

Membrane Potentials and Action Potentials

Electrical potentials exist across the membranes of virtually all cells of the body. Some cells, such as nerve and muscle cells, generate rapidly changing electrochemical impulses at their membranes, and these impulses are used to transmit signals along the nerve or muscle membranes. In other types of cells, such as glandular cells, macrophages, and ciliated cells, local changes in membrane potentials also activate many of the cells' functions.

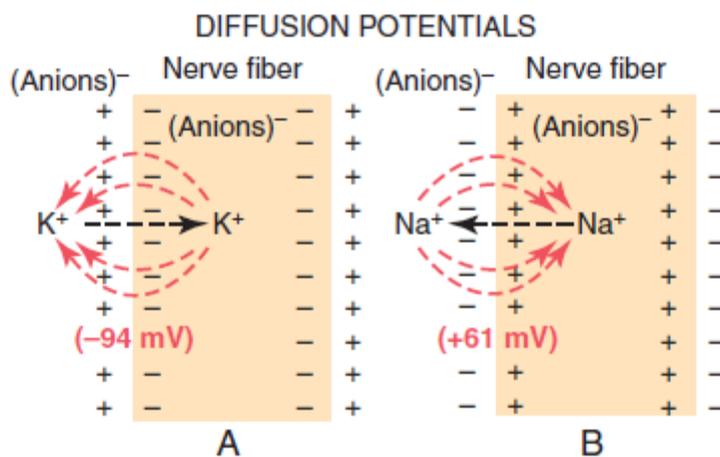


Figure 5-1. **A**, Establishment of a diffusion potential across a nerve fiber membrane, caused by diffusion of potassium ions from inside the cell to outside through a membrane that is selectively permeable only to potassium. **B**, Establishment of a diffusion potential when the nerve fiber membrane is permeable only to sodium ions. Note that the internal membrane potential is negative when potassium ions diffuse and positive when sodium ions diffuse because of opposite concentration gradients of these two ions.

NEURON ACTION POTENTIAL

Nerve signals are transmitted by *action potentials*, which are rapid changes in the membrane potential that spread rapidly along the nerve fiber membrane. Each action potential begins with a sudden change from the normal resting negative membrane potential to a positive potential and ends with an almost equally rapid change back to the

negative potential. To conduct a nerve signal, the action potential moves along the nerve fiber until it comes to the fiber's end.

the changes that occur at the membrane during the action potential, with the transfer of positive charges to the interior of the fiber at its onset and the return of positive charges to the exterior at its end. The lower panel shows graphically the successive changes in membrane potential over a few 10,000ths of a second, illustrating the explosive onset of the action potential and the almost equally rapid recovery.

The successive stages of the action potential are as follows.

Resting Stage. The resting stage is the resting membrane potential before the action potential begins. The membrane is said to be “polarized” during this stage because of the -90 millivolts negative membrane potential that is present.

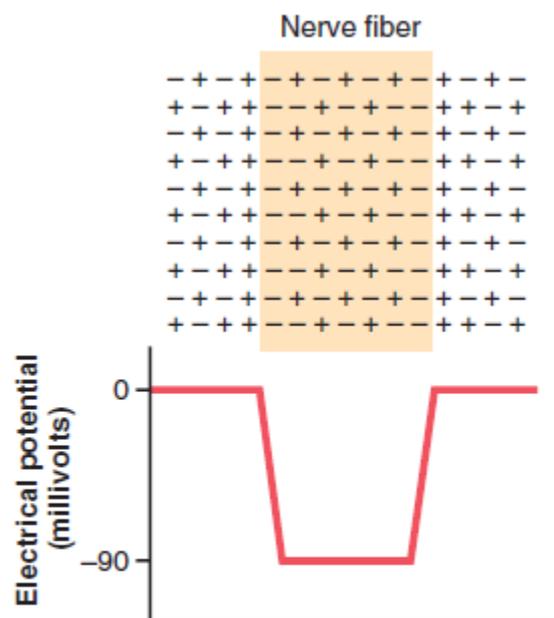
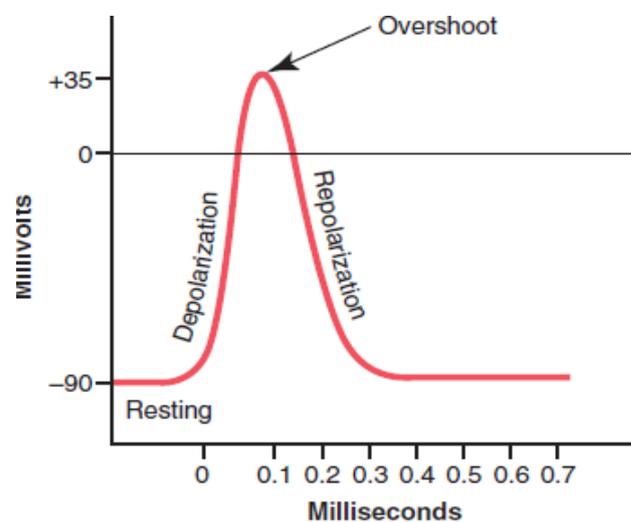


Figure 5-3. Distribution of positively and negatively charged ions in the extracellular fluid surrounding a nerve fiber and in the fluid inside the fiber. Note the alignment of negative charges along the inside surface of the membrane and positive charges along the outside surface. The lower panel displays the abrupt changes in membrane potential that occur at the membranes on the two sides of the fiber.

Depolarization Stage. At this time, the membrane suddenly becomes permeable to sodium ions, allowing tremendous numbers of positively charged sodium ions to diffuse to the interior of the axon. The normal “polarized” state of -90 millivolts is immediately neutralized by the inflowing positively charged sodium ions, with the potential rising rapidly in the positive direction—a process called *depolarization*. In large nerve fibers, the great excess of positive sodium ions moving to the inside causes the membrane potential to actually “overshoot” beyond the zero level and to become somewhat positive. In some smaller fibers, as well as in many central nervous system neurons, the potential merely approaches the zero level and does not overshoot to the positive state.

Repolarization Stage. Within a few 10,000ths of a second after the membrane becomes highly permeable to sodium ions, the sodium channels begin to close and the potassium channels open to a greater degree than normal. Then, rapid diffusion of potassium ions to the exterior re-establishes the normal negative resting membrane potential, which is called *repolarization* of the membrane.



Basically, any factor that causes sodium ions to begin to diffuse inward through the membrane in sufficient numbers can set off automatic regenerative opening of the sodium channels. This automatic regenerative opening can result from *mechanical* disturbance of the membrane, *chemical* effects on the membrane, or passage of *electricity*

through the membrane. All these approaches are used at different points in the body to elicit nerve or muscle action potentials: mechanical pressure to excite sensory nerve endings in the skin, chemical neurotransmitters to transmit signals from one neuron to the next in the brain, and electrical current to transmit signals between successive muscle cells in the heart and intestine.

Inhibition of Excitability—“Stabilizers” and Local Anesthetics

In contrast to the factors that increase nerve excitability, still others, called *membrane-stabilizing factors*, can *decrease excitability*. For instance, *a high extracellular fluid calcium ion concentration* decreases membrane permeability to sodium ions and simultaneously reduces excitability. Therefore, calcium ions are said to be a “stabilizer.”

Local Anesthetics. Among the most important stabilizers are the many substances used clinically as local anesthetics, including *procaine* and *tetracaine*. Most of these substances act directly on the activation gates of the sodium channels, making it much more difficult for these gates to open, thereby reducing membrane excitability.