Of All The Nerve!

Our nervous system is responsible for coordinating all body functions, providing the means to communicate so our bodies can respond and adapt to internal and external conditions and be able to move about as needed. The nervous system is comprised mainly of two types of cells—neurons and neuroglia. Neurons are the “conducting” or “communicating” cells and are quite complex and specialized (there are three types: motor, sensory, and connector). Neuroglia are the supporting cells of the nervous system and they help to supply nutrients, maintain electrical potential, act as barriers, help in the production of spinal fluid, form myelin sheathing on neurons, and digest microorganisms and waste products. While neurons are very busy sending and receiving messages, neuroglia are busy with all the necessary ‘housekeeping’ requirements to keep the nervous system in tip-top shape. This business of communicating is quite a complex one—if we were required to actually think about and perform everything deliberately, under conscious acts of will, there is no doubt we would soon be too distracted to do much other than keep our hearts beating and stomachs churning (fortunately, a part of the nervous system does this automatically for us)!

The human nervous system ties our entire body together into a vast array of one-way streets, so to speak, providing the means to function and respond appropriately to all stimuli, whether internal or external. Neurons, uniquely structured cells, are the one-way streets. Figure 1 illustrates the basic anatomy of the neuron. The axon and dendrites are cytoplasmic processes, or extensions, of the cell body. The axon, enclosed in a membrane of its own (axolemma) conducts impulses away from the cell body to the terminals, and dendrites conduct impulses toward the cell body. The axon can be very short or very long, ends in terminal branches, and is covered in either a myelinated or non-meylinated sheath. In mylenated neurons, Schwann cells are essentially wrapped around the axon, creating gaps called “Nodes
Figure 1: Myelinated and Non-Meylinated Neurons
of Ranvier” (these figure heavily into why this type of neuron can communicate so rapidly, sending impulses at stunning speeds of up to 268mph!). In non-myelinated neurons, transmission speed is significantly slower. Axons are as long as necessary to transmit a message from dendrite to terminal button (which can be up to about a meter long!), thus the mylenated sheath is a necessary adaptation to the neuron in order to speed the process of communicating.

So, how do the neurons actually “talk” or “communicate”? The ‘message’ can be described loosely as an electro-chemical impulse, or a difference in potential that must move from dendrite through cell body, down axon and out the terminal buttons (motor end-plates in the case of fibers connecting directly to muscle tissue receptors)—and the message will only move if the potential is significant enough to exceed the threshold. The cell’s ability to communicate is either “switched on” or “switched off,” but never “on a little bit” or “nearly off.” With the nervous system, its full-on or full-off—thus an impulse is always the same strength; it either occurs, or it doesn’t.

At rest (off, not on, or unstimulated), a difference of potential between the axon’s interior and exterior exists (approximately −70mv). At this time, the membrane is highly permiable to potassium and the axon is polarized (negative inside the cell, positive outside of it). This is a near-equilibrium state for potassium, so there is little leakage of potassium (just enough through a “slow gate” channel to maintain the resting state). In this state, extracellular fluid has a higher concentration of sodium, and intracellular fluid has a higher concentration of potassium. The “fast gates” or channels for potassium and sodium are both closed during this polarized state (recall the sodium-potassium ion pumps of unit three and how they operate. See endnotes). The cell will remained polarized (at resting potential) until it is stimulated to a degree that its
threshold is exceeded—the ability of the cell to respond to stimulation and create an impulse is called *excitability*.

**Figure 2, Plate A**: When an impulse is initiated (enough neurotransmitter has bound to the cell's receptor sites to excite it, or exceed its threshold potential), the neuron begins to flood with sodium ions at the point of initiation (sodium “fast” gates in the cell membrane open in response to the impulse). This reverses the polarity at that point (de-polarizing), so the inside of the membrane is positive and the outside is negative.

**Figure 2, Plate B**: The opening of Sodium gates at the point of impulse causes two events to occur:

1. Potassium “fast” gates open to push potassium out of the cell in that area in order to begin repolarizing (this also keeps the impulse from moving backwards), and...
2. Sodium “fast” gate closes, and Sodium “fast” gates adjacent to those previously opened will open.

This continues along the length of the axon until the impulse reaches the end of the terminal buttons and fires across synaptic clefts to excite other neurons or stimulate actions in muscles or organs.

**Figure 2, Plate C**: After the impulse passes and repolarizing has begun, all fast gates close and the “slow” gates, or Sodium-Potassium Ion pumps restore the cell polarity, returning the cell to its resting potential (negative inside and positive outside). This is known as the refractory period, and during this time, the cell is unable to be excited. This ensures that one and only one impulse passes completely through the cell at a time, permitting only “one-way” communication and prevent impulse “traffic jams.” Multiple synaptic inputs will converge in the cell body and only one impulse moves along the axon.

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The action potential or impulse moves faster in mylenated neurons than non-mylenated. Mylenated sheaths are formed by Schwann cells that wrap around the axon, leaving spots where the axon membrane faces the outer environment directly. These areas are known as the nodes of Ranvier. The action potential in a mylenated neuron is passed from node to node, where the sodium and potassium gating previously described will take place—and the impulse “skips” along the nodes, de-polarizing and re-polarizing the membrane node to node, rather than along the full length of the membrane as in the case of non-mylenated neurons. This allows the impulse to move along the axon much faster, as illustrated in Figure 3.

Figure 3: Mylenated Sheath Impulse Potential Traveling Node to Node

Figure 4: Actual Node of Ranvier, Human Tissue (Red Arrow)
Moving the impulse from one end of a neuron to the other (receptor site to terminal button) seems easy enough with a little help from potassium and sodium “flood” gates and mylenated sheathing…but the impulse must continue its rapid journey. The impulse leaves one cell and ‘leaps’ to other cells across synaptic gaps (the space between the terminal button and the next cell). See Figure 5.

**Figure 5: The Synaptic Cleft**

It is amazing to think that nerve cells do not physically touch one another, but in fact they do not. The terminal button, also called the pre-synaptic terminal, is separated from the next cell by a very thin layer of fluid. Across the gap, or synaptic cleft, from the terminal is the next cell’s receptor site where it will receive neurotransmitter substance, resulting in either localized
depolarization of the membrane (excitatory synapse, which starts the depolarization-
repolarization ripple as the impulse passes through the next cell) or in hyper-polarization
(inhibitory synapse, creating a localized extra-negative polarity in the membrane, inhibiting the
impulse). \(^2\) Terminal buttons may pass neurotransmitter substance to the dendrites and cell
body of other neurons, to receptors (sensory neurons) of effectors (muscles or glands). There
are many types of synapses and many kinds of neurotransmitters—but they are either excitatory
(sodium-potassium permeability) or inhibitory (chlorine-potassium permeability).

Synaptic vesicles in the terminal form and are filled with the neurotransmitter substance.
As the impulse reaches the end of an axon, channels open to permit calcium into the terminal
membrane. Calcium promotes fusion of the vesicles with the cell membrane and they open to
the synaptic cleft, releasing their contents (exocytosis). The neurotransmitter then diffuses
across the cleft, binds with receptor sites, and chemically activates channels in the post-
synaptic cell membrane—sodium gates to prompt depolarization and chlorine-potassium
permeable gates to inhibit sodium entry and permit greater potassium leakage, causing the
inside of the cell to be even more negatively charged.

\(^2\) Assuming the threshold is exceeded.
Bibliography


http://www.kumc.edu/instruction/medicine/anatomy/histoweb/nervous/nervous.htm, University of Kansas Medical Center, Department of Anatomy and Cell Biology, December 2005; Photograph of Nodes of Ranvier.