HOW-TO-DO-IT

How Neurons Work: An Analogy & Demonstration Using a Sparkler & a Frying Pan

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Information in the nervous system is conveyed by impulses called action potentials: large, transient electrochemical changes in a neuron's membrane. Though action potentials are a basic feature of neurons, teachers often have trouble explaining this neurophysiological concept, and students have difficulty understanding it. While easy-to-understand analogies exist to help students conceptualize the heart as a pump or the kidney as a filter, there are fewer examples to provide insight into the workings of the nervous system. This void may be responsible in part for the lack of discussion about the nervous system from elementary school through introductory college courses despite the inherent appeal of understanding the brain and behavior. In this article I present an analogy and demonstration using a sparkler (a wire coated with combustible or explosive material similar to gunpowder) and a frying pan to illustrate how neurons generate and propagate action potentials. Analogies have been used for centuries to help explain scientific concepts, and are effective in helping students integrate new knowledge (Glynn et al., 1995).

Neurons

Neurons or nerve cells are extremely diverse in their size and shape; despite this diversity, most exhibit finger-like projections or processes. For example, the neuron that senses when you stub your toe has a very long process called an *axon* that extends from sensory endings in the toe to the spinal cord and up to the brain. In a tall individual, this axon is almost 2 m (2,000,000 μm) long but is only about 2 μm in diameter. Neurons also can have many shorter processes called *dendrites*. Typically, the axon of one neuron originates from the *cell body* at a region called the *axon hillock*, and the axon carries an action potential toward the dendrites of other neurons. At the *axon terminal*, chemicals called neurotransmitters are released and diffuse from the axon terminal to the cell body and dendrites of the target cell which has receptors to receive these chemical signals. Thus, the cell body and dendrites of a neuron receive inputs from the axon terminals of other neurons, integrate this information, and send an output via its axon. Figure 1 shows a schematic of a neuron, similar to those found in most texts, indicating the important structures and the direction of information flow. See Kandell et al. (1995) and Chudler (2003) for useful descriptions and illustrations of neurons.

The main function of neurons is communication. In the example above, the physical impact on the toe is sensed by a neuron with endings in the skin of the toe; the axon of this neuron conducts action potentials to the spinal cord and, from there, to the brain. This first neuron, a sensory neuron, releases neurotransmitter onto a second neuron that can also generate an action potential, and thus conducts the signal to a third neuron. In the brain the generation of action potentials by additional neurons produces the perceptions and thoughts that we associate with stubbing one's toe–pain, discomfort, anger. Subsequent activation of neurons that exit the brain can produce motor responses such as yelling or hopping around on the uninjured foot or rubbing the injured toe. If a local anesthetic such as lidocaine is administered, the action potentials are blocked and there is no perception or response to the stimulus.

The example above illustrates the importance of action potentials in the nervous system. The analogies below using a sparkler and a frying pan may aid in the understanding and teaching of how action potentials are generated and propagate along the neuronal membrane. These analogies can be used at a basic level to demonstrate propagation and positive feedback and/or, at a more advanced level, to understand the molecular mechanisms underlying the action potential. The section below on the role of ions is an example at the molecular level. A background in ion transport across cell membranes via ion channel proteins is needed for this section. *Essentials of Neural Science and Behavior* by Kandel et al. (1995) and *Animal Physiology* by Randall et al. (2002) are examples of textbooks that specifically relate

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transport to neuronal function; examples of textbooks with additional molecular detail and numerous references to original experiments are *Molecular and Cellular Physiology of Neurons* by Fain (1999) and *From Neuron to Brain* by Nicholls et al. (1992).

The Role of Ions

Most animal cells have a membrane potential (described briefly below) because most cells have sodium-potassium pump proteins and passive potassium channel proteins in their membranes. Neurons have additional proteins–gated ion channels–that allow the membrane potential to change. These changes in membrane potential act as signals in neurons.

The sodium-potassium pump (also called the Na^+-K^+ ATPase) is a transport protein that uses energy in the form of ATP to simultaneously transport Na^+ out of the cell and K^+ into the cell. As a consequence of this active transport, the inside of a typical cell has a high K^+ concentration and a low Na^+ concentration compared to the respective concentrations outside the cell membrane (e.g., Randall et al., 2002).

The concentration gradients for both Na^+ and K^+ across the cell membrane exert concentration forces acting on each ion. The force on Na^+, which is higher in concentration outside, moves Na^+ into the cell and the force on K^+ moves K^+ out of the cell. However, at rest (when the neuron is not generating a signal) most of the sodium channels in the membrane are closed, and therefore sodium cannot move in. On the other hand, there are passive K^+ channels in the membrane, channels that are always open, and therefore some K^+ moves out of the cell by diffusion. This net outward diffusion of K^+ down its concentration gradient moves net positive charge out of the cell, leaving behind negatively charged molecules, large anions and charged proteins, that cannot easily diffuse across the membrane. These negative charges accumulate at the inside surface of the membrane to produce a resting membrane voltage or a *membrane potential* across the cell membrane (e.g., Kandel et al., 1995). A membrane potential exists because the membrane is selectively permeable to K^+. Thus, at rest, the membrane of a neuron is electrically charged or polarized, with the inside of the membrane negatively charged relative to the outside.

Neurons generate signals when the membrane potential changes. One such change in the neuron's membrane potential is the action potential, and a key molecule in generating an action potential is a membrane protein called the voltage-gated sodium channel. If the inside of the membrane becomes less negative (less polarized), the change in membrane potential is called a *de*polarization. A *depolarization* can trigger an action potential because a depolarization of the membrane increases the probability that the voltage-gated sodium channels will open (e.g., Fain, 1999). In the previous example, when the toe was stubbed, the skin was impacted and this mechanical force physically distorted the membrane of the sensory neurons at their sensory endings. This force opened mechanically-gated (mechanically-sensitive) channels that allowed net positive charge to enter and depolarize the membrane. This initial depolarization then acted on the voltage-gated channels located in the membrane near the axon hillock and axon to produce action potentials.

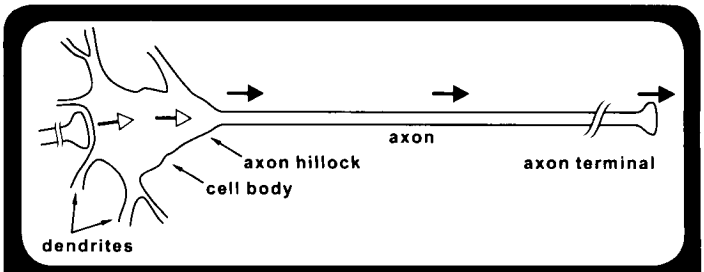

Figure 1. Structure of a neuron. Short branched processes called dendrites extend from the cell body. A longer process, the axon, ends in an axon terminal. The axon terminal of one neuron often contacts the dendrites and/or cell body of another neuron. The direction of information flow, from axon terminal of one neuron to the cell body and/or dendrites of a second neuron to the axon hillock and down the axon to the axon terminal of the second neuron, is indicated by the arrows. Open arrows indicate passive spread toward the axon hillock; the filled arrows indicate the active propagation of an action potential down the axon.

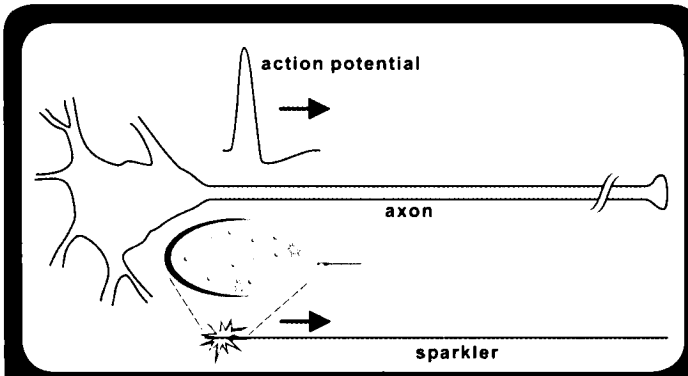

Figure 2. Neuron and sparkler. An action potential is propagated down the axon by mechanisms analogous to an explosion moving along a sparkler or fuse (see text). The action potential is a large (100 millivolt), rapid depolarization of the membrane. In the sparkler, the inset (under the magnifying glass) illustrates the initial events that occur when the sparkler is lit. Ignition of one particle of explosive material produces additional heat which causes additional particles to ignite.

The Action Potential

An action potential is a large, brief change in membrane potential consisting of a rapid depolarization to a peak (rising phase), a repolarization back toward the resting potential, an undershoot where the membrane hyperpolarizes to a more negative level, and a return to rest (see Figure 2). This sequence of events occurs if a sufficient initial depolarization occurs at the axon hillock. The requirement for a sufficient initial depolarization is a concept called *threshold*. Once threshold is reached, the action potential goes to

completion; if threshold is not reached, no action potential is generated. This concept is also sometimes referred to as the "all-or-none" property of an action potential. For the analogy presented in this paper, only the rising phase of the action potential is considered in detail.

As the name implies, voltage-gated channels open when the membrane voltage changes, specifically when the membrane depolarizes to threshold. An initial depolarization (such as one generated by mechanically-gated channels when the toe is stubbed) opens some voltage-gated Na^+ channels. When open, these channels allow Na^+ to move into the cell down their concentration gradient. Since Na^+ is a positively-charged ion, its entry makes the inside of the cell less negative, causing the membrane to depolarize further. This additional depolarization opens more voltage-gated Na^+ channels, and this allows more Na^+ to enter, which depolarizes the membrane further. This is an example of positive feedback, and very quickly the membrane depolarizes completely to zero and then becomes inside positive (called the overshoot) as voltage-gated Na^+ channels continue to open and Na^+ continues to enter the cell. This large, brief depolarization and overshoot are the rising phase of an action potential.

Following the peak of the action potential, the membrane repolarizes as the voltage-gated Na^+ channels close and other voltage-gated channels, voltage-gated K^+ channels, open (e.g., Purves et al., 2001). These additional open K^+ channels allow additional K^+ to diffuse out of the cell so that the membrane repolarizes and actually undershoots the resting membrane potential. Repolarization resets the membrane so that subsequent action potentials can be generated after a brief refractory period. The sparkler analogy is not very useful in illustrating the mechanisms of repolarization or the refractory period.

The Analogy

The axon of a neuron is represented by a sparkler, a wire coated with explosive material, with hundreds of explosive particles similar to gunpowder at any point. An action potential is analogous to the explosion at any point, and such an explosion can illustrate positive feedback, threshold, and the all-or-none property of an action potential. In this analogy, the heat-sensitive explosive is analogous to the voltage-gated (depolarization-sensitive) sodium channels, and heat is analogous to a depolarization. An explosive particle will ignite if it is heated to threshold, the initial heat usually supplied by a match. The ignition of one particle produces additional heat which causes additional particles of explosive to ignite. Thus, an explosion is an example of positive-feedback in which heat produces more heat to produce a large amount of heat, an explosion. The cycle that generates such as explosion is illustrated in the magnified inset of Figure 2.

In the case of an action potential, via voltage-gated sodium channels, an initial threshold depolarization produces more depolarization to produce a very large depolarization, an action potential. An explosion is a large amount of heat; an action potential is a large depolarization. With an explosive material like gunpowder, once the heat reaches threshold, an explosion will occur; thus an explosion is all-or-none. In the axon, once the depolarization reaches threshold, an action potential will occur. Thus, all of the characteristics of an action potential–threshold, positive-feedback, and the all-or-none property–can be illustrated with the explosive used in a sparkler.

Continuous Propagation of the Action Potential

Once generated, the action potential travels down the axon to the axon terminals (e.g., Purves et al., 2001). Since the action potential must travel over relatively long distances, an active mechanism is needed; passive spread would not produce a large depolarization at the end of the axon. Passive spread is easily explained using a heat analogy. If you heated one end of a long piece of bare wire, the heat would spread passively in the wire so that the wire right next to the place you heated would get hot, but the other end would not get very hot. If neurons used only passive spread, the signal (the change in membrane potential) would become very small as it spread passively down the axon (e.g., Kandel et al., 1995).

Neurons avoid this problem by producing a new action potential in each patch of membrane along the axon. The action potential does not passively spread from one end of the axon to the other; rather a new action potential is generated at each point along the axon. This mechanism is fittingly called regeneration or propagation. The action potential that occurs in the axon of the sensory neuron near the toe, the action potential in the axon farther up the leg, and the action potential that occurs in the axon as it enters the spinal cord are each full, all-or-none, newly-made action potentials, and the signal therefore maintains its amplitude and fidelity.

The sparkler can be used to illustrate the propagation of the action potential along the axon's membrane. In some axons (called unmyelinated axons) a new action potential is generated at each point along the axon because there are voltage-gated sodium channels all along the axon. The action potential at one point, itself a huge depolarization, easily depolarizes the adjacent membrane so that this adjacent region reaches threshold and generates a new action potential. Propagation that occurs all along the axon is called continuous propagation. Another form of propagation (found in myelinated axons) is called saltatory conduction, but is not easily demonstrated with the sparkler.

The Sparkler Demonstration

A sparkler consists of explosive material spread along a metal wire and is a good model to demonstrate continuous propagation. The presence of explosive all along the sparkler is analogous to the presence of voltage-gated channels all along the axon's membrane. When the sparkler is lit and reaches threshold to produce an initial explosion, the heat from the initial explosion easily spreads (passively) to the next region of the sparkler, bringing it to threshold and causing a new explosion there. Thus, a new explosion is made at each point along the sparkler, and the size of explosion at the end is the same magnitude as the initial explosion. An

action potential is similar. Once threshold is reached, the large depolarization from the initial action potential brings the next region of the axon to threshold and generates a new action potential there. Thus, a new action potential is made at each point along the axon.

Narrating the Demonstration

I demonstrate the above concepts by first reviewing the parts of a neuron and the generation and propagation of an action potential. I then explain the analogy, making a simple chart indicating what properties are analogous:

heat = depolarization

explosion = action potential

explosion is a large amount of heat

action potential is a large depolarization

I then light a match (or a candle) and briefly heat the sparkler saying something like, "This is not a large enough depolarization; we have not yet reached threshold; therefore there is no action potential." I then heat the sparkler longer (or better yet, use a larger candle), and when it still doesn't light, I repeat the above and heat longer. When the sparkler finally does light, I wait a few seconds for the "ohs and ahs" to end and then say, "Now we've reached threshold and an 'all or none' action potential occurs." As the sparkler burns, I narrate continuous propagation. Figure 2 provides a graphic to compare the axon and the sparkler.

It is useful to perform the demonstration again, possibly having students help narrate the sequence of steps involved in generating and propagating an action potential. The goal is for the students to see the sparkler and yet talk about the neuron in terms of depolarization, threshold, action potentials, propagation, and refractory periods. Students should be warned not to repeat the demonstration unsupervised. One also needs to be aware of smoke detectors, and give appropriate notification that you will be lighting a sparkler in your classroom.

The Synapse

A related analogy, heating and/or cooling a frying pan, is useful to explain how information is transferred from one neuron to another. When the action potential in one neuron reaches the axon terminal, it triggers the release of neurotransmitter molecules which diffuse to a target, often the dendrites or cell body of another neuron. The specialized structures involved in the release of neurotransmitter and the structures involved in its action on the target neuron occur at a region called a *chemical synapse*. In the example above, the sensory neuron from the toe makes chemical synapses with second-order neurons in the spinal cord and brain. At each target neuron, the chemical neurotransmitter binds to receptor proteins causing ion channels in the target neuron's membrane to open or close. These chemically-gated channels produce small changes in the membrane potential of the target cell called *post-synaptic potentials*. Post-synaptic potentials determine whether the target cell reaches a threshold depolarization and generates an action potential or not. The post-synaptic potential in the second-order neuron plays the same role as the potential in the receptor end (receptor potential) in the sensory neuron, such as the response in the toe. If the second or subsequent neurons do not generate an action potential, perception of a sensory stimulus may not occur. In fact, animals do not perceive most of the sensory information that impinges on them.

Depending on the specific synapse, post-synaptic potentials can be either *excitatory* or *inhibitory* (e.g., Kandel et al., 1995). Excitatory post-synaptic potentials depolarize the target and help it reach threshold. Inhibitory post-synaptic potentials make it more difficult for the target to reach threshold (one mechanism of inhibition is a hyperpolarization, making the membrane more polarized or more negative inside). The change in potential at each synapse is usually small, so that many excitatory post-synaptic potentials are needed to reach threshold (or many inhibitory post-synaptic potentials to prevent the target from reaching threshold). A typical neuron in the central nervous system receives more than 1,000 synapses (e.g., Kandel et al., 1995). The opposing mechanisms–depolarization from excitation and hyperpolarization from inhibition–are combined or integrated in the membrane potential of each target cell. This *integration* or summing of excitation and inhibition determines whether the target cell will reach threshold or not.

An Analogy of Synaptic Integration

In most texts, chemical synapses are shown impinging onto the cell body or dendrites, and the action potential is generated near the axon hillock, a region where the axon protrudes from the cell body. An iron frying pan can be used as a model of the cell body (or more generally, the receiving region of a neuron) with synapses occurring onto the pan, and with the iron handle of the pan analogous to the axon hillock. One can add a long, large sparkler or fuse extending from the handle, analogous to the axon (see Figure 3). For a neuron with an axon 10 mm in length, the fuse or sparkler representing the axon would need to be about 20 m in length to be at the same scale as the frying pan.

Heating is again analogous to a depolarization at an excitatory synapse, while cooling signifies a hyperpolarization at an inhibitory synapse. At an analogous excitatory synapse onto the frying pan, heat is generated; thus, excitatory synapses can be represented as sources of heat such as little torches or candles that can heat (depolarize) the frying pan (cell body). The iron pan integrates (sums) all these excitatory inputs and gets hot. If there is enough heat added to the pan, the handle gets hot and, if it reaches threshold, would ignite the fuse. By analogy, in a neuron, excitatory synapses cause depolarizing post-synaptic potentials that sum together toward threshold. From the synapse to the axon hillock, the spread of depolarization is passive, allowing depolarizations from many synapses to sum together. These potentials are not 'all or none' but rather are graded in amplitude. If threshold is reached at the axon hillock, an action potential is generated and actively propagated down the axon to the next synapse. In Figure 1, this passive spread is indicated by the open arrows; the active propagation is indicated by the filled arrows.

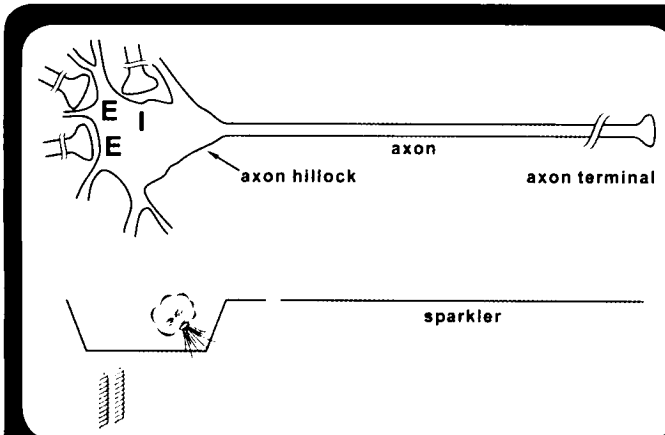

Figure 3. Synaptic integration. Three axon terminals are shown making chemical synapses with the cell body and dendrites of a neuron. Excitatory synapses are indicated by **E**; inhibitory synapses by **I**. The resultant post-synaptic potentials are integrated and spread to the axon hillock, where an action potential will be generated if threshold is reached. Below, a frying pan is used to represent the cell body and dendrites. Inputs that heat the pan, shown by candles, are analogous to excitatory inputs. Inputs that cool the pan, shown by the wind, are analogous to inhibitory inputs. These inputs spread to the handle (analogous to the axon hillock), where, if the handle gets hot enough, the sparkler will ignite.

Inhibition is just as important in the nervous system as excitation; for example, in diseases such as Parkinson's, the tremors are thought to be the result of a lack of normal inhibition in a part of the brain that controls movement (Côté & Crutcher, 1991). In the analogy, cooling occurs at inhibitory synapses; to convey the concept of integration of inhibition, I have students imagine cold sources such as little fans, wind, or ice cubes cooling the frying pan. Cooling the target represents the hyperpolarizing post-synaptic potentials at inhibitory synapses. One can imagine the frying pan, the input region of the neuron, being heated and/or cooled by the candles and/or wind (Figure 3). If there is sufficient inhibition (cooling), threshold will not be reached. Typical neurons receive hundreds to thousands of synaptic inputs, some excitatory and some inhibitory. The resulting depolarizations and hyperpolarizations of the target sum together to determine whether threshold is reached. If the target neuron depolarizes to threshold, an all-or-none action potential will be initiated and propagated. If threshold is not reached, no action potential will be generated in the target neuron.

A discussion of the neurons involved in the perception and response to stubbing one's toe can illustrate the importance of synaptic integration. The axon of the sensory neuron from the toe enters the central nervous system at the spinal cord and makes synapses in the cord and in the brain onto second-order neurons which conduct action potentials and make synapses with third-order neurons. Each individual synapse onto a target neuron is one of hundreds or thousands, some bringing information from other sensory neurons, some bringing additional information from the brain. Some of these synapses are excitatory and some are inhibitory, and all the active synapses are integrated (summed together) as described above, at each and every level of the nervous system. This partly explains why the perceptions and responses to stimuli can be so variable. If you've been awakened in the middle of the night by a wrong telephone number or a car alarm outside and are already upset, stubbing your toe can cause an intense reaction as more excitatory inputs are active and summed together. On the other hand, if you're playing ball, you may not even notice that you've stubbed your toe because inhibitory inputs to these sensory circuits concerned with the toe dominate while excitation to muscles and circuits associated with the game are occurring.

Discussion & Conclusions

In this paper analogies are presented to illustrate some of the fundamental physiological mechanisms that neurons use to communicate information in the nervous system. Analogies can help students integrate new knowledge with existing knowledge and have been shown to be useful tools for teaching science (e.g., Glynn et al., 1995). When

analogies are used, it is particularly important to clearly map the scientific concepts being illustrated (referred to as the target in the analogy literature) to the analogy, and to delineate where the analogy breaks down (Glynn, 1991; Harrison & Treagust, 1994).

The first analogy presented was between heat-sensitive explosive material to produce an explosion that travels along a wire in a sparkler and voltage-gated sodium channels to generate an action potential that propagates along the axon in a neuron. The analogy illustrates the rising phase of the action potential and the concepts of threshold, positive feedback, passive spread, propagation, and the "all-or-none" principle.The sparkler analogy does not illustrate repolarization or the refractory period that follows the peak of an action potential. A second analogy was between sources of heat and cold that change the temperature of a frying pan and the excitatory and inhibitory synapses made onto the cell body of a neuron. This analogy illustrates the concepts of excitation, inhibition, and integration, but does not address the physiological mechanisms that occur at a chemical synapse.

The current concepts of neurophysiology indicate that all our reactions, perceptions, thoughts, and memories are determined by which neurons are generating action potentials (Kandel et al., 1995; Nicholls et al., 1992). The activity of neurons, in turn, depends on the integration of excitation and inhibition that occurs at the receiving region, the cell body and dendrites, of each cell. If threshold is reached, an action potential will be generated and propagated to the next part of the circuit. Furthermore, most therapeutic and recreational drugs exert their effects by mimicking neurotransmitters to produce excitation or inhibition, by blocking the effects of these neurotransmitters, or by controlling the availability of these neurotransmitters. For example, morphine and codeine mimic the action of endogenous opiate neurotransmitters; selective serotonin reuptake inhibitors (SSRIs) such as Prozac increase the amount of serotonin, another neurotransmitter (Snyder, 1996). An increase in neurotransmitter at the synapse will increase the amount of excitation or inhibition, depending on the action of that neurotransmitter at that synapse. The concepts of synaptic integration, excitation, inhibition, threshold, and the generation and propagation of action potentials provide a basis for discussing the functions of the nervous system. These concepts can be applied to the variety of more specific topics from sensory perception to psychological disorders to cognition.

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