

Introduction The nervous system coordinates the activities of many other organ systems. The initiation of an action potential is, therefore, an "all-or-none" event; it is generated completely or not at all. Action Potential Gating Mechanisms. The depolarizing and repolarizing phases of the action potential can be explained by relative changes in membrane conductance (permeability) to sodium and potassium.

During the rising phase, the nerve cell membrane becomes more permeable to sodium; as a consequence, the membrane potential begins to shift more toward the equilibrium potential for sodium.

However, before the membrane potential reaches E_{Na} , sodium permeability begins to decrease and potassium permeability increases. This change in membrane conductance again drives the membrane potential toward E_K , accounting for repolarization of the membrane. The action potential can also be viewed in terms of the flow of charged ions through selective ion channels. These voltage-gated channels are closed when the neuron is at rest. When the membrane is depolarized, these channels begin to open. The Na^+ channel quickly opens its activation gate and allows Na^+ ions to flow into the cell. The influx of positively charged Na^+ ions causes the membrane to depolarize. In fact, the membrane potential actually reverses, with the inside becoming positive; this is called the overshoot. In the initial stage of the action potential, more Na^+ than K^+ channels are opened because the K^+ channels open more slowly in response to depolarization. This increase in Na^+ permeability compared to that of K^+ causes the membrane potential to move toward the equilibrium potential for Na^+ .

Propagation and Speed of the Action Potential. After an action potential is generated, it propagates along the axon toward the axon terminal; it is conducted along the axon with no decrement in amplitude. The mode in which action potentials propagate and the speed with which they are conducted along an axon depend on whether the axon is myelinated. The diameter of the axon also influences the speed of action potential conduction: larger-diameter axons have faster action potential conduction velocities than smaller-diameter axons. In unmyelinated axons, voltage-gated Na^+ and K^+ channels are distributed uniformly along the length of the axonal membrane. An action potential is generated when the axon hillock is depolarized by the passive spread of synaptic potentials along the somatic and dendritic membrane. The hillock acts as a "sink" where Na^+ ions enter the cell. It activates muscles for movement, controls the secretion of hormones from glands, regulates the rate and depth of breathing, and is involved in modulating and regulating a multitude of other physiological processes. We examine the specialized membrane properties of nerve cells that endow them with the ability to produce action potentials, explore the basic mechanisms of synaptic transmission, and discuss aspects of neuronal structure necessary for the maintenance of nerve cell function. This cycle of membrane depolarization, sodium channel activation, sodium ion influx, and membrane depolarization is an example of positive feedback, a regenerative process that results in the explosive activation of many sodium ion channels when the threshold membrane potential is reached. The propagation of action potentials, the release of neurotransmitters, and the activation of receptors constitute the means whereby nerve cells communicate and transmit information to one another and to non-neuronal tissues. The membrane of the initial segment contains a high density of voltage-gated sodium and potassium ion channels. When the membrane of the initial segment is depolarized, voltage-gated sodium channels are opened, permitting an influx of sodium ions. To perform these functions, the nervous system relies on neurons, which are

designed for the rapid transmission of information from one cell to another by conducting electrical impulses and secreting chemical neurotransmitters. The action potential is a transient change in the membrane potential characterized by a gradual depolarization to threshold, a rapid rising phase, an overshoot, and a repolarization phase. The electrical impulses propagate along the length of nerve fiber processes to their terminals, where they initiate a series of events that cause the release of chemical neurotransmitters. If the depolarization of the initial segment does not reach threshold, then not enough sodium channels are activated to initiate the regenerative process. Electrical signals that depend on the passive properties of the neuronal cell membrane spread electrotonically over short distances. The influx of these positively charged ions further depolarizes the membrane, leading to the opening of other voltage-gated sodium channels.

PASSIVE MEMBRANE PROPERTIES, THE ACTION POTENTIAL, AND ELECTRICAL SIGNALING BY NEURONS

Neurons communicate by a combination of electrical and chemical signaling. Generally, information is integrated and transmitted along the processes of a single neuron electrically and then transmitted to a target cell chemically. The activation of these receptors either excites or inhibits the postsynaptic neuron. The repolarization phase is followed by a brief afterhyperpolarization (undershoot) before the membrane potential again reaches resting level. In most neurons, the axon hillock (initial segment) is the trigger zone that generates the action potential. Released neurotransmitters bind with their receptors on the postsynaptic cell membrane. Action potentials depend on a regenerative wave of channel openings and closings in the membrane. Depolarization of the axon hillock to threshold results in the generation and propagation of an action potential.