CHAPTER 2

Memory and the Brain

The word *brain* really means different things to different people. In everyday usage, the word *brain* is nearly synonymous with the word *mind*. We say that we have something “in our brain that we cannot get out,” meaning we have been thinking about something. You call someone a “brain” if you think that his or her intelligence is that person’s chief characteristic. However, underlying this metaphor is the certainty that the brain is the biological organ responsible for thinking, memory, reasoning, and language. In this chapter, we will explore the science of how the brain produces memory.

For a neurosurgeon, the brain is a mass of soft tissue inside the head that has to be handled very carefully when damaged. The brain itself has no pain receptors, so neurosurgeons are less concerned about anesthesia than other doctors. However, the brain is surrounded and infused with millions of blood vessels, so surgeons must be very careful when probing around the brain, lest they accidentally induce a hemorrhage. Neurosurgeons understand the critical nature of the human brain for what it is to be human, yet for a surgeon, its identity is a biological tissue.

For a cognitive neuroscientist, the brain is a complex assortment of separate areas and regions, each of which has its own unique function. For example, the frontal regions are for planning, thinking, and monitoring, while the back of the brain processes vision. Viewed this way, the brain is not really one organ but many dozens of distinct regions each with its own appearance, its own micro-anatomy, and its own function. In each way of looking at the brain, however, is the assumption that the biological organ located inside the skull is the organ directly involved in memory, language, and thought. It was not always thus. Aristotle famously mistook the heart as the organ of thought and thought that the brain was merely for cooling the blood. This theory has long since been discredited; any physician who advanced such a notion today would find himself or herself without patients very quickly.

We live in an age in which we are at the cusp of tremendous breakthroughs in our understanding of the relation of brain and cognition (Sylwester, 2005). Recent technological advances have provided unrivaled methods for examining how the brain works and how memories are formed, stored, and retrieved. Most of these advances come from neuroimaging technology, which allows us to peer inside the normal functioning brain. Despite these advances, however, much still remains a mystery, and neuroscientists will be researching the correlation between brain function and memory processes for many years to come.
Nonetheless, this chapter would have been much less detailed if it had been written 10 years ago. We are in the midst of a neuroimaging revolution, and we know much about brain function because of it. And for a number of reasons, research on the cognitive neuroscience of memory has been leading the way.

OLD QUESTIONS, NEW ANSWERS

To introduce the neuroscience of memory, we will start with one of the older questions in this area—namely, where in the brain are memories stored? This question is of interest for a number of reasons. First, it is a deeply philosophical question; how is it that this brain stuff (shortly to be called neurons) can contain information about the taste of oranges, the name of the 10th president of the United States, and the image of one’s long-departed great-grandmother? Second, it is an important practical question. If there are certain areas of the brain that store memories, then we need to respect these areas when probing the brain during neurosurgery. The consensual wisdom on this topic for some time is that memories are not stored in any particular location in the brain but are distributed throughout the brain. The memory of your great-grandmother is stored in many parts of the brain—her image is in your visual cortex, her voice is in your auditory cortex, and the emotions from childhood her memory elicits are in yet other areas of the cortex. Fourth, this consensual wisdom has been challenged. We will briefly review some data that support the idea that specific areas of the brain are for specific memories. These data are based on neuroimaging techniques using the newest and most sophisticated technology.

Many years ago, Karl Lashley labeled this question the “search for the engram”—the engram being the physical unit of storage of a memory (Schacter, 2001). For example, when you learn that “Bratislava is the capital of Slovakia,” there must be some change in the brain that marks this new information. If somebody asks you what the capital of Slovakia is, the question activates the engram, which stores the association between the names “Bratislava” and “Slovakia.” Lashley suspected that there might be specific cells or groups of cells that transform when new information has been acquired. He spent his entire career looking for these memory-specific cells but never found any. Finally, at the end of his career, Lashley was forced into concluding that there are no engrams—that memory representation occurs because of a connection between disparate areas in the brain. Nowadays, there is good evidence to support this idea. The Conway et al. (2003) study discussed in Chapter 1, which shows that visual areas of the brain are activated during autobiographical recall, supports this idea. Thus, the current view is that stored memories are distributed throughout the brain and have more to do with connections across spatially separate areas of the brain than in any specific area. Thus, the memory of your great-grandmother is the result of axonal connections between areas in the visual brain, auditory brain, emotion centers, and perhaps many others.

This was the conventional wisdom from Lashley’s time to the present. However, Quiroga, Reddy, Kreiman, Koch, and Fried (2005), using functional magnetic resonance imaging
(fMRI) technology, which was never available to Lashley, apparently has found specific areas in the brain that seem to support very specific knowledge structures. In Quiroga et al.’s studies, people see photographs or printed names of various celebrities while the fMRI is scanning their brains. In general, the photographs elicit greater responses in the visual areas of the brain, whereas the printed names evoke responses in areas of the brain involved in reading. But embedded in the temporal lobe, Quiroga et al. found areas of the brain that respond specifically to information about particular people. That is, these areas of the brain respond selectively to either the picture or the name of one celebrity but not another celebrity. For example, many of Quiroga et al.’s participants actually had “Halle Berry” areas of the brain, that is, neurons that respond to her name or her photograph, even across a range of characters from movies. Nearby the Halle Berry is a “Harrison Ford” area, which responds to his name and his picture, but much less so than to Halle Berry. The specificity of these areas to the recognition of individual people makes it look like there just may be engrams after all. There are many who question these data. In fact, many think that there are other explanations of Quiroga et al.’s data and that citing their findings as support of an engram theory is premature. However, Quiroga et al.’s study has definitely raised the possibility that Lashley’s search may not have been in vain. There may be engrams after all. Still, most researchers think that memory storage is widely distributed across the brain and that distributed models such as that of Farah and McClelland (1991) offer better explanations.

BRAIN AND MEMORY

Understanding how the brain forms, stores, and retrieves memory has tremendous practical applications in educational and medical settings because learning is such an important human process. First, consider the medical implications of understanding brain-memory relationships. In particular, knowing how the brain forms memories means that we may be better able to intervene in memory loss, especially the memory loss associated with pathological aging, such as Alzheimer’s disease. Alzheimer’s disease is one of many dementia-type illnesses that are more common in older adults than they are in younger adults. Memory is the first deficit detected in this disease. Alzheimer’s disease (go to www.sagepub.com/schwartz for more information) is a terminal illness whose initial signature is the development of amnesic (memory loss) symptoms. It is a disease that affects the brain, clearly illustrating the brain-memory relation. Early Alzheimer’s patients have trouble learning new information and retrieving recent events. Later stage Alzheimer’s involves the loss of knowledge of the past and eventually the identity of close relatives. Understanding the neural processes of memory will help medical research to be able to prevent Alzheimer’s or alleviate the symptoms of those with the disease. Preventing Alzheimer’s will have enormous consequences for untold millions and relieve fear among many who would never develop it.
Normal aging is also characterized by memory loss, albeit mild compared to the ravages of Alzheimer’s. Much of this loss is correlated to changes in the brain. Therefore, even for normal older adults, understanding brain-memory relationships could wind up benefiting them.

Memory deficits are also a common symptom of traumatic brain injuries (TBIs; for more information, go to www.sagepub.com/schwartz). TBIs occur when the brain violently and suddenly hits a hard object, such as an automobile windshield. These are usually called closed-head injuries because the windshield seldom completely cracks the skull. TBIs often can occur in open-head injuries as well, such as when the brain is penetrated by an object such as a bullet. In many cases, the closed-head injury can result in greater damage to the brain than the open-head injury. According to the CDC (Centers for Disease Control and Prevention, 2010), 1.7 million people suffer TBIs every year. Most of these are minor, but 50,000 a year are fatal.

The biggest source of TBIs is from motor vehicle crashes. In fact, 17% of TBIs result from motor vehicle crashes (Centers for Disease Control and Prevention, 2010). TBIs are a leading cause of death among young adults, particularly among young male adults. In many severe auto accidents, the head strikes the windshield, causing damage to the prefrontal lobes of the brain. This damage to the frontal lobe can result in long-term deficits in memory, emotional complications, and difficulties in planning and organization. In addition, temporal lobe areas may also be damaged, causing further memory complications. The counter-coup (that is, the blow to the back of the head) may bring damage to the occipital lobe, resulting in visual deficits as well. Better understanding of the nature of memory in the brain could bring much-needed relief to these individuals as well. In the near term, however, buckle up and don’t disconnect your airbag!

The care and treatment of patients with brain damage falls in the domain of clinical neuropsychology. Since most auto accident victims are young adults with long lives in front of them, the treatment and rehabilitation of TBIs is of tremendous social importance in our autocentric culture. Therefore, clinical neuropsychology focuses on rehabilitation and restoration of cognitive skills for auto accident victims. However, due to the usual pattern of widespread damage in an auto accident, auto accident victims are seldom used in research examining the relation of brain and behavior.

Alzheimer’s and TBIs are two major sources of individuals with memory-related brain damage. But there are other sources as well. Strokes affect the brains of many older adults, as do tumors. Each of these may create deficits in memory. We will return to each of these phenomena in this book—as understanding memory deficits are an important part of memory science. But the primary goal of this chapter is to understand how the brain processes result in the cognitive processes of memory. It is therefore important to begin with an understanding of the underlying structure in the brain.
Our brains contain billions of microscopic cells called neurons. Neurons are biological cells that specialize in the transmission and retention of information (see Figure 2.1). As such, neurons are the basic building block of both our brain and our entire nervous system. Neurons form huge networks of communicating cells in the brain and also connect to neurons in the nerves and muscles of the body. They innervate all of the sensory systems and muscular systems and allow us to move, see, think, and remember. Understanding memory or any other cognitive process requires a fundamental understanding of how neurons transmit information. To understand how they transmit information, you must first understand their basic anatomy.

Figure 2.1  A typical neuron.

Like all biological cells, the neuron contains a nucleus, which houses the individual’s chromosomes. The chromosomes contain the genes, which contain each individual’s DNA. Surrounding the nucleus is the soma or cell body. The soma contains all the apparatuses that keep the cell working, such as mitochondria and other organelles. In this way, neurons are similar to all other cells of the human body. What make neurons unique are the fibers that extend outward from the soma. These fibers allow neurons to conduct the transmission of information from one part of the brain or nervous system to another part of the brain or nervous system. There are two types of fibers, one that leads into the neuron and one that leads out of the neuron. Each of these fibers conducts electricity, although each fiber does so in a different manner. Indeed, the transmission of information in the brain occurs through small electric currents racing through the neurons of the brain.

The part of the neuron that receives information from other neurons is the **dendrite**. Any neuron may have many hundreds of dendrites, each one receiving different pulses from other neurons. Some of these pulses may make the voltage higher within the cell, and some of the pulses may make the voltage lower in the cell. The voltage refers to the electrical potential of the cell. The various inputs sum at the soma and determine the electrical state of that neuron at that particular instant of time. This sum total of electric input at any given time can then cause that particular cell to start a signal to other cells. The message leaves the cell via the other unique fiber in the neuron.

Each neuron has only one **axon**, which transmits messages to other neurons. A neuron has only one axon, but it may branch out and be connected to many hundreds of other neurons. But each of those neurons gets the same electrical pulse as all the others because the cell has only one axon. Transmission in an axon is an electrochemical process called an **action potential**. This is because transmission of electricity along the axon is not simply like a wire. Chemical processes keep the message strong regardless of the length of the axon.

The axon of one neuron does not actually touch the dendrite of the next neuron. A gap exists between the two neurons, called the **synapse**. The synapse is extremely small—but electricity does not pass from the axon of one cell to the dendrite of the next. Instead, the transfer of information from one neuron to the next occurs chemically, rather than electrically. The axon does so in the following manner. At the end of the axon are little nodules called **terminal buttons**. When the electrical signal reaches the terminal buttons, the signal triggers the terminal buttons to release **neurotransmitters**, which...
are chemicals (such as dopamine) that cross the synapse and induce an electric flow in the next cell (see Figures 2.2 and 2.3). Thus, the flow of information in the neurons is both chemical and electrical.

A few important things to note about this process are as follows. First, transmission of information in the dendrites is electrical. The longer dendrites will show a greater loss of electrical power than will shorter dendrites. This is similar to the transmission of electricity through power lines. More energy is lost when the electricity is transported over long distances than over short distances. As such, dendrites tend to be very short. Because the flow of information in the dendrite is electrical, it is also extremely fast. Indeed, in terms of the size of biological organisms, transmission in the dendrites is said to be instantaneous.

Transmission of information in the axons is electrochemical. It is electrical over very short segments but then gets a power boost (called action potentials) via a chemical process as it moves down the axon. This allows axons to be quite long (indeed, you have 1-meter-long axons going up your spinal cord), as the action potentials keep the electric potential constant as it flows along the axon. However, because of these action potentials, information

Figure 2.2 Components of the neuron.

flow in the axon is relatively slow (sometimes as slow as 10 meters per second). Incidentally, it is likely the slowness of axon transmission that caused big animals such as dinosaurs to evolve a second “brain” (really a large nerve ganglion) in their tail. Finally, transmission
of information is completely chemical at the synapse when neurotransmitters carry the information from one axon to the next dendrite. This transmission also slows down the general speed of neural transmission.

Most axons are coated with a myelin sheath, which speeds the flow of information in the axons. Myelin is a fatty substance, which acts as an insulator would to a copper wire. The myelin, therefore, allows the electric signal to travel faster along the axon. The loss of myelin along human axons is associated with the disease known as multiple sclerosis (MS). The loss of movement and coordination seen in MS is because of the slowdown of information flowing through the axons.

**Neurotransmitters**

The brain and nervous system make use of many different neurotransmitters depending on the type of neuron and the part of the brain. Neurotransmitters are proteins produced by the nervous system. To be classified as a neurotransmitter, a chemical must bridge the synapse and induce an electric current in a dendrite. Neurotransmitters may either excite the dendrite or inhibit it, and the same neurotransmitter may be excitatory or inhibitory in different neural circuits. Neurotransmitters that increase activity in the neuron are said to be excitatory. In contrast, neurotransmitters that decrease activity in the neuron are said to be inhibitory. That is, inhibition causes the neuron to make fewer action potentials rather than more. Common neurotransmitters include dopamine, acetylcholine, serotonin, gamma-aminobutyric acid (GABA), and norepinephrine. GABA is the most commonly used neurotransmitter in the human brain. Acetylcholine is used by neurons that innervate and control our muscles.

If some of these chemicals’ names seem familiar to you, it is because of their importance. Many neurological diseases are associated with malfunction of the systems that produce these chemicals. Moreover, many psychiatric conditions are treated by altering the process by which neurotransmitters are produced in the body. Finally, orally consumed drugs can alter the functioning of many of these neurotransmitters. Indeed, many of the drugs we consume (both legal and illegal) affect the function of the brain by changing the chemistry at the synapse. This section will provide just a few examples of this, but there are many more.

In Parkinson’s disease, for example, a part of the brain (the substantia nigra) is no longer able to produce enough dopamine. This loss of dopamine then results in the characteristic disorders of movement associated with Parkinson’s. Patients with Parkinson’s disease may have difficulties initiating movements, frozen facial expressions, and tics about which they are not aware. If left untreated, the symptoms get worse as the disease progresses. However, there are medicines available that can control the symptoms—at least to some extent.

The medicine given to patients with Parkinson’s disease contains a precursor of dopamine, which the body can convert into dopamine. This gives patients with Parkinson’s disease
short-term reduction of their symptoms. The medicine can be given to constantly replenish the dopamine in the synapses.

Many illegal drugs affect the brain by altering the transmission of neurotransmitters at the synapse. Serotonin, for example, is used in the circuits that regulate mood. The drug ecstasy (MDMA) affects people’s moods by affecting the release of serotonin at the synapse. Cocaine blocks the flow of dopamine. LSD (lysergic acid diethylamide) is a powerful hallucinogenic drug. Not used much by the youth of today, it was popular in the United States during the 1960s. LSD affects both dopamine and serotonin channels, increasing the release of neurotransmitters by axons in sensory areas of the brain. This increase of activity in sensory areas is responsible for the strong visual illusions, auditory illusions, and even illusions of balance that occur when a person is under the influence of LSD.

Legal drugs can also affect neurotransmitters. Caffeine—common in coffee and tea—affects neurotransmitters in neurons, which innervate our muscles. Caffeine also causes the release of the neurotransmitter dopamine in our prefrontal cortex. Nicotine increases the activation of neurons that innervate our muscles. This is why some baseball players used to chew tobacco. The influx of nicotine into the nervous system allowed them to react just a tad faster to an incoming fastball. Chocolate induces additional release of serotonin.

Sensory systems have specialized neurons called receptor cells. These neurons have essentially modified their dendrites. Instead of receiving information from other neurons, these cells transform physical energy, such as light, into an electrochemical neural signal. For example, the rods and cones on the retina of the eye respond to light by converting the light (electromagnetic energy) into a neural signal, which travels up the optic nerve and synapses in the brain (go to www.sagepub.com/schwartz for more information).

Learning at the Cellular Level

Few scientific facts are more certain than the fact that the brain uses its neurons to transmit information. The neuroanatomy described above has resulted from the painstaking research of many neuroscientists, some of whom have received Nobel Prizes for their effort. However, understanding how these neurons encode and represent information—that is, memory—is still just being unraveled. We know much about processes that are involved in learning but little about how these relate to higher order organization of memory. Thus, what happens in the brain and to the brain when we learn something new is still an area of great mystery and dispute. Some researchers have examined what might be happening at a neural level when learning takes place. One possible cellular mechanism of learning is called long-term potentiation.

Long-term potentiation occurs when there has been a consistent pattern of activation between two connected neurons. What does this mean? Think of two neurons connected to each other. The axon of one transmits a message to the synapse between them. Neurotransmitters forge the gap between the two. The dendrite of the second neuron picks
up the signal and fires along to its next cell. Now, the rate of action potentials along the axon determines how much neurotransmitters will be released. The amount of neurotransmitters crossing the synapse determines how much of a signal will be initiated in the dendrite of the second cell. Usually, more neurotransmitter release means more of a signal in the dendrite. However, long-term potentiation means that constant signaling between these two cells will lower the amount of neurotransmitters needed to elicit a signal in the dendrite of the second cell. Thus, a change in the rate of firing in the second cell is caused by the experience of that cell. Thus, long-term potentiation is one possible model for learning at the level of the neuron. With this possible cellular basis for learning, we will leave our discussion of the microscopic and consider the gross anatomy of the human brain (go to www.sagepub.com/schwartz for more information).

STRUCTURES OF THE HUMAN BRAIN

The human brain is an incredibly complex biological organ containing more than 100 billion neurons (Murre & Sturdy, 1995). In addition to the neurons themselves are many other cells that support the functioning of the neurons. The brain weighs about 1,300 to 1,400 grams (3 pounds), larger than all other primate brains but smaller than those of dolphins, whales, and elephants. Even though the brain represents only about 2% of the average human’s body weight, it is an energy-intensive organ, using about 25% of the oxygen used by the body at any given moment. For this reason, the brain is heavily profused by a large blood supply, necessary to provide all that oxygen for the brain.

In earlier times, the brain was thought of as a single organ, in which areas within the brain were equally involved in all of its functions. We now know that the brain is composed of many separate anatomical and functional areas. In this section, we will review some of the main anatomical regions of the brain, explore what their function is, and describe how this relates to learning and memory (see Figure 2.4). This is not a textbook in neuroanatomy; thus, our tour of the brain’s anatomy will be merely an incomplete sketch of the incredible complexity of the brain’s organization.

First, the brain is divisible into two symmetrical halves, oriented in the left-right direction. These are the **right hemisphere** and the **left hemisphere**. With respect to human cognition, the left and right hemispheres do tend to have specific specializations, with the left hemisphere in particular being devoted to language and, with respect to memory, the interaction of language and memory. The right hemisphere is heavily involved in spatial cognition—that is, our understanding of space around us. The right hemisphere also allows us to process music. Although hemispheric specialization is the rule in human brains, there is also great overlap in function...
and considerable cross-talk between the two hemispheres. Therefore, the popular distinction between “left-brained people” (logical, verbal, and cold) and “right-brained people” (emotional, musical, and warm) has no reality in the brain. Indeed, modern neuroimaging is showing that although the left and right hemispheres are anatomically separate, functionally, with respect to higher cognition, there is less hemispheric specialization than previously thought.

In the top-to-bottom direction, the brain is divided into cortical (the surface of the brain) and subcortical (below the surface) structures. Subcortical structures are the many areas of the brain that rest below the brain’s surface. These are “evolutionarily old” areas of the brain that we, by and large, share with nonhuman animals. Subcortical structures are critical in maintaining basic life functions. They control the regulation of heartbeat, breathing, hunger, thirst, sleep, and many aspects of movement. Some subcortical areas
are also involved in memory and emotion. We will focus on those areas in this chapter. The thin top layer of the brain (see Figure 2.4) is the cerebral cortex, which is most closely associated with the processes that we study in psychology. Language, memory, complex emotion, creativity, problem solving, and music (to name a few) are all largely a function of this thin crust of the brain. It is our large cerebral cortex that also distinguishes our brains from those of other species. In this chapter, we will consider only those areas of the brain that are involved in memory function. Suffice it to say that the brain regulates everything we do externally, internally, consciously, and unconsciously. But our focus is memory. At the level of large-scale anatomy of the brain, memory functions appear to be most critical in the subcortical structures, the hippocampus, and the amygdala and in the frontal and temporal lobes of the cortex. We will review these areas next.

Subcortical Structures

**Hippocampus.** The hippocampus (see Figure 2.5) is in a network of the brain called the limbic system, located in and below the medial temporal lobe (a part of the temporal lobe just behind your ear). The hippocampus is considered a subcortical structure. Like most brain structures, it is bilateral—that is, there is one hippocampus on each side of the brain. To some (although not your author), its physical shape is reminiscent of a seahorse, hence the name “hippocampus,” which means “seahorse” in Greek. The main function of the limbic system seems to revolve around both memory and emotion, but the hippocampus is a structure very much associated with memory. In particular, the hippocampus appears to be an important part of the circuit, which encodes new memories, both conscious and unconscious. It does not, however, appear to be involved in the storage or representation of information in memory. However, when we retrieve information, the hippocampus does become activated. Interestingly, the hippocampus is involved in memory across a wide range of species. Rats, monkeys, and songbirds all have hippocampuses that are involved in memory. Thus, it is likely that the hippocampus has served a memory function for an extremely long time during the evolution of animal life on earth.

In humans, damage to the hippocampus can cause amnesia (that is, acquired memory loss). In particular, damage to the hippocampus causes difficulties in acquiring new information. Some research suggests that, in humans, the left hippocampus takes on more responsibility for verbal memory, whereas the right hemisphere is more involved in the memory for the spatial world around us and directions within the world (Amaral &
There is a parallel in bird memory. Research with a variety of bird species shows that the left hemisphere is responsible for the memory of song, whereas the right hemisphere is responsible for the memory of migratory routes (Colombo & Broadbent, 2000). Birds with either the left or right hippocampus damaged become amnesic as well. If the left hemisphere is damaged, they can no longer sing, but if the right hippocampus is damaged, they do not fly south properly in the winter (or whatever their migratory pattern is). Similarly, data show that in humans, damage to the left hippocampus is more likely to affect memory for stories and words, but damage to the right hemisphere will affect memory for directions and pictures.

**Amnesia**: memory deficits acquired through brain damage.

**Amygdala**: a part of the brain critical in emotional learning, fear, and memory.

**Hypothalamus**: an area of the brain associated with basic emotions.

The **amygdala** is also in the limbic system (amygdala means “almond” in Greek). The amygdala appears to have an important role in connecting features of memory with aspects of emotion. It is highly connected to the
hippocampus, consistent with its role in memory and also with the hypothalamus, an area of the brain associated with basic emotions. Because of these connections, the amygdala is associated with both fear conditioning and emotional learning.

Diencephalon. This part of the brain includes the structure known as the thalamus and the hypothalamus. The thalamus, in particular, is an area of the brain heavily connected to other areas of the brain. It appears to serve as a routing center, connecting disparate parts of the brain. Parts of the thalamus are crucial in the transmission of information from our sensory organs (eyes and ears, for example) to the cortical areas responsible for sensation. With respect to memory, the diencephalon includes massive connections between the medial temporal lobes and the hippocampus with the prefrontal lobes, which are involved in memory as well. Damage to the diencephalon can incur tremendous costs in terms of memory deficits. The amnesic syndrome associated with Korsakoff’s disease is associated with damage to the diencephalon. We will discuss Korsakoff’s disease in Chapter 10. Korsakoff’s disease involves deficits in new learning, deficits in retrieving well-stored information, and an impairment in the ability to distinguish between true and false memories.

Cortical Areas of the Brain Associated With Memory

The cerebral cortex consists of four main anatomical areas: the frontal lobe, the temporal lobe, the parietal lobe, and the occipital lobe (see Figure 2.6). Each area is named to agree with the name of the skull bone under which it lies. Each of these lobes is bilateral—that is, there is one on the left side and one on the right side of the brain.

The cerebral cortex, or simply cortex, is the evolutionarily most recent area of the brain and the area of the brain most different in humans than in other animals. In particular, the frontal lobe marks humans apart from other primates, especially the areas most anterior (i.e., toward the front) in the brain, usually referred to as the prefrontal areas. The surface of the human cortex is very wrinkly. These wrinkles allow the brain to pack more surface area of cortex inside the skull.

The function of the occipital lobe is visual processing. With respect to memory, this area of the brain is important in providing visual imagery when people remember events from their lives or what people or visual scenes look like. Therefore, when you recall what Brad Pitt looked like in Benjamin Button, your visual cortex will become activated. Similarly, when you think about the time you saw the
The two main functions of the parietal lobe are somatosensory perception and attention. Somatosensory perception refers to our various senses of touch (fine touch, pain, heat, cold, and pressure). This area of the parietal lobe is located toward the front of the parietal lobe, adjacent to the frontal lobe. Toward the back of the parietal lobe, near the occipital lobe, are networks engaged in spatial attention in the right hemisphere and attention to verbal material in the left hemisphere.
Occipital—vision
Parietal—somatosensory; attention
Temporal—audition, language, memory
Frontal—higher emotion, decision making, metacognition, memory

The two lobes most directly involved in memory processing are the temporal lobes and the frontal lobes.

**Temporal Lobes.** The areas of the temporal lobe most involved in memory processing are the areas directly adjacent to the hippocampus. These areas are called the medial temporal cortex. Like the hippocampus, the medial temporal lobe appears to be involved in the encoding of information into memory but not in the actual storage or representation of that information. In humans, there is some evidence that the left temporal lobe is more involved in the processing of verbal information and the right temporal lobe is more involved in the processing of spatial information. Damage to the medial temporal lobe produces amnesia similar to that seen with hippocampus damage. Other areas of the temporal lobe are involved in language, auditory processing, and interpreting and labeling visual images.

**Frontal Lobes.** The areas of the frontal lobe most involved in memory are the areas most anterior (i.e., to the front) of the brain called the prefrontal areas. The function of these prefrontal areas includes initiating memory (starting the conscious process of remembering). It also includes source monitoring, that is, determining from what source a memory came. Source monitoring means being able to distinguish if a memory is a personally experienced event or something someone told you. Source monitoring includes reality monitoring, which means distinguishing between fact and imagination. For example, one might have a vivid memory of surfing big waves in Hawaii but then realize this is a memory of dreaming that one participated in such an activity, rather than a memory of actually surfing. Patients with damage to the prefrontal lobes are known to confabulate (telling untruths but not knowing they are untrue). The confabulation occurs because they cannot distinguish real memories from fantasies, as in the example above. The prefrontal lobes are also associated with metamemory and self-regulation. Metamemory involves our awareness and knowledge of our own memory, and self-regulation involves our control of our memory system. The prefrontal lobes have other functions aside from the regulation of memory. They are also involved in higher emotion (i.e., jealousy, respect) and various aspects of problem solving and creativity.

That concludes our brief sketch of neuroanatomy. As we delve in greater detail into the cognitive psychology of memory, we will touch on the underlying neuroanatomy when the
relation between memory function and brain anatomy is known. In these sections, greater
detail on the anatomy-functional relations will be provided. Next we turn to the great tools
for learning about memory and the brain—namely, neuroimaging and neuropsychology.

INTERIM SUMMARY

The brain is a remarkably complex organ, composed of many intersecting parts and layers.
Fundamental to the study of memory and the brain is its division into left and right hemi-
spheres and its division into cortical and subcortical areas. The left and right hemispheres
of the cortex have slightly different functions. The right hemisphere is more likely to take
on roles related to spatial memory, to imagery, and to music, whereas the left hemisphere
focuses on language and verbal learning. The cortical areas of the brain tend to be involved
in higher levels of memory processing, whereas the subcortical areas, such as the hip-
pocampus, are more involved directly in encoding or, as in the case of the amygdala, emo-
tion and emotional learning.

NEUROIMAGING

The first decade of the 21st century has truly been the “decade of the brain.” Improvements
in technology and lowering of costs have allowed memory researchers to employ modern
neuroimaging techniques to explore the relation between memory processes and the
physical brain in ways in which researchers even in the 1990s would not have thought pos-
sible. We are beginning to get good snapshots of not only where in the brain various processes
occur but how these processes unfold over time (Conway et al., 2003). Neuroimaging is the
technology that allows us to create images that demonstrate which regions of the brain are
working during a particular memory or cognitive task. In this section, we will give a rudimentary
description of how the technology works and then focus on what the technique can tell us
about human memory. It is always worth keeping in mind, though, that neuroimaging is a
correlational technique. It shows correlations between cognitive performance and areas of
the brain that are active. This does not necessarily mean that these areas cause the activ-
ity. Three main neuroimaging techniques are outlined here: EEG, PET, and fMRI.

EEG (Electroencephalography)

EEG (electroencephalography) is the oldest of the neuroimaging techniques, dating back into
the 1940s. EEG technology is based on the fact that neurons conduct electricity. This electri-
cal conduction can be measured by sensitive electrodes, which are placed on the skull of a
person. As electrical activity moves from one area of the brain to another, it can be measured
as distinct “waves” of electrical activity (see Figure 2.7). In particular tasks, some areas of the brain will be more active. This activity will produce a larger wave of electricity, which EEG can detect. More important today is that the electrical activity of the brain can be measured every millisecond (1/1,000th of a second). Therefore, EEG is very sensitive to changes in time in the brain. However, even when 64 electrodes are placed on the skull, EEG is not as good as the other techniques at developing maps as to where processes occur in the brain.

Figure 2.7 EEG patterns. When an EEG is recorded on paper, it produces a pattern that looks like this. Although the EEG measures the electrical activity of millions of neurons, it can be used to make reliable inferences about brain function.
During sleep, our brains produce characteristic electric waves, whose form can be captured by the EEG. These waves are associated with the various stages of sleep (Massimini et al., 2005). EEG is also important in the diagnosis of epilepsy.

To study memory, researchers use a particular method called the event-related potential (ERP). In the ERP technique, EEGs are measured in response to particular stimuli (or events). The EEG starts recording when the stimulus is presented to a participant. It continues for the duration of the trial. The stimulus is then presented in many trials, and the EEGs are then averaged across the trials to eliminate the random activity that may be present during any given trial. What remains is a very clear wave. Once the trials have been averaged together, the resulting data can present a picture as to how electrical activity changes over time in response to the stimulus. Event-related potential can be used to probe the time course of cognitive processes in the brain. One such example involves a brainwave known as the p300. We discuss this here as an example of the usefulness of the technique. For example, when presenting words during a memory experiment, a particular wave occurs about 300 milliseconds after the stimulus is presented. It is called the p300 because it is a positive change in voltage. In a famous paradigm (known as the von Restorff effect), a list of words is presented to a participant. All but one of the words are from the same category. The out-of-category word is called the von Restorff item or the oddball. For example, the oddball item might be the name of a city in California among a list of names of kinds of fish. The p300 part of the event-related potential is distinctly higher for the oddball item than it is for in-category items (Metcalf, 1993). Being able to see in the ERP exactly where the p300 is and how it correlates to the person’s memory allows researchers to make a hypothesis about how memory is processed in the brain.

**Positron Emission Tomography (PET)**

Positron emission tomography (PET) technology allows scientists to get a detailed image of a living human brain without having to damage any living tissue. It does involve, however, injecting a small amount of a radioactive substance into a person’s blood, which does have potentially negative effects. Therefore, it is not a procedure that should be done repeatedly. PET is useful for both medical purposes (it can pinpoint a tumor) and research because it can isolate functional areas of the brain. PET offers, relative to earlier techniques, a superior ability to determine where in the brain a particular function is occurring. However, it does not allow for the detailed description of how in time information is changing in the brain. This is because it requires about 30 seconds of exposure to get a good image of the brain. Thus, activity in the brain is blurred over a 30-second window.

PET is based on a simple assumption: that areas of the brain that are being used will require more blood. Your brain is a biological organ, which is powered by the oxygen and sugars supplied by the blood. Because neurons that are active will require more oxygen, the body should send more blood to those neurons that are engaged in any particular cognitive, emotional, or behavioral task. Therefore, if you can trace where the blood is going to during a particular memory or cognitive task, then you can correlate that area of the brain with that particular cognitive function. Thus, if you can measure to what parts of the brain
the blood is flowing during a particular memory process, you know that the area of the brain is critical for that process.

In PET, a small amount of radioactive tracer is injected into the blood of a willing volunteer. The tracer travels through the bloodstream to all parts of the body and brain. However, the areas of the brain that are active will draw more blood from the circulatory system. Thus, greater amounts of the radioactive tracer will go to areas of the brain that are more active than to those that are less active. PET scans use complex measurements to determine which areas of the brain are emitting more radioactivity. Those areas that are more “radioactive” are associated with whatever cognitive task the volunteer is engaging in.

PET allows for very precise maps of the brain to be drawn. Increased activity is often restricted to very small regions of the brain, which can be determined via the PET. Cabeza and Nyberg (1997) used PET technology to isolate hemispheric differences in memory processing. They showed that when people were actively trying to learn new information—as opposed to passively registering information—there was increased activity in both the hippocampus and areas of the left frontal lobe. During retrieval, however, right frontal regions were more active. Other studies show that the right prefrontal lobe is more involved in retrieving events from your personal past, whereas the left prefrontal lobe is more involved in encoding new verbal information (go to www.sagepub.com/schwartz for more information on PET technology).

**Functional Magnetic Resonance Imaging (fMRI)**

MRIs are now the medical and research standard. Functional magnetic resonance imaging (fMRI) is the state of the art for cognitive neuroscience research. MRIs, like PET, allow for complex imaging of the brain without any invasive procedures. And today, MRIs are both safer and better at imaging than PET, as they involve no radiation. Magnetic resonance imaging is a common medical tool to examine structural damage in internal organs. It is routinely used to detect tumors, growths, and other damage in the brain. The term MRI means a structural MRI—these images are used to produce a detailed picture of the intact human brain. MRIs are of use medically, if you want to known where tumors or brain damage occur in the brain. They can also specify individual differences in the brain. As such, structural MRIs are useful for medical diagnosis and procedure. fMRI refers to a variant that tracks where in the brain particular functional components occur. That is, fMRIs track blood flow and thus can determine where in the brain certain processes are. The blood flow scan can be superimposed on MRI to reveal the structure responsible. Thus, in addition to acquiring a structural map, the fMRI can show dynamic changes in the brain (see Figure 2.8).

MRI works because different molecules in the brain react differently when placed in an extremely strong magnetic field. For structural images of the brain, typical of an MRI, the detector looks for changes in structures in water molecules in the brain. For fMRI, which has been developed to specify cognition-brain region correlations, the detector looks for changes in blood flow, much as PET does. Neither MRI nor fMRI require the introduction of harmful radioactive chemicals, and at present, there are no known adverse effects of the magnet itself. MRIs and fMRIs offer also much greater spatial resolution of where events take place in the brain than any other neuroimaging technique. fMRI can rescans the brain every .5 seconds, thus offering a much better time window than does PET, although still not as good as the EEG technology.
Research using fMRI technology has far-reaching consequences. As an example of the power of fMRI to answer previously unanswered question, Koshino et al. (2008) were interested in the differences in working or short-term memory for faces in autistic individuals. Autism is a disorder in which people may have linguistic, social, and emotional problems. Working memory is the memory system that handles information over short periods of time and that we currently have accessible in consciousness. It turns out that autistic individuals have a deficit in remembering faces, and Koshino and colleagues wanted to determine if it was a perceptual phenomenon or a memory one. If it is a perceptual phenomenon, the autistic individuals would have difficulties seeing the faces, which would show up in the fMRI as decreased activity in the areas associated with vision. If it is a memory phenomenon, the autistic individuals would see the face but then have difficulties matching it later. This would show up in the fMRI as a decrease in activation in memory areas of the brain. Koshino et al. asked people with and without autism to match faces while being monitoring by an fMRI. They found that, relative to the normal controls, the autistic individuals showed lower levels of activation in areas of the left prefrontal lobe, known to be involved in working memory. Thus, the neuroimaging data support the memory interpretation of this deficit in autism. For more on this study, go to www.sagepub.com/schwartz.

Figure 2.8 The brain as seen through an MRI. This image shows the brain from the side. Can you identify any areas of the brain associated with memory?
NEUROPSYCHOLOGY: MEMORY DEFICITS AND AMNESIA

The oldest methodology for examining the relation between memory and the brain is examining patients with brain damage. This is because examining patients with neuropsychological deficits does not require technology. Researchers must locate patients who have suffered brain damage, which is not a difficult task, and then observe the cognitive and behavioral deficits in the patients. Going back to the famous case of Phineas Gage in 1848, research has been directed at how brain damage affects cognition and behavior (Fleischman, 2002). Gage was a foreman on a railroad crew who was severely injured in a railroad construction accident. A poorly timed dynamite blast shot a medal rod through his frontal lobe. Although he survived the accident and lived many more years, the resulting brain injury changed his cognitive and emotional abilities, as well as drastically altered his personality. The study of the change in his behavior as a result of this accident set the stage for the development of neuropsychology. The research goal of neuropsychology is to correlate behavioral deficits or cognitive changes with the area of the brain that is damaged. The assumption, then, is that the damaged area of the brain is normally involved in the function of the affected behavior or cognitive ability.

Just over 100 years after Gage, in September 1953, a 27-year-old man known as HM underwent a risky and experimental surgery to alleviate symptoms of debilitating and extreme epilepsy. During the surgery, parts of his medial temporal lobe were removed on both sides, including most of both of his hippocampuses. As a direct result of the surgical procedure, HM suffered from strong anterograde amnesia, that is, a deficit in learning and retaining new information. This means he could not learn new facts, such as memorizing a phone number. He also suffered some but relatively mild retrograde amnesia, that is, the loss of memory of events before the injury. That is, he could remember events and facts from his life prior to the surgery no worse than a normal adult. While this surgery has never been repeated on any other human being, HM’s memory was studied extensively for the next 50 years (Corkin, 2002). HM passed away in 2008 at the age of 82. Although his ability to encode new events into episodic memory was strongly affected, research showed that his working memory (short-term memory) and procedural learning (skills) were largely intact.

Many other patients have been studied since then. These patients have varied from having very mild amnesia, just barely different from the memory of normal people without brain damage, to very severe. Moreover, the particular pattern of deficits is different in each patient, and the pattern of these deficits can be linked to where in the brain the damage occurs in that patient.

Neuropsychological studies allow researchers to examine the relation of deficits in cognition and behavior with the locus of damage within the person’s brain. In fact,
most brain damage is fairly diffuse, spread around large areas of the brain. However, in some cases, often the result of bullet wounds, strokes, or indeed surgery, as seen in the case of HM, the damage can be quite localized, allowing clear correlations to be drawn between the memory deficits and the brain damage. We will examine amnesia and other effects of brain damage on memory in detail in Chapter 10. The website sagepub.com/Schwartz has links to neuropsychological research (go to www.sagepub.com/schwartz). 7

CHEMICAL ENHANCEMENT OF MEMORY

From an early age, children in our society are warned of the dangers of illegal drug use. Paradoxically, over-the-counter drugs, prescription drugs, and legally available brain-altering drugs are ever present in our society. Indeed, there are few illegal drugs that have such a profound effect on our nervous system as these three legal drugs—caffeine, alcohol, and nicotine.

We take drugs when we have a cold, drugs to keep us happy, drugs to wake us up, and drugs to help us sleep. So it is not surprising that many people wonder if they can take drugs—legal or otherwise—that will help them remember new information. Unfortunately, the empirical data are mixed here. There are drugs that we can take that improve our memory, but most of them work by improving our alertness, influencing how long we can stay awake and focused, rather than memory per se. On the other hand, there is no doubt that there are drugs that prevent the formation of new memories. Indeed, these drugs may be considered to induce temporary amnesic symptoms. Some of these drugs—the antianxiety benzodiazepines—are widely prescribed and available.

The only prescription drugs available to improve memory are cholinergics (McDaniel, Maier, & Einstein, 2002). Although there is no evidence that these drugs improve memory in healthy individuals, they have been shown to boost memory performance in those who suffer from memory disorders such as Alzheimer’s. They do so by providing chemicals that serve as precursors to vital neurotransmitters in the human brain. Because many memory circuits use the neurotransmitter acetylcholine, the cholinergics provide acetylcholine precursors. The first available drug in this category was piracetam; it is now not regulated in the United States but is available with a prescription in most of Europe.

The data on caffeine, the active drug in common products such as coffee and colas, are mixed. Some data show that caffeine improves memory, whereas others point to decrements (Lesk & Womble, 2004). In any case, the advantage that caffeine may offer to memory is allowing an individual to study longer before falling asleep, rather than making the actual learning process more efficient. Indeed, new research suggests that caffeine, although it may help people study by allowing them to remain awake longer, reduces the efficiency

Cholinergics: drugs prescribed to patients with Alzheimer’s disease that alleviate memory loss in early phases of the disease.
of learning (Mednick, Cai, Kanady, & Drummond, 2008). That is, caffeine may hurt learning by making the number of items learned per unit of time actually less. However, caffeine may benefit memory by giving us more awake time to study.

On the herbal side, the leaves of the ginkgo tree have been used for generations and generations as a memory enhancer. It is marketed as such in health food stores, herbal stores, and even supermarkets. Marketers are allowed to do this because the extract from ginkgo is not considered a medicine. However, virtually no data demonstrate any positive effects that this herb has on memory (Elsabagh, Hartley, Ali, Williamson, & File, 2005). Thus, it is likely that, like many “folk” remedies, ginkgo only works via the placebo effect.

In short, there really does not yet exist a “memory drug,” that is, a simple pill that can increase your memory skills without affecting other aspects of your cognition or emotion. There are drugs that clearly interfere with memory, causing temporary amnesia.

**Benzodiazepines**, such as diazepam (i.e., Valium), lorazepam, triazolam, and midazolam, are the most commonly consumed drugs in the world because of their effects on anxiety, insomnia, and muscle relaxation (Kaplan, 2005). However, they are also strong amnesia-inducing drugs, especially within the episodic memory domain. Episodic memory refers to the memory for individual events from a person’s life. Many benzodiazepines also affect semantic memory, or our knowledge of the world. The benzodiazepines that are the most commonly studied in cognitive research are diazepam, lorazepam, and midazolam. The pattern of memory impairment differs slightly from one benzodiazepine to another, but all of the benzodiazepines impair the learning of new information, creating temporary anterograde amnesia (Danion, 1994).

**OLFACTION, MEMORY, AND THE BRAIN**

Olfaction refers to our sense of smell. Human beings have long been aware of the intimate relation between the sense of smell and memory, particularly the retrieval of highly personal autobiographical memory. Most people can describe the relation of a particular smell to some salient event from their life (Herz, 2007). For example, the smell of naphthalene (mothballs) always reminds your author of visits to his grandmother’s apartment as a young child. The famous writer Proust describes how the scent of a French pastry called a madeleine transported him back to his childhood in the south of France (Proust, 1928). Many people report associations between a particular perfume or cologne with a girlfriend or boyfriend, even if the relationship ended years ago. As is clear from the examples, the connection between memory and smell is also connected to emotion. The memories elicited by odor are usually highly emotional memories.
The neural reason for this strong connection between our senses of smell, emotion, and our memories rests in the limbic system. The limbic system is involved in both memory and emotion but is also the primary area for processing odors. Located within the limbic system is the **olfactory bulb**, the primary organ in the brain for processing odors. It receives input directly from the olfactory nerves coming from the hair cells in the nose. Only after information passes through the olfactory bulb does it go to higher areas of the brain in the cortex. But the olfactory bulb is heavily connected neurally to two important memory centers in the limbic system, the hippocampus and the amygdala. These strong connections provide the neural basis for the strong association between odors and both memory and emotion. Interestingly, it is only after these connections between the olfactory bulb and the limbic system occur that information is processed by the olfactory cortex and other areas in the prefrontal lobe. This may account for the “gut” feeling that is characteristic of these strong odor-memory-emotion associations (Herz, 2005). For more on research on memory and olfaction, go to www.sagepub.com/schwartz.

**MEMORY, MUSIC, AND THE BRAIN**

In many ways, music has similar effects on memory. A particular song may remind you of a long-ago dance with your high school sweetheart. Another song will rouse memories of the good old days in college. Yet another song may bring back pleasant childhood memories. In addition, many performers develop powerful abilities to learn and remember music. I have seen professional pianists play for hours straight without consulting sheet music. The sheer number of finger movements that must be memorized to accomplish this task is enormous. How is it that musicians are able to remember so much and retrieve it while playing?

These intuitions have been documented in the psychological laboratory. Janata, Tomic, and Rakowski (2007) played segments from a large set of popular songs to participants in their experiment. The participants were asked to describe any autobiographical memories or any emotions that were elicited by the songs. More than 30% of songs, on average, elicited memories or feeling of nostalgia in each participant. In some cases, the participants reported vivid memories or strong emotions.

We also know that, unlike language, musical perception is mainly processed in the right hemisphere of the brain. In many professional musicians, however, hearing or playing music activates both the left and the right hemispheres equally. Many cortical areas and nearly every cortical lobe are involved in some aspect of music. Whereas the occipital lobe (vision) is mainly sidelined (except for reading music), the other three main cortical lobes all have important roles in the processing of music. The temporal lobes house the auditory cortex, the first area of the brain that processes sounds, including musical sounds. The sensory
cortex in the parietal lobe is essential in providing feedback in playing an instrument or in dancing. And the prefrontal cortex is necessary in interpreting and appreciating music (see Levitin, 2006).

The brain is able to create powerful memories of music. This can be seen in our ability to make auditory images of music. Most of us can mentally “play” a song without any actual music or voices occurring. Imagine the opening strains of Beethoven’s Fifth Symphony, and you probably hear an orchestra. Imagine Billy Joel singing “Piano Man” and you probably “hear” his raspy voice. Indeed, research shows that auditory centers in the brain are just as active when you are imagining music as when you actually hear music (Janata, 2001). To have such strong imagery, we must have an accurate memory of the song, what it sounds like, and how one singer’s voice differs from another. For most current students, of course, the musical selections in the example will not trigger strong autobiographical memories, but they might for older adults. Thus, the connection between music and autobiographical memory is also important. Thus, like with olfaction, there are strong connections between memory and music, which we are beginning to understand are rooted in connections in the brain. For more on music and the brain, go to www.sagepub.com/schwartz.  

SUMMARY

The cognitive psychology of memory is increasingly becoming influenced by the neuroscience of memory, forming the hybrid field known as cognitive neuroscience. Cognitive neuroscience is the science that examines the relation between brain anatomy and cognitive function. Foremost in this field are the successes of neuroimaging, which have greatly contributed to our understanding of how the brain creates, represents, interprets, and retrieves memories. At the level of cells, the brain is composed of billions of neurons, which talk to each other electrically. At higher levels, there are several key components of the brain involved in memory, including the amygdala, the hippocampus, the diencephalon, the medial temporal lobes, and areas in the prefrontal lobes. Damage to these areas of the brain can cause various forms of amnesia, or disorders of memory. Neuroimaging studies reveal how these areas are active during memory processes. There are three main neuroimaging techniques: PET scans, MRI and fMRI, and EEG. Each technique has different advantages and disadvantages, although fMRI has become the state of the art in cognitive neuroscience.

The brain uses chemicals called neurotransmitters to bridge the gap in the synapse between cells. Neurotransmitter function can be influenced by drugs. Some drugs, such as benzodiazepines, interfere with memory processing, but the search continues for drugs that can improve memory performance directly. We also discussed the neural explanation for why such a strong connection exists between some odors and certain strong autobiographical memories. Finally, we concluded with a brief section on the intersection between music and memory and the how this relation occurs in the brain.
1. What is meant by the term *engram*? What did Lashley hope to achieve by identifying it? How does the Quiroga et al. (2005) experiment relate to the concept of the engram?

2. What is a traumatic brain injury?

3. Describe the flow of information through the neuron, including how information is transmitted through the axon, dendrite, and synapse. Include the purpose of neurotransmitters.

4. What is long-term potentiation?

5. Describe the functional significance of each of the following brain regions: (1) hippocampus, (2) amygdala, (3) diencephalon, (4) temporal lobe, and (5) frontal lobe.

6. How does the EEG measure activity in the brain? What is the EEG good for?

7. What advantages does fMRI have over EEG and PET technology?

8. What is amnesia? What is the difference between anterograde and retrograde amnesia?

9. How do benzodiazepines affect memory? How do cholinergics affect memory?

10. Why is the olfactory (sense of smell) system so tied to emotion and memory?
ONLINE RESOURCES

1. For more on Alzheimer’s disease, see http://www.alz.org/index.asp.
2. For more on traumatic brain injuries, see http://www.traumaticbraininjury.com.
3. For more on neurotransmitters, go to http://faculty.washington.edu/chudler/chnt1.html or http://www.neurotransmitter.net.
4. For more information on cellular neuroscience, go to http://www.estrellamountain.edu/faculty/farabee/biobk/BioBookNERV.html.
6. For the complete article on fMRI in autism, go to http://cercor.oxfordjournals.org/cgi/content/abstract/18/2/289.
7. For more information on neuropsychology, go to http://www.neuropsychologycentral.com.
8. For more on research on memory and olfaction, go to http://www.rachelherz.com.
9. For more on music and the brain, go to http://faculty.washington.edu/chudler/music.html.

Go to www.sagepub.com/schwartz for additional exercises and study resources. Select Chapter 2, Memory and the Brain for chapter-specific resources.