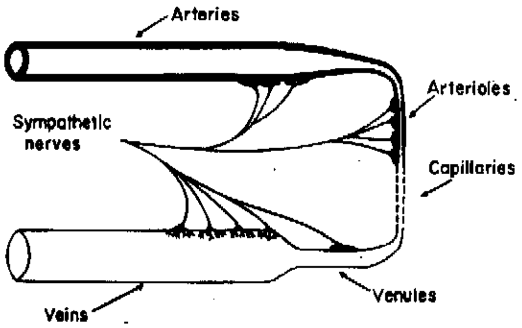

Figure 20–7. Innervation of the systemic circulation.

innervation was discussed in Chapter 13. It will be recalled that sympathetic stimulation markedly increases the activity of the heart, increasing the heart rate and enhancing its strength of pumping.

Parasympathetic Control of the Circulation—Control of the Heart. Though the parasympathetic nervous system is exceedingly important for many other autonomic functions of the body, it plays only a minor role in regulation of the circulation. Its only really important effect is its control of heart rate. It also has a slight influence on the control of cardiac contractility; however, this effect is far overshadowed by the sympathetic nervous system control of contractility. Parasympathetic nerves pass to the heart in the vagus nerve, as illustrated in Figure 20–6.

The effects of parasympathetic stimulation on heart function were discussed in detail in Chapter 13. Principally, parasympathetic stimulation causes a marked *decrease* in heart rate and slight decrease in contractility.

The Sympathetic Vasoconstrictor System and Its Control by the Central Nervous System

The sympathetic nerves carry both vasoconstrictor and vasodilator fibers, but by far the most important of these are the *sympathetic vasoconstrictor* fibers. Sympathetic vasoconstrictor fibers are distributed to essentially all segments of the circulation. However, this distribution is greater in some tissues than in others. It is rather poor in both skeletal and cardiac muscle and in the brain, while it is powerful in the kidneys, the gut, the spleen, and the skin.

The Vasomotor Center and Its Control of the Vasoconstrictor System—Vasomotor Tone. Located bilaterally in the reticular substance of the lower third of the pons and upper two thirds of the medulla, as illustrated in Figure 20–8, is an area called the *vasomotor center.* This center transmits impulses downward through the cord and thence through the vasoconstrictor fibers to all the blood vessels of the body.

The upper and lateral portions of the vasomotor center are *tonically active.* That is, they have an inherent tendency to transmit nerve impulses all the time, thereby maintaining even normally a slow rate of firing in essentially all vasoconstrictor nerve fibers of the body at a rate of about one-half to two impulses per second. This continual firing is called *sympathetic vasoconstrictor tone.* These impulses maintain a partial state of contraction in the blood vessels, a state called *vasomotor tone.*

The brain stem can be severed above the lower third of the pons without significantly changing the normal activity of the vasomotor center. This center remains tonically active and continues to transmit approximately normal numbers of impulses to the sympathetic vasoconstrictor fibers throughout the body.

Figure 20–9 demonstrates the significance of vasoconstrictor tone. In the experiment of this figure, total spinal anesthesia was administered to an animal, which completely blocked transmission of nerve impulses from the cent

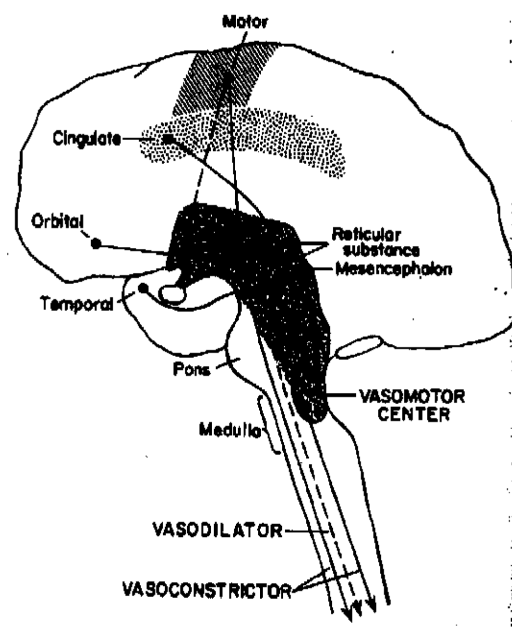

Figure 20–8. Areas of the brain that play important ro in the nervous regulation of the circulation.

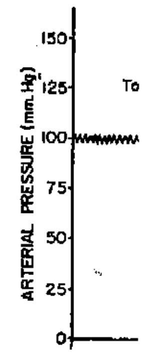

Figure 20–9.
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Control of Center. At center is con striction, it a al portions excitatory ir nerve fibers and contract the vasomoto