
**Key Features**

- Integrates the latest findings in our rapidly increasing understanding of genetic influences on behavior
- Contains an eye-catching four-color interior design and extensive illustrative art program
- Covers intelligence and consciousness in separate chapters, which is unique compared to the competition
- Explores drugs and addiction in a separate chapter
- Brings the focus on behavior to the forefront
- Presents opening vignettes of real life topics that help to make the chapter content come alive
- Offers features throughout the text such as “Concept Checks” and marginal questions designed to assist the student in assessing their comprehension
- Provides useful end-of-chapter pedagogy such as: “In Perspective” sections followed by a summary of key points in the chapter; “For Further Thought” sections that require the student to think about and complete brief projects that will assist them in their understanding of the chapter content; and “Test Your Understanding” sections

**The Robust Ancillaries Include**

- **Instructor’s Resource CD-ROM** that contains a computerized test bank, PowerPoint Slides, sample syllabi, and suggested in-class and homework assignments
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- The **Student Study Guide** that offers chapter outlines; learning objectives; summary and guided reviews (incorporating key terms and concepts); short-answer and essay questions; and a chapter post-test
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Glossary
References
Bob Garrett is currently a visiting research scholar at California Polytechnic State University, San Luis Obispo. He was Professor of Psychology at DePauw University in Greencastle, Indiana, and held several positions there, including Chairperson of the Department of Psychology, Faculty Development Coordinator, and Interim Dean of Academic Affairs. He received his BA from the University of Texas at Arlington and his MA and PhD from Baylor University.
Preface

A Message From the Author

A benefit of growing up poor was that I learned the value of an education. And it didn’t take long to discover that the real value of education isn’t just a ticket to a better job but all the learning along the way about life and the world and what makes both of them work. That is what led me, after trying one major after another, to discover psychology.

A child of Sputnik and enamored with science, I was especially attracted by the young and promising discipline of biological psychology. And as I pursued that promise, I was attracted to another—sharing my enthusiasm through teaching. For many years, I taught at DePauw University, where practically every student does two or three internships and the value of research is judged by what students learn from working alongside their mentors; similarly, the guiding principle at my current university, Cal Poly, San Luis Obispo, is that students should “learn by doing.” I believe in knowledge for its own sake, but I value knowledge that is useful even more. Perhaps that is why I needed to write *Brain and Behavior,* it is my testimonial to the usefulness of scientific knowledge.

Now that the second edition is done, I can look forward to more leisurely ways of spending my time: beach walks and tennis with my wife, hiking the hillsides near our home, and watching our grandchildren grow. But you can be sure I’ll be watching out of the corner of my eye to see whether students are enjoying what I have written and whether they are experiencing the same thrill of discovery I had when I was their age.

To Instructors

When I first wrote *Brain and Behavior,* I had one goal, to entice students into the adventure of biological psychology. There are other good texts out there, but they read like they were written for serious junior and senior psychology majors who appreciate the importance of biological psychology in its own right. This book is for them, too, but I wrote it so any student who is interested in behavior, including the newly declared sophomore major or the curious student who has wandered over from the history department, could have the deeper understanding that comes from a biological perspective as they take other courses in psychology.

It is not enough to draw students in with lively writing or by piquing their interest with case studies and telling an occasional story along the way; unless they feel they’re learning something significant, they won’t stay—they’ll look for excitement in more traditional places. As I wrote, I remembered the text I struggled with in my first biopsychology class; it wasn’t very interesting because we knew much less about the biological underpinnings of behavior than we do now. Since that time, we’ve learned how the brain changes during learning, we’ve discovered some of the genes and brain deficiencies that cause schizophrenia, and we’re beginning to understand how intricate networks of brain cells produce language, make us intelligent, and help us play the piano or find a mate. In other words, biopsychology has become a lot more interesting. So the material is there; now it is my job to communicate the excitement I’ve felt in discovering the secrets of the brain and to make a convincing case that biopsychology has the power to answer the questions *students* have about behavior.
A good textbook is all about teaching, but there is no teaching if there is no learning. Over the years, my students taught me a great deal about what they needed to help them learn. For one thing, I realized how important it is for students to build on their knowledge throughout the course, so I made several changes from the organization I saw in other texts. First, the chapter on neuronal physiology precedes the chapter on the nervous system, because I believe that you can’t understand how the brain works unless you know how its neurons work. And I reversed the usual order of the vision and audition chapters, because I came to understand that audition provides a friendlier context for introducing the basic principles of sensation and perception. The chapters on addiction, motivation, emotion, and sex follow the introduction to neurophysiology; this was done to build student motivation before tackling sensation and perception. Perhaps more significantly, some topics have been moved around among chapters so they can be developed in a more behaviorally meaningful context. So language is discussed along with audition, the body senses with the mechanisms of movement, the sense of taste in the context of feeding behavior, and olfaction in conjunction with sexual behavior. Most unique, though, is the inclusion of a chapter on the biology of intelligence and another on consciousness. The latter is a full treatment of recent developments in the field, rather than limited to the usual topics of sleep and split-brain behavior. These two chapters strongly reinforce the theme that biopsychology is personally relevant and capable of addressing important questions.

Brain and Behavior has several features that will motivate students to learn and encourage them to take an active role in their learning. It engages the student with interest-grabbing opening vignettes, illustrative case studies, and In the News items and Application boxes that take an intriguing step beyond the chapter content. Throughout each chapter, marginal questions keep the student focused on key points, a Concept Check at the end of each section serves as a reminder of the important ideas, and On the Web icons point the way to related information on the Internet. At the end of the chapter, In Perspective emphasizes the importance and implications of what the student has just read, a summary helps organize that information, and Testing Your Understanding assesses the student’s conceptual understanding as well as factual knowledge. Then, For Further Reading is a guide for students who want to explore the chapter’s topics more fully. I have found over the years that students who use the study aids in a class are also the best performers in the course.

New in the Second Edition

As you would expect, the second edition of Brain and Behavior includes a number of changes. Foremost, and reflecting the rapid advances in biological psychology and neuroscience, this edition contains 500 new references. More than 60 illustrations have been added, and 25 others were significantly revised to increase their informational and educational value. In addition, new tables have been added where there was a need to organize or summarize complex material. In addition, most of the In the News and Application features have been either replaced or updated with more recent information. The material on research techniques that was previously
in the appendix has been expanded and combined with two topics from the first edition’s introductory chapter, Science, Research, and Theory and Research Ethics, to form the new chapter “The Methods and Ethics of Research.” This provides research methodology the emphasis it deserves while giving the introductory chapter a sharper focus.

The new edition continues its theme of showcasing our rapidly increasing understanding of genetic influences on behavior with discussions of numerous recent findings, particularly with regard to obesity, hostility and aggression, Parkinson’s disease, Alzheimer’s disease, autism, and schizophrenia. Another theme that has been strengthened is the broader societal relevance of biopsychology, from the ethical implications of stem cell research to the cost of addictions and disorders, to new strategies for treating brain and spinal cord damage.

To the Student

Brain and Behavior is my attempt to reach out to students, to open a door and beckon them inside to experience the fascinating world of biological psychology. These are exceptionally exciting times, comparable in many ways to the renaissance that thrust Europe from the Middle Ages into the modern world. In Chapter 1, I quote Kay Jamison’s comparison of neuroscience, which includes biopsychology, to a “romantic, moon-walk sense of exploration.” I know of no scientific discipline with greater potential to answer the burning questions about ourselves than neuroscience in general and biopsychology in particular. I hope this textbook will convey that kind of excitement as you read about discoveries that will revolutionize our understanding of what it means to be human.

I want you to succeed in this course, but, more than that, I want you to learn more than you ever imagined you could and to go away with a new appreciation of the promise of biological psychology. So now I’m going to start sounding like a parent. I want you to sit near the front of the class, because those students usually get the best grades. That’s probably because they stay more engaged and ask more questions; but to ask good questions you should always read the text assignment before you go to class. And so you’ll know where you’re going before you begin to read, take a look at “In this chapter you will learn,” then skim the chapter subheadings, and read the summary. Use the marginal questions as you go through, answer the Concept Check questions, and be sure to test yourself at the end. Later, it’s a good idea to explore the Web sites listed in On the Web and the resources in For Further Reading. You won’t just do better in this course; you’ll leave saying, “I really got something out of that class!”

I wrote Brain and Behavior with you in mind, so I hope you will let me know where I have done things right and, especially, where I haven’t (bgarrett@calpoly.edu). I wish you the satisfaction of discovery and knowledge as you read what I have written for you.
Supplemental Material

Student Study Guide

This affordable student study guide and workbook to accompany Bob Garrett’s Brain and Behavior, Second Edition will help students get the added review and practice they need to improve their skills and master their course. Each part of the study guide corresponds to the appropriate chapter in the text and includes the following: chapter outline, chapter summary, study quiz, and a chapter posttest.

Student Study Site

This free student study site provides additional support to students using Brain and Behavior, Second Edition. The Web site includes e-flashcards, study quizzes (students can receive their score immediately), relevant SAGE journal articles with critical thinking questions, and relevant Internet resources. Also included are animations of key figures in the text. Visit the study site at www.sagepub.com/garrettbb2study.

Instructor’s Resources on CD-ROM

This set of instructor’s resources provides a number of helpful teaching aids for professors new to teaching biological psychology and to using Brain and Behavior, Second Edition. Included on the CD-ROM are PowerPoint slides, a computerized test bank to allow for easy creation of exams, lecture outlines, suggested class activities and critical thinking questions, and video and Internet resources for each chapter of the text.

Acknowledgments

I have had a number of mentors along the way, to whom I am forever grateful. A few of those special people are Wayne Kilgore, who taught the joys of science along with high school chemistry and physics; Garvin McCain, who introduced me to the satisfactions of research; Roger Kirk, who taught me that anything worth doing is worth doing over and over until it’s right; and Ellen Roye and Ouida Piner, who shared their love of language. These dedicated teachers showed me that learning was my responsibility, and they shaped my life with their unique gifts and quiet enthusiasm.

My most important supporter has been my wife, Duejean; love and thanks to her for her patient understanding and her appreciation of how important this project is to me. And then, applause all around for the people at Sage, especially Cheri Delello, Sarah Quesenberry, Stephanie Adams, Deya Saoud, Lara Grambling, and Ravi Balasuriya, whose competence and professionalism convinced me that Sage is “the natural home for authors”; but most of all, thanks to Vicki Knight, editor for both editions and the guiding force behind Brain and Behavior. Then, my appreciation to DePauw University for the sabbatical leave that started this whole project and to Cal Poly for all the resources it has provided.
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— Bob Garrett
Things were looking good for Jim and his wife. She was pregnant with their first child and they had just purchased and moved into a new home. After the exterminating company treated the house for termites by injecting the pesticide chlordane under the concrete slab, Jim noticed that the carpet was wet and there was a chemical smell in the air. He dried the carpet with towels and thought no more about it, not realizing that chlordane can be absorbed through the skin. A few days later, he developed headaches, fatigue, and numbness. Worse, he had problems with memory, attention, and reasoning. His physician referred him to the toxicology research center of a large university medical school. His intelligence test score was normal, but the deficiencies he was reporting showed up on more specific tests of cognitive ability. Jim and his wife had to move out of their home. At work, he had to accept reduced responsibilities because of his difficulties in concentration and adapting to novel situations. The chlordane had not damaged the structure of his brain as you might suspect, but it interfered with the functioning of the brain cells by impairing a mechanism called the...
sodium-potassium pump (Zillmer & Spiers, 2001). Jim’s unfortunate case reminds us that the nervous system is as delicate as it is intricate. Only by understanding how it works will we be able to appreciate human behavior, to enhance human performance, and to treat behavioral problems such as drug addiction and psychosis.

The Cells That Make Us Who We Are

To understand human behavior and the disorders that affect it, we must understand how the brain works. And to understand how the brain works we must first have at least a basic understanding of the cells that carry messages back and forth in the brain and throughout the rest of the body. Neurons are specialized cells that convey sensory information into the brain, carry out the operations involved in thought, feeling, and action, and transmit commands out into the body to control muscles and organs. It is estimated that there are about 100 billion neurons in the human brain (Figure 2.1; Williams & Herrup, 1988, 2001). This means that there are more neurons in your brain than stars in our galaxy. But as numerous and as important as they are, neurons make up only 10% of the brain’s cells and about half its volume. The other 90% are glial cells, which we will discuss later in the chapter.

Neurons

Neurons have the responsibility for all the things we do—our movements, our thoughts, our memories, and our emotions. It is difficult to believe that anything so simple as a cell can measure up to this task, and the burden is on the neuroscientist to demonstrate that this is true. As you will see, the neuron is deceptively simple in its action but impressively complex in its function.

Basic Structure: The Motor Neuron

First let’s look inside a neuron, because I want to show you that the neuron is a cell, very much like other cells in the body. Figure 2.2 is an illustration of the most prominent part of the neuron, the cell body or soma. The cell body is filled with a watery liquid called cytoplasm and contains a number of organelles. The largest of these organelles is the nucleus, which contains the cell’s chromosomes. Other organelles are responsible for converting nutrients into fuel for the cell, constructing proteins, and removing waste materials. So far, this could be the description of any cell; now let’s look at the neuron’s specializations that enable it to carry out its unique role. Figure 2.3 illustrates a typical neuron. I say “typical” guardedly, because there are three major kinds of neurons and variations within those types. This particular type is a motor neuron, which carries commands to the muscles and organs. It is particularly useful for demonstrating the structure and functions that neurons have in common.
Dendrites are extensions that branch out from the cell body to receive information from other neurons. Their branching structure allows them to collect information from many neurons. The axon extends like a tail from the cell body and carries information to other locations, sometimes across great distances. The myelin sheath that is shown wrapped around the axon supports the axon and provides other benefits that we will consider later. Branches at the end of the axon culminate in swellings called end bulbs or terminals. The terminals contain chemical neurotransmitters, which the neuron releases to communicate with a muscle or an organ or the next neuron in a chain. In our examples, we will talk as if neurons form a simple chain, with one cell sending messages to a single other neuron, and so on; in actuality, a neuron receives input from many neurons and sends its output to many others.

Neurons are usually so small that they can be seen only with the aid of a microscope. The cell body is the largest part of the neuron, ranging from 0.005 to 0.1 millimeter (mm) in diameter in mammals. (In case you are unfamiliar with metric measurements, a millimeter is about the thickness of a dime.) Even the giant neurons of the squid, favored by researchers for their conveniently large size, have cell bodies that are only 1 mm in diameter. Axons, of course, are smaller; in mammals, they range from 0.002 to 0.02 mm in diameter. Axons can be anywhere from 0.1 mm to more than a meter in length.

Other Types of Neurons

The second type of neuron is the sensory neuron. Sensory neurons carry information from the body and from the outside world into the brain and spinal cord. Motor and sensory neurons have the same components but they are configured differently. A motor neuron’s axon and dendrites extend in several directions from the cell body, which is why it is called a multipolar neuron. Sensory neurons can be either unipolar or bipolar. The sensory neuron in Figure 2.4a is called a unipolar neuron because of the single short stalk from the cell body that divides into two branches. Bipolar neurons have an axon on one side of the cell body and a dendritic process on the other (Figure 2.4b). Motor and sensory neurons are specialized for transmission over long distances; their lengths are not shown here in the same scale as the rest of the cell.

The third type is neither motor nor sensory. Interneurons connect one neuron to another in the same part of the brain or spinal cord. Notice in Figure 2.4c that...
this neuron is also multipolar, but its axon appears to be missing; for some interneurons this is so, and when they do have axons, they are often so short that they are indistinguishable from dendrites. Because interneurons make connections over very short distances, they do not need the long axons that characterize their motor and sensory counterparts. In the spinal cord, interneurons bridge between sensory neurons and motor neurons to produce a reflex. In the brain, they connect adjacent neurons to carry out the complex processing that the brain is noted for. Considering the major role they play, it should come as no surprise that interneurons are the most numerous.

The different kinds of neurons operate similarly; they differ mostly in their shape, which fits them for their specialized tasks. We will examine how neurons work in the next few sections. The types of neurons and their characteristics are summarized in Table 2.1.

Table 2.1 The Major Types of Neurons

<table>
<thead>
<tr>
<th>Type</th>
<th>Function</th>
<th>Form and Location</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor</td>
<td>Conducts messages from brain and spinal cord to muscles and organs</td>
<td>Multipolar; throughout nervous system</td>
<td>Axon, dendrites extend in several directions from cell body</td>
</tr>
<tr>
<td>Sensory</td>
<td>Carries information from body and world to brain and spinal cord</td>
<td>Unipolar; outside brain</td>
<td>Single short stalk from cell body divides into two branches</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bipolar; outside brain and spinal cord</td>
<td>Axon and dendritic processes on opposite sides of cell body</td>
</tr>
<tr>
<td>Interneuron</td>
<td>Conducts information between neurons in same area</td>
<td>Multipolar; brain and spinal cord</td>
<td>Short axon or no axon</td>
</tr>
</tbody>
</table>

The Neural Membrane and Its Potentials

The most critical factor in the neuron’s ability to communicate is the membrane that encloses the cell. The membrane is exceptionally thin—only about 8 micrometers (millionths of a meter) thick—and is made up of lipid (fat) and protein (see Figure 2.5). Each lipid molecule has a “head” end and a “tail” end. The heads of the molecules are water soluble, so they are attracted to the seawater-like fluid around and inside cells. The tails are water insoluble, so they are repelled by the fluid. Therefore, as the heads orient toward the fluid and the tails orient away from...
the fluid, the molecules turn their tails toward each other and form a double-layer membrane.

The membrane not only holds a cell together but also controls the environment within and around the cell. Some molecules, such as water, oxygen, and carbon dioxide, can pass through the membrane freely. Many other substances are barred from entry. Still others are allowed limited passage through protein channels (shown here in green) that open and close under different circumstances. This selective permeability contributes to the most fundamental characteristic of neurons, *polarization*, which means that there is a difference in electrical charge between the inside and the outside of the cell. A difference in electrical charge between two points, such as the poles of a battery or the inside and outside of a cell, is also called a *voltage*.

*The Resting Potential.* Just as you would measure the voltage of a battery, you can measure a neuron’s voltage (see Figure 2.6). By arbitrary convention, the voltage is expressed as a comparison of the inside of the neuron with the outside. The difference in charge between the inside and outside of the membrane of a neuron

---

**Figure 2.5**

*Cross Section of the Cell Membrane of a Neuron.*

Notice how the lipid molecules form the membrane by orienting their heads toward the extracellular and intracellular fluids.

---

**Figure 2.6**

*Recording Potentials in a Neuron.*

Potentials are being recorded in the axon of a neuron, with an electrode inside the cell and one in the fluid outside. On the right is a microscopic view of a microelectrode about to penetrate a neuron.

Source: © Bob Jacobs, Colorado College.
at rest is called the **resting potential**. This voltage is negative and varies anywhere from –40 to –80 millivolts (mV) in different neurons but is typically around –70 mV. You should understand that neither the inside of the neuron nor the outside has a voltage, because a voltage is a *difference* and is meaningful only in comparison with another location. Note that this voltage is quite small—the voltage of a 1.5-V flashlight battery is 25 times greater. No matter; we’re moving information, and very little power is required.

The resting potential is due to the unequal distribution of electrical charges on the two sides of the membrane. The charges come from *ions*, atoms that are charged because they have lost or gained one or more electrons. Sodium ions (Na⁺) and potassium ions (K⁺) are positively charged. Chloride ions (Cl⁻) are negative and so are certain proteins and amino acids that make up the organic anions (A⁻). The fluid outside the neuron is high in Na⁺ and Cl⁻ ions, while there is more K⁺ and A⁻ on the inside (Figure 2.7); there are more negative ions on the inside and more positive ions on the outside, which accounts for the negative resting potential. Next, we will look at why these ions aren’t evenly distributed.

If you remember from grade-school science that there is a tendency for molecules to diffuse from an area of high concentration to one of low concentration, then you are probably wondering how this imbalance in ion distribution can continue to exist. In fact, there are two forces that work to balance the location of the ions. Because of the **concentration gradient**, ions move through the membrane to the side where they are less concentrated. And, as a result of the **electrical gradient**, ions are attracted to the side that is oppositely charged.

In spite of these two forces, a variety of other influences keep the membrane polarized. The force of both gradients would move the anions out, but they are too large to pass through the membrane. At the same time, the anions’ negative charge repels the chloride ions, canceling out the concentration gradient that would otherwise force them inside. The “real players” then turn out to be potassium and sodium ions. There is a slightly greater tendency for potassium to move outward (concentration gradient is stronger than electrical gradient), while the force of both gradients attracts sodium inside. However, ions may cross the membrane only through channels like those in Figure 2.7 that are selective for particular ions. But in the neuron’s resting state, both the sodium channel and the potassium channel are closed, and only a few ions trickle through.

The few ions that do make it through are returned by the **sodium-potassium pump**, which consists of large protein molecules that move sodium ions through the cell membrane to the outside and potassium ions back inside. Its exchange rate of three sodium ions for every two potassium ions helps keep the inside of the membrane more negative than the outside. The pump is a metabolic process, which means that it uses energy; in fact, it accounts for an estimated 40% of the neuron’s energy expenditure. But you will soon see that this energy is well spent, because the resting potential stores the energy to power the action potential.

---

**Figure 2.7**

**Distribution of Ions Inside and Outside the Resting Neuron.**

Ions on the outside are mostly Na⁺ and Cl⁻ ions; inside, the ions are mostly K⁺ ions and organic anions. The arrows represent the sodium-potassium pump, returning sodium ions to the outside and potassium ions to the inside.
The Action Potential. A neuron is usually excited by input that arrives on the neuron's dendrites and cell body from another neuron or from a sensory receptor. An excitatory signal causes a partial depolarization, which means that the polarity in a small area of the membrane is shifted toward zero. This partial depolarization disturbs the ion balance in the adjacent membrane, so the disturbance flows down the dendrites and across the cell membrane. This looks at first like the way the neuron might communicate its messages through the nervous system; however, because a partial depolarization is decremental—it dies out over distance—it is effective only over very short distances. For this reason, the partial depolarization is often called the local potential. Fortunately, the membrane of the axon has unique physical properties. If the local potential exceeds the threshold for activating that neuron, typically about 10 mV more positive than the resting potential, it will cause the normally closed sodium channels in that area to open, which triggers an action potential.

The action potential is an abrupt depolarization of the membrane that allows the neuron to communicate over long distances. The voltage across the resting neuron membrane is stored energy, just as the term potential implies. Imagine countless sodium ions being held outside the neuron against the combined forces of the concentration gradient and the electrical gradient. Opening the sodium channels allows the sodium ions in that area to rush into the axon at a rate 500 times greater than normal; they are propelled into the cell's interior so rapidly that the movement is often described as explosive. A small area inside the membrane becomes fully depolarized to zero; the potential even overshoots to around +30 or +40 mV, making the interior at that location temporarily positive. This depolarization is the action potential.

Just as abruptly as the neuron “fired,” it begins to recover its resting potential. At the peak of the action potential the sodium channels close, so there is no further depolarization. About the same time, the potassium channels begin to open. The positive charge inside the membrane and the force of the concentration gradient combine to move potassium ions out; this outward flow of potassium ions returns the axon to its resting potential. The action potential and recovery require about 1 millisecond (ms; one thousandth of a second) or so to complete; the actual duration varies among individual neurons. Figure 2.8 illustrates these ion movements, while Figure 2.9 shows how the movement of sodium and potassium ions parallels the voltage changes of the action potential and recovery.

The action potential causes nearby sodium channels to open as well. Thus, a new action potential is triggered right next to the first one. That action potential in turn triggers another farther along, creating a chain reaction of action potentials that move through the axon; thus, a signal flows from one end of the neuron to the other. Nothing physically moves down the axon. Instead, a series of events occurs in succession along the axon's length, much as a line of dominoes stood on end knock each other over when you tip the first one. When the action potential reaches the terminals, they pass the signal on to the next
neuron in the chain (or to an organ or muscle). We will talk more about transmission from neuron to neuron later; for now we need to examine the action potential a bit further.

Although the neuron has returned to its resting potential, a number of extra sodium ions remain inside, and there is an excess of potassium ions on the outside. Actually, only the ions in a very thin layer on either side of the membrane have participated in the action potential, so the dislocated ions are able to diffuse into the surrounding fluid. Eventually, though, the ions must be replaced or the neuron cannot continue firing. The sodium-potassium pump takes care of this chore. (Perhaps you can see now why Jim was in such a bad way after his bout with chlordane.)

The action potential differs in two important ways from the local potential that initiates it. First, the local potential is a graded potential, which means that it varies in magnitude with the strength of the stimulus that produced it. The action potential, on the other hand, is ungraded; it operates according to the all-or-none law, which means that it occurs at full strength or it does not occur at all. A larger graded potential does not produce a larger action potential; like the fuse of a firecracker, the action potential depends on the energy stored in the neuron. A second difference is that the action potential is nondecremental; it travels down the axon without any decrease in size, propagated anew and at full strength at each successive point along the way. The action potential thus makes it possible for the neuron to conduct information over long distances.

However, because the action potential is all-or-none, its size cannot carry information about the intensity of the initiating stimulus. One way stimulus intensity is represented is in the number of neurons firing, because a more intense stimulus will recruit firing in neurons with higher thresholds. There is, though, a way in which the individual neuron can encode stimulus strength, as we will see in the discussion of refractory periods.

**Figure 2.9**

**Ion Movements and Corresponding Voltage Changes During the Action Potential.**

(1) Sodium channels open; sodium ions rush in, depolarizing the neuron. (2) Potassium channels begin to open. (3) Sodium channels close; resting potential recovers as potassium ions are repelled outward by positive charge inside. (4) Potassium channels close; resting potential restored.

---

**What is the role of the sodium-potassium pump following an action potential?**

**How is an action potential different from a graded potential?**

---

**Refractory Periods**

Right after the action potential occurs, the neuron goes through the absolute refractory period, a brief time during which it cannot fire again; this occurs because the sodium channels cannot reopen. This delay in responsiveness has two important effects. First, the absolute refractory period limits how frequently the neuron can fire. If a neuron takes a full millisecond to recover to the point where it can fire again, then the neuron can fire, at most, a thousand times a second; many neurons have much lower firing rate limits. A second effect of this recovery period is that the action potential will set off new action potentials only in front of it (the side toward the terminals), not on the side where it has just passed. This is critical, because backward-moving potentials would block responses to newly arriving messages.
A second refractory period plays a role in intensity coding in the axon. The potassium channels remain open for a few milliseconds following the absolute refractory period, and the continued exit of potassium makes the inside of the neuron slightly more negative than usual (the “dip” in Figure 2.9a). During the relative refractory period, the neuron can be fired again, but only by a stronger-than-threshold stimulus. A stimulus that is greater than threshold will cause the neuron to fire again earlier and thus more frequently. The axon encodes stimulus intensity not in the size of its action potential but in its firing rate, an effect called the rate law.
Glial Cells

Glial cells are nonneural cells that provide a number of supporting functions to neurons. The name *glia* is derived from the Greek word for glue, which gives you some idea how the role of glial cells has been viewed in the past. However, glial cells do much more than hold neurons together. One of their most important functions is to increase the speed of conduction in neurons.

Myelination and Conduction Speed

Survival depends in part on how rapidly messages can move through the nervous system, enabling the organism to pounce on its prey, outrun a predator, or process spoken language quickly. The speed with which neurons conduct their impulses varies from 1 to 120 meters (m) per second (s) or about 270 miles per hour. This is much slower than the flow of electricity through a wire, the analogy mistakenly used to describe neural conduction. Because conduction speed is so critical to survival, animals have evolved strategies for increasing it. One way is to develop larger axons, which provide less resistance to the flow of electrical potentials. By evolving motor neurons with a diameter of 0.5 mm, the squid has achieved conduction speeds of 30 m/s compared with 1 m/s in the smallest neurons.

However, conduction speed does not increase in direct proportion to axon size. To reach our four-times-greater maximum conduction speed of 120 m/s, our axons would have to be $42 = 16$ times larger than the squid axon, or 8 mm in diameter! Obviously, your brain would be larger than you could carry around. In other words, if axon size were the only way to achieve fast conduction speed, you would not exist.
Another way to improve conduction speed would be to rely on graded local potentials in the axon, because graded potentials travel down the axon faster than action potentials; however, you will remember that graded potentials die out over distance. Vertebrates (animals with backbones) have developed a best-of-both-worlds solution, called myelination. Glial cells produce myelin, a fatty tissue that wraps around the axon to insulate it from the surrounding fluid and from other neurons. Only the axon is covered, not the cell body. Myelin is produced in the brain and spinal cord by a type of glial cell called oligodendrocytes and in the rest of the nervous system by Schwann cells (see Figure 2.10).

Because there are very few sodium channels under the myelin sheath, action potentials cannot occur there; conduction in myelinated areas is by graded potential (Waxman & Ritchie, 1985). However, myelin appears in segments about 1 mm long, with a gap of one or two thousandths of a millimeter between segments. The gaps in the myelin sheath are called nodes of Ranvier (see Figure 2.10 again). At each node of Ranvier, where the membrane is exposed and there are plenty of sodium channels, the graded potential triggers an action potential; action potentials thus jump from node to node in a form of transmission called saltatory conduction. So myelination and the resulting saltatory conduction increase conduction speed through graded potentials while retaining the benefits of nondecremental action potentials.

Myelination provides an additional boost to conduction speed because the insulating effect of myelin reduces an electrical effect called capacitance, which resists the movement of ions during a graded potential. The overall effect of myelination is the equivalent of increasing the axon diameter 100 times (Koester & Siegelbaum, 2000). And speed is not the only benefit of myelination; myelinated neurons use exceedingly less energy because there is less work for the sodium-potassium pump to do.

Some diseases, such as multiple sclerosis, destroy myelin. As myelin is lost the capacitance rises, reducing the distance that graded potentials can travel before dying out. The individual is worse off than if the neurons had never been myelinated; due to the reduced number of sodium channels, action potentials may not be generated in the previously myelinated area. Conduction slows or stops in affected neurons.

**Other Glial Functions**

During fetal development, one kind of glial cells forms a scaffold that guides new neurons to their destination. Later on, glial cells provide energy to neurons and respond to injury and disease by removing cellular debris. Others contribute to the development and maintenance of connections between neurons. Neurons form seven times as many connections in the presence of glial cells, and if glial cells are removed from a laboratory dish, the neurons start to lose their synapses (Pfrieger & Barres, 1997; Ullian, Sapperstein, Christopherson, & Barres, 2001; see Figure 2.11). We will see later that glia play an important role in neural activity as well. An indication of the importance of glial cells is that as brain complexity increases across species, there is also a progressive increase in the ratio of astrocytes to neurons; astrocytes are the glial cells most intimately involved with neural activity (Figure 2.12).
How Neurons Communicate With Each Other

Before the late 1800s, microscopic examination suggested that the brain consisted of a continuous web. At that point, however, Camillo Golgi developed a new tissue-staining method that helped anatomists see individual neurons by randomly staining some entire cells without staining others (see the discussion of staining methods in Chapter 4). With this technique, the Spanish anatomist Santiago Ramón y Cajal (1937/1989) was able to see that each neuron is a separate cell. The connection between two neurons is called a **synapse**, a term derived from the Latin word that means “to grasp.” The neurons are not in direct physical contact at the synapse but are separated by a small gap called the **synaptic cleft**. Two terms will be useful to us in the following discussion: The neuron that is transmitting to another is called the **presynaptic** neuron; the receiving neuron is the **postsynaptic** neuron (see Figure 2.13).

### Chemical Transmission at the Synapse

Until the 1920s, physiologists assumed that neurons communicated by an electrical current that bridged the gap to the next neuron. The German physiologist Otto Loewi believed that synaptic transmission was chemical, but he did not know how to test his hypothesis. One night Loewi awoke from sleep with the so-

![Image of the synapse](https://example.com/synapse_image)

**Figure 2.13**

The Synapse Between a Presynaptic Neuron and a Postsynaptic Neuron.

Notice the separation between the presynaptic axon terminal and the postsynaptic neuron.

**CONCEPT CHECK**

- How is information conducted in the axon?
- How does the all-or-none law limit information transmission?
- What benefits do the refractory periods provide?
- How does myelin speed up conduction in axons?
olution to his problem (Loewi, 1953). He wrote his idea down so he would not forget it, but the next morning he could not read his own writing. He recalled that day as the most “desperate of my whole scientific life” (p. 33). But the following night he awoke again with the same idea; taking no chances, he rushed to his laboratory. There he isolated the hearts of two frogs. He applied electrical stimulation to the vagus nerve attached to one of the hearts, which made the heart beat slower. Then he extracted salt solution that he had placed in the heart beforehand and placed it in the second heart. If neurons used a chemical messenger, the chemical might have leaked into the salt solution. The second heart slowed, too, just as Loewi expected. Then he stimulated the accelerator nerve of the first heart, which caused the heart to beat faster. When he transferred salt solution from the first heart to the second, this time it speeded up (see Figure 2.14). So Loewi demonstrated that transmission at the synapse is chemical and that there are at least two different chemicals that carry out different functions.

It turned out later that some neurons do communicate electrically by passing ions through channels that connect one neuron to the next; their main function appears to be synchronizing activity in nearby neurons (Bennett & Zukin, 2004). In addition, some neurons release a gas transmitter. Still, Loewi was essentially correct because most synapses are chemical. By the way, if this example suggests to you that the best way to solve a problem is to “sleep on it,” keep in mind that such insight occurs only when people have paid their dues in hard work beforehand!

"I awoke again, at three o’clock, and I remembered what it was . . . I got up immediately, went to the laboratory, made the experiment . . . and at five o’clock the chemical transmission of the nervous impulse was conclusively proved."

—Otto Loewi

**Figure 2.14**

**Loewi’s Experiment Demonstrating Chemical Transmission in Neurons.**

Loewi stimulated the first frog heart. When he transferred fluid from it to the second heart, it produced the same effect there as the stimulation did in the first heart.
At chemical synapses, the neurotransmitter is stored in the terminals in membrane-enclosed containers called vesicles; the term means, appropriately, “little bladder.” When the action potential arrives at the terminals, it opens channels that allow calcium ions to enter the terminals from the extracellular fluid. The calcium ions cause the vesicles clustered nearest the membrane to fuse with the membrane. The membrane opens there and the transmitter spills out and diffuses across the cleft (Figure 2.15).

On the postsynaptic neuron, the molecules of neurotransmitter dock with specialized chemical receptors that match the molecular shape of the transmitter molecules. Activation of these receptors causes ion channels in the membrane to open. Ionotropic receptors open the channels directly to produce the immediate reactions required for muscle activity and sensory processing; metabotropic receptors open channels indirectly and slowly to produce longer-lasting effects. Opening the channels is what sets off the graded potential that initiates the action potential. We will see in the next section that the effect this has on the postsynaptic neuron depends on which receptors are activated.

The chemical jump across the synapse takes a couple of milliseconds; that is a significant slowing compared with transmission in the axon. In a system that places a premium on speed, inserting these gaps in the neural pathway add some compensating benefit. As you will see in the following sections, synapses add important complexity to the simple all-or-none response in the axon.

**Excitation and Inhibition**

Opening ion channels on the dendrites and cell body has one of two effects: It can cause the local membrane potential to shift in a positive direction toward zero, partially depolarizing the membrane, or it can shift the potential farther in the negative direction. Partial depolarization, or hypopolarization, is excitatory and facilitates the occurrence of an action potential; increased polarization, or hyperpolarization, is inhibitory and makes an action potential less likely to occur. The value of excitation is obvious, but inhibition can communicate just as much information as excitation does. Also, the message becomes more complex because input from one source can partially or completely negate input from another. In addition, inhibition helps prevent runaway excitation; one cause of the uncontrolled neural storms that sweep across the brain during an epileptic seizure is a deficiency in an inhibitory transmitter system (Baulac et al., 2001).

What determines whether the effect on the postsynaptic neuron is facilitating or inhibitory? It depends on which transmitter is released and the type of receptors on the postsynaptic neuron. A

---

**Figure 2.15**

A Presynaptic Terminal Releases Neurotransmitter at the Synapse.
particular transmitter can have an excitatory effect at one location in the nervous system and an inhibitory effect at another; however, some transmitters typically produce excitation and others most often produce inhibition. If the receptors open sodium channels, this produces **hypopolarization of the dendrites and cell body, which is an excitatory postsynaptic potential (EPSP)**. Other receptors open potassium channels, chloride channels, or both; as potassium moves out of the cell or chloride moves in, it produces a **hyperpolarization of the dendrites and cell body, or an inhibitory postsynaptic potential (IPSP)**.

At this point, we have only a graded local potential. This potential spreads down the dendrites and across the cell body to the **axon hillock** (where the axon joins the cell body). At the axon, a positive graded potential that reaches threshold will produce an action potential; a negative graded potential makes it harder for the axon to fire. Most neurons fire spontaneously all the time, so EPSPs will increase the rate of firing and IPSPs will decrease the rate of firing (Figure 2.16). So now we have added another form of complexity at the synapse: The message to the postsynaptic neuron can be **bidirectional**, not just on-off.

You should not assume that excitation of neurons always corresponds to activation of behavior or that inhibition necessarily suppresses behavior. An EPSP may activate a neuron that has an inhibitory effect on other neurons, and an IPSP may reduce activity in an inhibitory neuron. An example of this paradox at the behavioral level is the effect of Ritalin. Ritalin and many other medications used to treat hyperactivity in children are in a class of drugs called stimulants, which increase activity in the nervous system. Yet they calm hyperactive individuals and improve their ability to concentrate and focus attention (Cox, Merkel, Kovatchev, & Seward, 2000; Mattay et al., 1996). They probably have this effect by stimulating frontal areas of the brain where activity has been found to be abnormally low (Faigel, Szudajderman, Tishby, Turel, & Pinus, 1995).

Next we will see that the ability to combine the inputs of large numbers of neurons expands the synapse’s contribution to complexity even further.

**Postsynaptic Integration**

The output of a single neuron is not enough by itself to cause a postsynaptic neuron to fire, or to prevent it from firing. In fact, an excitatory neuron may depolarize the membrane of the postsynaptic neuron by as little as 0.2 to 0.4 mV (Kandel & Siegelbaum, 2000b); remember that it takes an approximately 10-mV depolarization to trigger an action potential. However, a typical neuron receives input from around a thou-

**What are the differences between an EPSP and an IPSP?**

**Figure 2.16**

**Effect of Inhibition on Spontaneous Firing Rate.**


**Figure 2.17**

**A Cell Body Virtually Covered With Axon Terminals.**

Source: © Science VU/Lewis-Everhart-Zeevi / Visuals Unlimited
sand other neurons (Figure 2.17); because of the branching of the terminals, this amounts to as many as 10,000 synaptic connections in most parts of the brain and up to 100,000 in the cerebellum (Kandel & Siegelbaum, 2000a).

Because a single neuron has such a small effect, the postsynaptic neuron must combine potentials from many neurons to fire. This requirement is actually advantageous: It ensures that a neuron will not be fired by the spontaneous activity of a single presynaptic neuron, and it allows the neuron to combine multiple inputs into a more complex message. These potentials are combined at the axon hillock in two ways. Spatial summation combines potentials occurring simultaneously at different locations on the dendrites and cell body. Temporal summation combines potentials arriving a short time apart. Temporal summation is possible because it takes a few milliseconds for a potential to die out. Spatial summation and temporal summation occur differently, but they have the same result. Summation is illustrated in Figure 2.18.

As you can see in Figure 2.19, summation combines EPSPs so that an action potential is more likely to occur. Alternatively, summation of IPSPs drives the membrane’s interior even more negative and makes it more difficult for incoming EPSPs to trigger an action potential. When both excitatory and inhibitory impulses arrive on a neuron, they will also summate, but algebraically. The combined effect will equal the difference between the sum of the hypopolarizations and the sum of the hyperpolarizations. Spatial summation of two excitatory inputs and one inhibitory input is illustrated in Figure 2.20. The effect from temporal summation would be similar.

Because the neuron can summate inputs from multiple sources, it rises above the role of a simple message conductor—it is an information integrator; and, using that information, it serves as a decision maker, determining whether to fire or not. Thus, the nervous system becomes less like a bunch of telephone lines and more like a computer. In subsequent chapters, you will come to appreciate how important the synapse is in understanding how we see, how we learn, and how we succumb to mental illness.
Terminating Synaptic Activity

The neurotransmitter's story does not end when it has activated the receptors. Usually, the transmitter must be inactivated to prevent it from "locking up" a circuit that must respond frequently, or from leaking over to other synapses and interfering with their function. Typically, the transmitter is taken back into the terminals by a process called reuptake; it is repackaged in vesicles and used again. At some synapses, the transmitter in the cleft is absorbed by glial cells. The neurotransmitter acetylcholine (ACh), on the other hand, is deactivated by acetylcholinesterase, an enzyme that splits the molecule into its components of choline and acetate. Choline is then taken back into the terminals and used to make more acetylcholine.

Controlling how much neurotransmitter remains in the synapse is one way to vary behavior, and many drugs capitalize on this mechanism. Cocaine blocks the uptake of dopamine; some antidepressant medications block the reuptake of serotonin, norepinephrine, or both, while others (MAO inhibitors) prevent monoamine oxidase from degrading those transmitters as well as dopamine and epinephrine; and drugs for treating the muscular disorder myasthenia gravis increase ACh availability by inhibiting the action of acetylcholinesterase.

Figure 2.20

Spatial Summation of Excitatory and Inhibitory Potentials.

Note that inhibitory potentials cancel out excitatory potentials of equal strength (and vice versa).
Regulating Synaptic Activity

We have been describing a system that amounts to neuron A stimulates neuron B, neuron B stimulates neuron C, and so on. However, such a simple system cannot transmit the complex information required to solve a math equation, write a symphony, or care for a newborn baby. Not only that, but as messages flow from neuron to neuron, activity would soon drift out of control; some activity would fade out, while other activity would escalate until it engulfed an entire area of the brain. A nervous system that controls complex behavior must have several ways for regulating its activity.

One of the ways is through axoaxonic synapses. The synapses described so far are referred to as axodendritic and axosomatic synapses, because their targets are dendrites and cell bodies. At axoaxonic synapses, a third neuron releases transmitter onto the terminals of the presynaptic neuron (see #1 in Figure 2.21). The result is presynaptic excitation or presynaptic inhibition, which increases or decreases, respectively, the presynaptic neuron’s release of neurotransmitter onto the postsynaptic neuron. One way an axoaxonic synapse adjusts a presynaptic terminal’s activity is by regulating the amount of calcium entering the terminal, which, you will remember, triggers neurotransmitter release.

Neurons also regulate their own synaptic activity in two ways. Autoreceptors on the presynaptic terminals sense the amount of transmitter in the cleft; if the amount is excessive, the presynaptic neuron reduces its output (Figure 2.21, #2). Postsynaptic neurons participate in regulation of synaptic activity as well. When there are unusual increases or decreases in neurotransmitter release, postsynaptic receptors change their sensitivity or even their numbers to compensate (Figure 2.21, #3). You will see in a later chapter that receptor changes figure prominently in psychological disorders such as schizophrenia.

We are now learning that glial cells also contribute to the regulation of synaptic activity. They surround the synapse and prevent neurotransmitter from spreading to other synapses. More important, they sometimes absorb neurotransmitter in the synaptic cleft and recycle it for the neuron’s reuse (Figure 2.22); they influence synaptic transmission by granting or withholding transmitter absorption (Oliet, Piet, & Poulain, 2001). They even release...
the neurotransmitter glutamate, in response to transmitter levels in the synapse; this stimulates the presynaptic terminal to enhance or depress further transmitter release (Newman, 2003).

**Neurotransmitters**

Table 2.2 lists several transmitters, grouped according to their chemical structure. This is an abbreviated list; there are other known or suspected transmitters, and there are doubtless additional transmitters yet to be discovered. This summary is intended to illustrate the variety in neurotransmitters and to give you some familiarity with the functions of a few of the major ones. You will encounter most of them again as we discuss various behaviors in later chapters.

Having a variety of neurotransmitters multiplies the effects that can be produced at synapses; the fact that there are different subtypes of the receptors adds even more. For example, two types of receptors detect acetylcholine: the nicotinic receptor, so called because it is also activated by nicotine, and the muscarinic receptor, named for the mushroom derivative that can stimulate it. Nicotinic receptors are excitatory; they are found on muscles and, in lesser numbers, in the brain. Muscarinic receptors are more frequent in the brain, where they have an excitatory effect at some locations and an inhibitory one at others. Other transmitters have many more receptor subtypes than acetylcholine does.

For decades, neurophysiologists labored under the erroneous belief, known as Dale’s principle, that a neuron was capable of releasing only one neurotransmitter. We learned only fairly recently that many neurons ply their postsynaptic partners with two to four and perhaps even more neurotransmitters. Since then, most researchers have thought that the combination invariably consisted of a single fast-acting “traditional” neurotransmitter and one or more slower-acting neuropeptides that prolong and enhance the effect of the main transmitter (Hökfelt, Johansson, & Goldstein, 1984). Peptides are chains of amino acids (longer chains are called proteins); neuropeptides, of course, are peptides that act as neurotransmitters.

Recent studies have found that some neurons release two fast transmitters (Rekling, Funk, Bayliss, Dong, & Feldman, 2000). Even more surprising, we have learned that the same neuron can release both an excitatory transmitter and an inhibitory transmitter (Duarte, Santos, & Carvalho, 1999; Jo & Schlichter, 1999). It appears that the two types of transmitters are released at different terminals (Duarte et al., 1999; Sulzer & Rayport, 2000). This corelease suggests that a neuron can...
### Table 2.2  Some Representative Neurotransmitters

<table>
<thead>
<tr>
<th>Neurotransmitter</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetylcholine</td>
<td>Transmitter at muscles; in brain, involved in learning, etc.</td>
</tr>
<tr>
<td><strong>Monamines</strong></td>
<td></td>
</tr>
<tr>
<td>Serotonin</td>
<td>Involved in mood, sleep, and arousal, and in aggression, depression, obsessive-compulsive disorder, and alcoholism.</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Contributes to movement control and promotes reinforcing effects of abused drugs, food, and sex; involved in schizophrenia and Parkinson’s disease.</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>A hormone released during stress. Functions as a neurotransmitter in the brain to increase arousal and attentiveness to events in the environment; involved in depression.</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>A stress hormone related to norepinephrine; plays a minor role as a neurotransmitter in the brain.</td>
</tr>
<tr>
<td><strong>Amino Acids</strong></td>
<td></td>
</tr>
<tr>
<td>Glutamate</td>
<td>The principal excitatory neurotransmitter in the brain and spinal cord. Vitally involved in learning, and implicated in schizophrenia.</td>
</tr>
<tr>
<td>Gamma-aminobutyric acid (GABA)</td>
<td>The predominant inhibitory neurotransmitter. Its receptors respond to alcohol and the class of tranquilizers called benzodiazepines. Deficiency in GABA or receptors is one cause of epilepsy.</td>
</tr>
<tr>
<td>Glycine</td>
<td>Inhibitory transmitter in the spinal cord and lower brain. The poison strychnine causes convulsions and death by affecting glycine activity.</td>
</tr>
<tr>
<td><strong>Peptides</strong></td>
<td></td>
</tr>
<tr>
<td>Endorphins</td>
<td>Neuromodulators that reduce pain and enhance reinforcement.</td>
</tr>
<tr>
<td>Substance P</td>
<td>Transmitter in neurons sensitive to pain.</td>
</tr>
<tr>
<td>Neuropeptide Y</td>
<td>Initiates eating and produces metabolic shifts.</td>
</tr>
<tr>
<td><strong>Gas</strong></td>
<td></td>
</tr>
<tr>
<td>Nitric Oxide</td>
<td>One of two known gaseous transmitters, along with carbon monoxide. Can serve as a retrograde transmitter, influencing the presynaptic neuron’s release of neurotransmitter. Viagra enhances male erections by increasing nitric oxide’s ability to relax blood vessels and produce penile engorgement.</td>
</tr>
</tbody>
</table>
act as a two-way switch (Jo & Schlichter, 1999); one example is in cells in the eye that produce excitation when a viewed object moves in one direction and inhibition when movement is in the opposite direction (Duarte et al., 1999).

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**Computer Models and Neural Networks**

Underlying our discussion has been the assumption that we can explain behavior by understanding what neurons do. But we cannot make good on that promise as long as we limit ourselves to talking about simple chains of neurons. However, researchers have found the neural connections involved in learning, vision, and movement to be discouragingly complex. Early attempts to model brain activity with computers by writing programs that told the computer what to do had little success. Now researchers are turning to programs that, instead of mimicking behavior, attempt to duplicate the structure of the brain itself.

*Artificial neural networks*, which consist of simulated neurons that carry out cognitive-like functions, learn how to perform the task like we do, by trial and error. An artificial neural network consists of a layer that receives input (say, from a keyboard or a video camera), one or more “hidden” layers where the processing occurs, and a layer that sends output to a printer or a robot arm or other device. The hidden layer has simulated “neurons” that are all connected to each other, as well as to the input and output layers (see Figure 2.23). The researcher “trains” the network...
network by presenting it with a series of inputs and giving it the correct output to compare with its own output. Usually the network sends feedback to the layers above, and the network adjusts the strength of the connections between the neurons. At first the network’s performance is random, but it improves with practice. For example, NETtalk (Sejnowski & Rosenberg, 1987), designed to read and speak English text, initially produced random sounds, which were replaced with babbling and then pseudowords; but after just 10 training trials, the speech was intelligible and sounded like a small child’s. Artificial neural networks have other features in common with brains. Information is distributed throughout the network rather than strictly localized, so “damage” does little harm until it becomes extensive. Yet distributed storage does not “use up” the neurons because a neuron can participate in several functions; this efficiency allows NETtalk to function with only 300 neurons, reminiscent of the tremendous storage capacity of the brain.

These networks mimic human and animal behavior surprisingly well. One program simulated rats learning to find an escape platform hidden just below the surface of murky water (Brown & Sharp, 1995). When the platform was deleted from the program, the virtual rats would “swim” past the platform’s original location, then turn and swim back in the other direction. Thanks to neural networks, robots are learning to walk over varied terrain with a humanlike gait (Manoonpong, Geng, Kulvicius, Porr, & Wörgötter, 2007) and Japanese androids are developing incredibly human gestures and facial expressions just by “watching” human models (Figure 2.24; Matsui, Minato, MacDorman, & Ishiguro, 2005). Artificial neural networks have also been used to simulate the brain’s performance in analyzing visual scenes (Lau, Stanley, & Dan, 2002) and in locating sounds in space (Furukawa, Xu, & Middlebrooks, 2000).

In the long run, artificial neural networks should help us understand how the brain carries out these tasks. The real networks are very complex and inaccessible, but the artificial networks allow us to test hypotheses experimentally; then the researcher can often learn just how the network solved its problem by examining the new configuration of neurons in the hidden layer. For example, when researchers examined the network of a robot after it learned to navigate through its environment, they found that it had spontaneously developed specialized neurons that functioned like so-called place cells found in the brains of rats (Fleischer, Gally, Edelman, & Krichmar, 2007).

While we know very little about the real neural networks, I find that they provide a useful way to think about mental processes. The next time you are trying to remember a person’s name that is “on the tip of your tongue,” imagine your brain activating individual components of a neural network until one produces the name you’re looking for. If you visualize the person’s face as a reminder, imagine that the

Figure 2.23
A diagrammatic representation of a simple neural network.

Figure 2.24
A Life-Like Android.
Artificial neural networks allow the human-looking androids created by Hiroshi Ishiguro of Osaka University to learn lifelike facial expressions and gestural movements by “watching” human models. The real-life Hiroshi is on the left.
name and the image of the face are stored in the same or in related networks, so that activating one memory activates the other. We will talk more about how synaptic connections are formed to create memories when we get to Chapter 12.

**CONCEPT CHECK**

- How is information transmitted at the synapse?
- It can be said that integration transforms neurons from a “telephone line” to a computer. Explain.
- What difference would it make if there were no regulation of activity at the synapse?
- What is Dale’s principle, and in what way is it incorrect?
- How successful have artificial neural networks been in simulating human brain activity? What does this tell us about whether they work the same way the brain does?

**In Perspective**

It is impossible to understand the brain and impossible to understand behavior without first knowing the capabilities and the limitations of the neuron. Although more complexity is added at the synapse, a relatively simple off-or-on device is the basis for our most sophisticated capabilities and behaviors. However, what happens at the individual neuron is not enough to account for human behavior. Some researchers are using artificial neural networks to understand how neurons work together to produce thought, memory, emotion, and consciousness. In the next chapter, you will learn about some of the functional structures in the brain that are formed by the interconnection of neurons.

**Summary**

**The Cells That Make Us Who We Are**

- There are three major kinds of neurons: motor neurons, sensory neurons, and interneurons. Though they play different roles, they have the same basic components and operate the same way.
- The neural membrane is electrically polarized. This polarity is the resting potential, which is maintained by forces of concentration and electrical gradient, as well as by the sodium-potassium pump.
- Polarization is the basis for the neuron’s responsiveness to stimulation, in the form of the graded potential and the action potential.
- The neuron is limited in firing rate by the absolute refractory period and in its ability to respond to differing strengths of stimuli by the all-or-none law. More intense stimuli cause the neuron to fire earlier during the relative refractory period, providing a way to encode stimulus intensity (the rate law).
- Glial cells provide the myelination that enables neurons to conduct rapidly while remaining small. They also help regulate activity in the neurons and provide several supporting functions for neurons.
How Neurons Communicate With Each Other

- Transmission from neuron to neuron is usually chemical in vertebrates, involving neurotransmitters released onto receptors on the postsynaptic dendrites and cell body.
- The neurotransmitter can create an excitatory postsynaptic potential, which increases the chance that the postsynaptic neuron will fire; or it can create an inhibitory postsynaptic potential, which decreases the likelihood of firing.
- Through temporal and spatial summation, the postsynaptic neuron integrates its many excitatory and inhibitory inputs.
- Regulation of synaptic activity is produced by axoaxonic synapses from other neurons, adjustment of transmitter output by autoreceptors, and change in the number or sensitivity of postsynaptic receptors.
- Leftover neurotransmitter may be broken down, taken back into the presynaptic terminals, or absorbed by glial cells.
- The human nervous system contains a large number of neurotransmitters, detected by an even greater variety of receptors. A neuron can release combinations of two or more neurotransmitters.
- Several functions of the human brain are being simulated by computers, and artificial neural networks are helping us understand the brain’s neural networks.

Study Resources

For Further Thought

- What would be the effect if there were no constraints on the free flow of ions across the neuron membrane?
- What effect would it have on neural conduction if the action potential were decremental?
- Sport drinks replenish electrolytes that are lost during exercise. Electrolytes are compounds that separate into ions; for example, sodium chloride (table salt) dissociates into sodium and chloride ions. What implication do you think electrolyte loss might have for the nervous system? Why?
- Imagine what the effect would be if the nervous system used only one neurotransmitter.
- How similar to humans do you think computers are capable of becoming?

Testing Your Understanding

1. Describe the ion movements and voltage changes that make up the neural impulse, from graded potential (at the axon hillock) to recovery.
2. Discuss the ways in which the synapse increases the neuron’s capacity for transmitting information.
3. Describe how artificial neural networks function like the brain and what humanlike behaviors they have produced.

Select the best answer.

1. The inside of the neuron is relatively poor in __ ions and rich in __ ions.
   a. chloride, phosphate  
   b. sodium, potassium  
   c. potassium, sodium  
   d. calcium, sodium
2. The rate law
   a. explains how the intensity of stimuli is represented.
   b. does not apply in neurons outside the brain.
   c. describes transmission in myelinated axons.
   d. describes the process of postsynaptic integration.

3. Without the sodium-potassium pump, the neuron would become
   a. more sensitive because of accumulation of sodium ions.
   b. more sensitive because of accumulation of potassium ions.
   c. overfilled with sodium ions and unable to fire.
   d. overfilled with potassium ions and unable to fire.

4. There is a limit to how rapidly a neuron can produce action potentials. This is due to
   a. inhibition.
   b. facilitation.
   c. the absolute refractory period.
   d. the relative refractory period.

5. Saltatory conduction results in
   a. less speed and the use of more energy.
   b. greater speed with the use of less energy.
   c. less speed but with the use of less energy.
   d. greater speed but with the use of more energy.

6. General anesthetics open potassium channels, allowing potassium ions to leak out of the neuron. This
   a. increases firing in pain-inhibiting centers in the brain.
   b. increases firing in the neuron until it is fatigued.
   c. hypopolarizes the neuron, preventing firing.
   d. hyperpolarizes the neuron, preventing firing.

7. When the action potential arrives at the terminal button, entry of __ ions
   stimulates release of transmitter.
   a. potassium
   b. sodium
   c. calcium
   d. chloride

8. All the following neurotransmitters are deactivated by reuptake except
   a. acetylcholine.
   b. norepinephrine.
   c. serotonin.
   d. dopamine.

9. An inhibitory neurotransmitter causes the inside of the postsynaptic neuron to become
   a. more positive.
   b. more negative.
   c. more depolarized.
   d. neutral in charge.

10. Excitatory postsynaptic potentials are typically produced by movement of __ ions, whereas
    inhibitory postsynaptic potentials are typically produced by movement of __ ions.
    a. potassium; sodium or chloride
    b. potassium; sodium or calcium
    c. sodium; calcium or chloride
    d. sodium; potassium or chloride

11. Which of the following is not an example of regulation of synaptic activity?
    a. A neuron has its synapse on the terminals of another and affects its transmitter release.
    b. Autoreceptors reduce the amount of transmitter released.
    c. A presynaptic neuron inhibits a postsynaptic neuron.
    d. Postsynaptic receptors change in numbers or sensitivity.

12. The graph below shows three graded potentials occurring at the same time.

   Assume that the resting potential is –70 mV and that each graded potential individually produces a 5-mV change. What is the membrane's voltage after the graded potentials arrive?
   a. –65 mV
   b. –70 mV
   c. –75 mV
   d. +75 mV

13. The presence of synapses in a neuron chain provides the opportunity for
    a. increases in conduction speed.
    b. modification of neural activity.
    c. two-way communication in a pathway.
    d. regeneration of damaged neurons.

    a. rely on prewired “neural” connections.
    b. solve problems in a couple of trials by insight.
    c. are preprogrammed.
    d. learn how to carry out the task themselves.
The following Web sites are coordinated with the chapter’s content. Their numbers correspond to the numbers with the icons you saw throughout the chapter.

1. Neuroscience for Kids (don’t be put off by the name!) has a review of the resting and action potentials and an animation of their electrical recording at http://faculty.washington.edu/chudler/ap.html

2. The Cajal Medical and Scientific Illustration site has colorful artist’s renderings of neurons, synapses, and neuron membranes. You can also listen in on neurons whose action potentials have been amplified and transformed into sounds. Available at http://cajal.com/docs/nbuttons.htm

3. You can see for yourself how lifelike the Japanese androids are in a video at www.ed.ams.eng.osaka-u.ac.jp/development/mpeg/ReplieeQ1expo_DemoSelfIntroduction.mpg

You can see a video of a robot learning to walk up a ramp (a difficult task for a robot) at www.nld.ds.mpg.de/~poramate/RUNBOT/ManoonpongMovieS2.mpeg

Another site with informative articles and links to research sites is the American Association for Artificial Intelligence page on neural networks at www.aaai.org/ATOPics/html/neural.html

For Further Reading

1. *Synaptic Self* by Joseph LeDoux (Penguin Books, 2002) takes the position that “your ‘self,’ the essence of who you are, reflects patterns of interconnectivity between neurons in your brain.” A good read by a noted neuroscientist.

2. *Neurons and Networks: An Introduction to Behavioral Neuroscience* by John E. Dowling (Harvard University Press, 2001). Written by a well-known Harvard neuroscientist (you will see some of his work in Chapter 10), the first half of this book elaborates on the topics in this chapter. According to one student, the book “goes into depth without becoming murky.”


4. “Debunking the Digital Brain” (*Scientific American*, February 1997). This brief feature article describes the view of Christof Koch at the California Institute of Technology that neurons are much more complicated than we give them credit for, which has implications for computer simulation.

5. *The Computational Brain* by Patricia Churchland and Terrence Sejnowski (Bradford Books, 1994). This treatment of the attempt to model brain functioning with computers is by two of the foremost experts in the field; it is a bit difficult, but lively and well written.
### Key Terms

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“Garrett delivers...using every day applications to provide a context for making dry material much more interesting. I really like how he organizes the book sections—for instance that the body senses are treated with movement, and drug addiction with motivation.”

—Patricia Bach, Illinois Institute of Technology

“Much of the material is current, with many new references throughout the chapters and topics that are relevant and prominent in the media. The topics covered and the additions indicate that this is a very comprehensive, very current, and interesting text.”

—Mindy Miserendino, Sacred Heart University

“The key issues in teaching biopsychology are getting students focused on the material and making it interesting enough for them to be willing to read the text. Garrett does a good job of assisting with this process of making material meaningful and practical to students.”

—Susan A. Todd, Bridgewater State College