The word brain 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,” 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. 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. It is important, however, to note that our use of the word brain refers to the neural structures maintained inside the head and not to the various metaphorical roles that the word brain has acquired.

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 are other doctors. However, the brain is surrounded and infused with 400 miles of blood vessels, so surgeons must be very
Section 1  Memory and Processes of Memory

careful when probing 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 as a biological tissue that needs to be treated very carefully.

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 prefrontal regions are for planning, thinking, and monitoring, whereas the back of the brain processes vision. The temporal lobe is involved in memory and language, whereas the parietal lobe directs attention. Increasingly, cognitive neuroscience is also considering the role of the various white-matter tracts that connect different regions of the brain, and this is considerably enhancing our understanding of the brain (Dick, 2018). Viewed this way, the brain is not really one organ but many dozens of distinct regions, each with its own appearance, its own microanatomy, its own set of connections to other areas, and its own function.

Behind each way of looking at the brain, however, is the assumption that the biological organ located inside the skull is directly involved in memory, language, and thought. It was not always thus. Aristotle famously mistook the heart as the organ of thought and believed that the brain merely cooled 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. Moreover, it is important to understand what the brain is and how it physically achieves its function in order to understand the relation of brain and memory in human beings.

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 (Chatterjee, 2018; Slotnick, 2012; Weldon, 2015). Recent technological advances have provided unrivaled methods for examining how the brain works and how memories are physically 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, 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 25 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 oldest 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, knowing where memories are stored would allow neuropsychologists to predict particular forms of amnesia. Third, it is an important practical question. If certain areas of the brain store memories, then we need to respect these areas when probing the brain during neurosurgery. The question thus is whether it is possible to pinpoint an area in the brain responsible for a particular memory (Quiroga, 2013).
The consensual wisdom on this topic 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. However, this consensual wisdom has occasionally 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, 2001b). 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 that 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, and he wondered if one’s memory of the capital of Slovakia could be eliminated by excising a few cells. He spent his entire career looking for these memory-specific cells but never found any. His research focused on memory in rats, which probably did not know much about Slovakia but nonetheless can learn a great many things. Finally, at the end of his career, Lashley was forced into concluding that there are no engrams and that memory representation instead occurs because of a connection between disparate areas in the brain. Nowadays, there is good evidence to support this idea. For example, Addis, Knapp, Roberts, and Schacter (2012) showed that visual areas of the brain are activated during autobiographical recall as well as more standard memory areas, such as the hippocampus. Thus, the current view is that stored memories are distributed throughout the brain and that stored memories have more to do with connections across spatially separate areas of the brain than any specific area. Thus, the memory of your great-grandmother is the result of axonal connections among areas in the visual brain, auditory brain, emotion centers, and perhaps many others.

This is the conventional wisdom from Lashley’s time and to the present. However, Quiroga, Reddy, Kreiman, Koch, and Fried (2005) claimed to have found specific areas in the brain that seem to support specific knowledge structures. In Quiroga et al.’s (2005) studies, people saw photographs or printed names of various celebrities while the electrodes were recording directly from their brains. In general, the photographs elicited greater responses in the visual areas of the brain, whereas the printed names evoked responses in areas of the brain involved in reading. But embedded in the temporal lobe, Quiroga et al. found areas of the brain that responded specifically to information about particular people. These areas of the brain responded 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 responded to the actress’s name or her photograph, even across a range of characters from movies. Nearby the Halle Berry area 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 (see Bowers, 2009). The Quiroga study was done with single-cell recording of volunteers who were about to have brain surgery, and as a consequence, some have challenged the generalizability of
their findings. Moreover, there have also been failures to replicate this study. Thus, others 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 (Plaut & McClelland, 2010). Most researchers continue to believe that memory storage is widely distributed across the brain and that distributed models, such as that of Plaut and McClelland, offer better explanations than do engrams. However, the exact nature of memory representation in the brain is still a topic much in need of both research and theory.

**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 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. According to the Alzheimer’s Association, it is likely that there are 47 million people in the world currently suffering from Alzheimer’s (https://www.alz.org/global/overview.asp). As of 2019, there were 5.7 million people in the United States alone with Alzheimer’s disease (see https://www.alz.org/facts/). Alzheimer’s disease is a terminal illness; its initial signature is the development of amnesic (memory loss) symptoms. It affects the brain, clearly illustrating the brain–memory relation. In its early stage, Alzheimer’s patients have trouble learning new information and retrieving recent events. During later stages, 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 will also relieve fear among many who will 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, understanding brain–memory relationships could wind up benefiting normal older adults as well.

Memory deficits are also a common symptom of **traumatic brain injuries** (TBIs). TBIs occur when the brain violently and suddenly hits a hard object, such as an automobile windshield. TBIs are often 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 a bullet. Closed-head injuries often result in greater damage to the brain than open-head injuries. According to the Centers for Disease Control and Prevention (Taylor, Bell, Breiding, & Xu, 2017), 2.8 million people suffer TBIs every year. Most of these are minor, but 50,000 a year are fatal.

The largest source of TBIs is motor vehicle crashes. In fact, 17% of TBIs result from motor vehicle crashes (Corrigan, 2015). 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. Temporal lobe areas may also be damaged, causing further memory complications. The countercoup (that is, the blow to the back of the head) may damage 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. In the near term, however, buckle up, don’t text and drive, and don’t disconnect your airbag! The care and treatment of patients with brain damage falls in the domain of clinical neuropsychology, which focuses on rehabilitation and restoration of cognitive skills for auto accident victims—among their other tasks. 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 automobile-based culture. However, because of 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 is 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.

THE NEURON

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 blocks of both our brain and our entire nervous system. Neurons form huge communicating networks in the brain and connect to neurons in the muscles throughout 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.

Like all biological cells, the neuron contains a nucleus, which houses one set of the individual’s chromosomes. The chromosomes contain the genes, which hold the 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 makes neurons unique are the fibers that extend outward from the soma. These fibers allow neurons to transmit information from one part of the brain or nervous system to another part. 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 type of fiber does so in a different manner. Indeed, the transmission of information within the brain occurs through small electric currents racing through the neurons there.

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 any 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. The single axon may branch out and be connected to many hundreds of other neurons, transmitting the same electrical pulse to all of them. Transmission in an axon is an electrochemical process called an **action potential**. This is because transmission of electricity along the axon is not simply electrical, like electricity going through 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. An extremely small gap, called the **synapse**, exists between the two neurons. Electricity does not pass from the axon of one cell to the dendrite of the next. Instead, the transfer of information between neurons occurs chemically, rather than electrically. At the end of the axon are little nodules called **terminal buttons**. When the electrical signal reaches the terminal buttons, the signal triggers them 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 along the dendrites is electrical. Therefore, longer dendrites experience a greater loss of electrical power than do shorter dendrites. This is similar to the transmission of electricity through power lines, in which more energy is lost when the electricity is transported over long distances than when transported over short distances. As such, dendrites tend to be very short to minimize this loss of information. 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 practically instantaneous.

Transmission of information in the axons, in contrast, is electrochemical. It is electrical over very short segments but then gets a power boost (the action potential) via a chemical process as it moves down the axon. This allows axons to be quite long (indeed, you have one-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 flow in the axon is relatively slow (sometimes as slow as 10 meters per second—that is the speed of an Olympic sprinter). Incidentally, it is likely the slowness of...
FIGURE 2.2  Components of the neuron.
The illustration is of a multipolar motor neuron.


FIGURE 2.3  The synapse.
Notice the separation between the presynaptic axon terminal and the postsynaptic neuron.

axon transmission that led to the evolution of a large ganglion ("a second brain") in the tail of such animals as dinosaurs.

Finally, transmission of information is completely chemical at the synapse, where 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 that acts as an insulator does around 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 due to the slowdown of information flowing through the axons.

Sensory systems have specialized neurons called receptor cells. These neurons have essentially modified dendrites. Instead of receiving information from other neurons, receptor 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.

**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, the 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. These neurotransmitters elicit more action potentials per unit of time. In contrast, neurotransmitters that decrease activity in the neuron are said to be inhibitory. 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 found 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. For more information on the topic, see Sheng, Sabatini, and Südhof (2012).

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 difficulty 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, medicines are 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 and thereby replenish the dopamine in the synapses. This gives patients with Parkinson’s disease short-term reduction of their symptoms. Although Parkinson’s disease is mainly associated with physical disabilities, it may often affect areas of the frontal lobe, leading to decrements in certain aspects of memory performance, such as prospective memory (Costa et al., 2015).

Many illegal drugs affect the brain by altering the transmission of neurotransmitters at the synapse. The illegal drug ecstasy (MDMA) affects people’s moods by modifying the release of serotonin at the synapse. Cocaine blocks the flow of dopamine. Lysergic acid diethylamide (LSD) is a powerful hallucinogenic drug, popular during the 1960s. It 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.

Legal drugs also affect neurotransmitters. Caffeine—common in coffee and tea—affects neurotransmitters in the neurons that innervate our muscles. Caffeine also causes the release of the neurotransmitter dopamine in our prefrontal cortex. Nicotine, one of the main active drugs in tobacco products, increases the activation of the 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 the additional release of serotonin. Luckily, chocolate, unlike nicotine, is harmless.

STRUCTURES OF THE HUMAN BRAIN

The human brain is an incredibly complex biological organ containing more than 100 billion neurons (Herculano-Houzel, 2017). In addition to the neurons themselves, the brain consists of a greater number of other cells that support the functioning of neurons. In total, the human brain weighs about 1,300 to 1,400 grams (3 pounds), making it 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, accounting for about 25% of the body’s oxygen use at any given moment. For this reason, the brain has a very large blood supply.

In earlier times, the brain was thought of as a single organ whose entirety was equally involved in all of its functions. This is not entirely true. 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 functions are, and describe how they relate to learning and memory (see Figure 2.4). This is not a textbook in neuroanatomy; thus, our tour of the brain’s anatomy will provide an incomplete sketch of the incredible complexity of the brain’s organization.

The brain is divisible into two symmetrical halves, oriented in the left–right direction. These are the right hemisphere and the left hemisphere. The left and right hemispheres have some specific specializations, with the left hemisphere focused on 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 has a greater role in the processing of 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.

Because of this overlap, 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. There is no evidence that people who are linguistically talented, for example, have larger or more neuronal connections in their left hemisphere than they do in their right hemisphere. Indeed, evidence now suggests that the right hemisphere is indeed critical to many aspects of language (M. L. Blake, 2016). Modern neuroimaging is showing that, although the left and right hemispheres are anatomically separate, there is less hemispheric functional specialization with respect to higher cognition than previously thought (Dundas, Plaut, & Behrmann, 2013).

In the top-to-bottom direction, the brain is divided into cortical (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 in emotion.

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 distinguishes our brains from those of other species. Suffice it to say that the brain regulates everything.
we do externally, internally, consciously, and unconsciously, but we will consider only those areas of the brain that are involved in memory function. 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 part of a network in the brain called the limbic system, located in and below the medial temporal lobe (a part of the temporal lobe, which is 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, 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 involve both memory and emotion, but the hippocampus is a structure very much involved in memory function. Damage to the hippocampus can cause anterograde amnesia.

**Limbic system:** A set of brain structures located just beneath the cerebral cortex that includes the hypothalamus, the hippocampus, and the amygdala and functions as an important area for both memory and emotion.

**FIGURE 2.5** The location of the hippocampus.
associated with memory. In particular, the hippocampus is an important part of the circuit that encodes new memories, both conscious and unconscious. It does not 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 hippocampi that are involved in memory. That such diverse species use the hippocampus for memory suggests that this brain structure served the function of memory in the common ancestor of mammals and birds, many millions of years ago. Moreover, among songbirds, the right hippocampus is associated with spatial memory and migration, whereas the left hippocampus is associated with song memory, in a manner similar to our hemispheric lateralization (Bailey & Saldanha, 2015; Sherry & Hoschooley, 2009). Birds with damage to either the left or right hippocampus become “amnesic”; if the left hemisphere is damaged, they can no longer sing, and if the right hippocampus is damaged, they do not fly south properly in the winter (or whatever their migratory pattern is).

In humans, damage to the hippocampus can likewise cause amnesia (that is, memory deficits acquired through brain damage). In particular, damage to the hippocampus causes difficulties in acquiring new information. We will shortly discuss the patient H. M., who had damage to both his left and right hippocampi and had a severe form of amnesia that prevented almost all new learning. Some research suggests that in humans the left hippocampus takes on more responsibility for verbal memory, whereas the right hippocampus is more involved in the memory for the spatial world around us and directions within the world (Hartzell, Tobia, Davis, Cashdollar, & Hasson, 2015). 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.

**Amygdala**

The amygdala is also in the limbic system (amygdala means “almond” in Greek). The amygdala appears to play 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 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. In humans, the amygdala seems to have an important role in the symptoms of post-traumatic stress disorder. When people with post-traumatic stress disorder are asked to retrieve trauma-specific memories, significant activity can be seen in the amygdala (O’Doherty, Chitty, Saddiqui, Bennett, & Lagopoulos, 2015).

**Diencephalon**

This part of the brain includes the structures 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 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 known as Korsakoff's disease is associated with damage to the diencephalon. 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. We will discuss Korsakoff's disease at greater length in Chapter 10.

Cortical Areas of the Brain Associated With Memory

The cerebral cortex is the evolutionarily most recent area of the brain and the area of the brain most different in humans compared to other animals. The cerebral cortex (also known as the neocortex or simply the 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 in concordance 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 cognitive-functional specialties of each area can be summarized as follows:

- Parietal—somatosensory, attention
- Occipital—vision
- Frontal—higher emotion, decision-making, metacognition, memory
- Temporal—audition, language, memory

Let's begin our discussion of the role of the cerebral cortex in memory with the parietal and occipital lobes.

Parietal Lobe

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; Schwartz & Krantz, 2019). This perception 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). Though memory processes may be a secondary in the parietal lobe's set of tasks, increasing activity in the parietal lobe has been linked to memory. In particular, activity in the parietal lobe is critical to working memory tasks and prospective memory tasks—that is, memory of things to do in the future (Cona, Scarpazza, Sartori, Moscovitch, & Bisiacchi, 2015).

Occipital Lobe

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 Lupita Nyong'o looked like in Black Panther, your visual cortex will become activated. Similarly, when you think about the time you saw the Mona Lisa at the Louvre Museum
in Paris, your visual cortex will become activated. The occipital lobe is also involved in basic visual memory. V4 is an area of the brain involved in color processing. Patients with damage to this area forget the colors associated with objects. For example, a patient will forget that ripe bananas are yellow and that red lights mean for a driver to stop. This goes beyond color blindness—damage to V4 removes normal color perception but also interferes with the past memory of color. However, V4 is seldom damaged in isolation from the rest of the occipital lobe, but there have been patients with selective V4 damage who show impaired color memory without other memory deficits.

**Frontal Lobe**

The frontal lobe particularly distinguishes humans from other primates; especially different is the area most anterior (i.e., toward the front) in the brain, usually referred to as the prefrontal cortex (also called the prefrontal area). The prefrontal areas of the frontal lobe are those most involved in memory. Their functions include initiating memory (starting the conscious process of remembering). They are also involved with source monitoring. 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), because they cannot distinguish real memories from fantasies. 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 self-regulation of memory; they are also involved in higher emotion (e.g., jealousy, respect) and various aspects of problem-solving and creativity.

**Temporal Lobe**

The areas of the **temporal lobe** most involved in memory processing are those 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 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.

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 and provide greater detail on anatomy-functional relations. Next we turn to the tools for learning about memory and the brain—namely, neuroimaging and neuropsychology.

**INTERIM SUMMARY AND QUIZ**

The brain is a remarkably complex organ, composed of many intersecting parts and layers. Fundamental to the study of memory are the brain’s division into left and right hemispheres 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, imagery, and 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 hippocampus, are more directly involved in encoding or as in the case of the amygdala, emotion and emotional learning.
QUIZ

1. Quiroga et al. did an experiment on neural representation with patients about to have brain surgery. They were interested in
   a. If the surgery would affect their ability to recognize faces
   b. If there are specific neurons in the temporal lobe associated with specific knowledge, such as individual people
   c. If neural representation occurs in cortical areas or subcortical areas
   d. The exact location of the pineal gland

2. Research on memory in Parkinson’s patients shows that
   a. All Parkinson’s patients show deficits in recognition
   b. There is some evidence that, because of compromise to the occipital lobe, some Parkinson’s patients show global amnesia
   c. Because of damage to the prefrontal lobe, some Parkinson’s patients show deficits in prospective memory
   d. All of the above are true

3. The importance of the synapse is that
   a. In order for a neural signal to be sent, an electric charge must surge over the synapse
   b. In order for a neural signal to be sent, neurotransmitters must move from the axon of one neuron to the dendrite of the next
   c. The synapse generates the axon potentials that travel from one neuron to the next
   d. Electrochemical processes stop at the synapse—only terminal buttons can cross the synapse

4. Which of these limbic system structures is most associated with emotion and emotional learning?
   a. The amygdala
   b. The thalamus
   c. The hyperthalamus
   d. The hypothalamus

5. Self-regulation of memory is most associated with which lobe of the cerebral cortex?
   a. Parietal
   b. Occipital
   c. Frontal
   d. Temporal

   1. b
   2. c
   3. b
   4. a
   5. c

METHODS OF COGNITIVE NEUROSCIENCE

The second theme of this book is that learning and remembering are biological processes based in the brain. Neuroimaging studies over the last quarter century have continually supported and elaborated on that statement. Improvements in technology and reductions in 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 2000s, would not have thought possible. We are beginning to
get good snapshots of not only where various processes occur but how these areas are connected and how these processes unfold over time (Dick, 2018).

**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. Six main neuroimaging techniques are outlined here: EEG, PET, MEG, MRI, near-infrared spectroscopy (NIRS), and stimulation techniques.

**EEG (Electroencephalography)**

EEG (electroencephalography) is the oldest of the neuroimaging techniques, dating back to the 1940s. EEG technology is based on the fact that neurons conduct electricity. This electrical 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). During 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, as noted in Chapter 1, 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 the maximum of 128 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. 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.

There is also a form of EEG called **intracranial EEG** (also known as electrocorticography). Intracranial EEG means electrodes are placed directly on the surface of the brain. This form of EEG only occurs during surgery or post-surgery if the brain is still exposed. We have already seen data from this type of procedure when discussing the engram (Quiroga et al., 2005). Because the recording is now occurring directly on the surface of the brain rather than through the skull and intervening fluid, intracranial EEG can get much more precise readings of electrical activities in particular areas. For example, Perez et al. (2015) used intracranial EEG to follow patterns in the brain while patients were driving car simulators in the hospital. Perez found activity in the motor areas of the brain prior to drivers making decisions about routes, suggesting the importance of motor planning in such decision-making.

Returning to scalp EEGs, 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 averaged across the trials to eliminate random activity that may be present during any given trial. What remains is a clear wave. Once the trials have been averaged together, the resulting data present a picture of how electrical activity changes over time in response to the stimulus. ERP can be used to probe the time course of cognitive processes in the brain. One example involves a brainwave known as the p300. When words are presented during a memory experiment, a specific wave occurs about
FIGURE 2.7 | EEG patterns. When an EEG is recorded on paper, it produces a pattern that looks like this. The specific pattern of the EEG shown is not relevant here. Rather, the illustration demonstrates that reliable readings from multiple areas of the brain over time can be obtained. Although the EEG measures the electrical activity of millions of neurons, it can be used to make reliable inferences about brain function.

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, called the von Restorff item or the oddball, might be the name of a city in California among a list of names of kinds of fish. The $p300$ part of the ERP is
distinctly higher for the oddball item than it is for in-category items (Kiat, Long, & Belli, 2018; Shang, Huang, & Ma, 2015). 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.

**Magnetoencephalography (MEG)**

Magnetoencephalography (MEG) allows researchers to measure brain activity by detecting magnetic fields that the brain produces. As with scalp EEG, the measurements take place at the scalp and do not require any invasive procedures. Also like EEG, MEG can record highly accurate timing of when events occur in the brain, down to the millisecond. However, it can also produce more detailed spatial maps of the brain. In this way, MEG is useful in tracking the pathway of information, as particular processes work their way from one part of the brain to the next. MEG presents no risk to the person being studied; indeed, it has been used with infants (Sheridan, Matuz, Draganova, Esweran, & Preissl, 2010). In one study, Garrido, Barnes, Sahani, and Dolan (2012) showed that the amygdala is active when people are evaluating the emotional content of faces. In another study, Ueno, Masumoto, Sutani, and Iwaki (2015) showed that specific sensory areas of the brain were active when participants were recognizing words that had been presented to them either auditorily or visually.

**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 should not 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 an enhanced ability to determine where in the brain a particular function is occurring. However, it does not allow for the detailed description of how information is changing over time in the brain, because it requires about 30 seconds of exposure to capture a good image. Thus, activity in the brain is blurred over a 30-second window. Because of its dangers to patients and the fact that MRI is superior in spatial resolution, PET is seldom used for research anymore.

PET is based on an assumption that areas of the brain that are being used require more blood. The brain is a biological organ that is powered by the oxygen and sugars supplied by blood. Because neurons that are active 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 a researcher can trace where the blood is going during a particular memory or cognitive task, then he or she can correlate that area of the brain with that particular cognitive function.

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. Although all parts of the body receive the tracer, more active areas of the brain draw more blood from the circulatory system than do less active areas. 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.
Magnetic Resonance Imaging Technologies

As noted in Chapter 1, functional magnetic resonance imaging (fMRI) has advanced our understanding of the relation of brain and mind more than any other tool. MRI and fMRI, in particular, are safe and quick means of generating images of the structure and function of the brain (Oatley, 2018). Moreover, the latest technology involves combining MRI methods to allow detailed spatial mapping, excellent temporal resolution, as well as the ability to look at white matter (axon connections) and gray matter (neuron cell bodies). MRIs and fMRIs also offer much greater spatial resolution of where events take place in the brain than any other neuroimaging technique. fMRI can rescan the brain every 0.2 seconds, thus offering a better time window than does PET, although still not as good as EEG.

Standard magnetic resonance imaging (MRI) is a medical tool commonly used to examine structural damage in internal organs, and 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. MRI works because different molecules in the brain react differently when placed in an extremely strong magnetic field. To generate structural images of the brain, the detector looks for changes in the structures of water molecules in the brain. fMRI is a variant that shows where in the brain particular functional components occur by tracking blood flow. In addition, the blood flow scan can be superimposed on an MRI to reveal the structure responsible, thereby providing researchers with both a structural map and a display of dynamic changes in the brain (see Figure 2.8).

Diffusion MRI or diffusion tensor imaging (DTI) is another MRI technique. DTI compares the pattern of movement of molecules, particularly water, within tissue in order to derive structural images. DTI is particularly useful for examining white-matter connections in the brain. As such, it is useful in delineating pathways in the brain rather than neural centers. For example, Dick, Bernal, and Tremblay (2014) have looked at the white-matter pathways that connect areas of the brain associated with language. White-matter tracts, such as the arcuate fasciculus, are particularly involved in these processes. DTI also is very useful in medical diagnoses, such as distinguishing Alzheimer's disease from other forms of dementia (Parra et al., 2015).

Research using MRI techniques has far-reaching consequences. In an example of the power of fMRI...
to answer previously unanswered questions, Koshino et al. (2008) were interested in the differences in working or short-term memory for faces in individuals with autism. 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 individuals with autism have a deficit in remembering faces, and Koshino and colleagues wanted to determine whether it was a perceptual phenomenon or a memory phenomenon. If it is a perceptual phenomenon, the individuals with autism would have difficulty seeing the faces, and this difficulty would show up in the fMRI as decreased activity in the areas associated with vision. If it is a memory phenomenon, the individuals with autism would see the face but then have difficulty matching it later. This would show up in the fMRI as a decrease in activation in memory areas of the brain, such as the prefrontal lobe. Koshino et al. asked people with and without autism to match faces while being monitored by an fMRI. The researchers found that, relative to the normal controls, the individuals with autism 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.

Brain Stimulation Techniques

Another class of neuroscience techniques involves directly stimulating specific areas of the brain and observing the change in cognition that results from the stimulation. There are several techniques that stimulate the brain via different methods. The ones reviewed here are transcranial magnetic stimulation (TMS), transcranial direct current stimulation (tDCS), and deep brain stimulation (DBS).

In **transcranial magnetic stimulation (TMS)**, a magnetic charge is applied via the skull to a particular area of the brain. This is done by placing a magnetic coil near the surface of the scalp. This coil then produces electric currents, which surge into the adjacent areas of the skull and brain. TMS has a number of medical uses, including being an effective treatment for severe migraines (McWhirter, Carson, & Stone, 2015). In terms of research, TMS allows a small pulse of electricity to temporarily disrupt function in a small area of the brain. Researchers can then observe the changes in cognition and behavior that follow. In some cases, TMS will inhibit performance or decrease people’s ability to do a task, but in some cases, it can also enhance skills. In either case, the effects of the TMS wear off within a matter of minutes after the coil is removed from near the person’s scalp. Because TMS allows true experiments, one can infer causation from the disrupted area rather than simply correlation, as in neuroimaging work. Some examples of TMS research on memory include a study by Desmond, Chen, and Shieh (2005), who showed that verbal working memory (short-term memory for words) was impaired after a single TMS pulse to an area of the brain called the cerebellum. In addition, Bonni et al. (2015) applied a continuous TMS pulse to an area of the brain known as the precuneus. Stimulation of the precuneus led to a better ability to recognize the context of a particular memory—that is, participants were better able to recognize previously seen pictures and identify the color of the pictures.

In **transcranial direct current stimulation (tDCS)**, low current electricity is applied directly to the scalp in a continuous fashion. In TMS, the coil is kept away from the scalp and the current can either be a single pulse or continuous, but in tDCS, the coil is in...
contact with the scalp and the current is continuous. This technique has a number of medical applications, including improving cognitive performance after stroke, alleviating depression, and reducing memory deficits in early Alzheimer’s disease (Goldsworthy, Pitcher, & Ridding, 2015; Hordacre, Moezzi, & Ridding, 2018). In research, tDCS can help localize function in the brain. If current is applied to an area of the brain, and performance on a certain task improves (or gets worse), that area must be involved in that cognitive skill.

For example, Schaal, Javadi, Halpern, Pollok, and Banissy (2015) did an interesting study using tDCS, looking at the role of the right parietal cortex in the recognition of recently learned melodies. Melody refers to the main line of a song, the part of the music that we generally hum when we think of a song. In the study, participants listened to novel melodies, derived from folk songs, but otherwise never heard before. After the music had been listened to, the tDCS was applied directly to the scalp above the right parietal cortex. In a second experiment, the tDCS was also applied to the left parietal cortex to serve as a control. In addition, there was a group of participants who were set up with all the tDCS equipment, but the current was never turned on, which also served as a control. When the tDCS was being applied to the right parietal cortex, participants recognized fewer of the melodies as old in an old/new recognition test than did participants in the control condition. Thus, stimulation of the right parietal cortex interferes with memory for musical melodies. Because of this interference, one can speculate that the right parietal cortex must be an area critical in music perception or music learning. In deep brain stimulation (DBS), a device is implanted directly into the brain, which then sends electrical impulses to specific regions of the brain. DBS is exclusively implanted for medical reasons—it is useful for alleviating symptoms of Parkinson’s disease and other brain-based motor disorders. It has also been successful in treating major affective disorders, such as obsessive-compulsive disorder and major depression (Mavridis, 2015). In research, it is only used with patients who have the DBS device implanted for medical reasons. However, in a population of severely epileptic patients, J. P. Miller et al. (2015) found that DBS pulses to the fornix area of the brain improved the patients’ performance on a variety of memory tasks.

### SECTION QUIZ

1. If a cognitive neuroscientist wanted to determine the time course of an event recognition in the brain, which techniques would give that researcher the best temporal resolution?
   a. PET
   b. EEG
   c. DBS
   d. None of the above

2. Which correctly states the logic behind neuroimaging techniques?
   a. Blood flows to the areas of the brain being used; neuroimaging techniques can detect this flow.
   b. Electrical impulses cause chain reaction in the brain, which allow researchers to identify white-matter tracts.
3. Diffusion tensor imaging is especially good at detecting
   a. White-matter tracts that are used in a particular cognitive task
   b. Whether a patient has Parkinson’s disease or not
   c. Cognitive manipulations that work through the brainstem rather than the cortex
   d. All of the above

4. In transcranial magnetic stimulation (TMS), a coil is placed near the scalp, which generates an electrical current in the brain. Which statement is true about TMS?
   a. The coil is not placed directly on the skull.
   b. The stimulation can either improve performance or inhibit performance, depending on the task.
   c. Either a single pulse can be applied or a continuous current can be applied.
   d. All of the above are true.

1. b
2. a
3. a
4. d

NEUROPSYCHOLOGY: MEMORY DEFICITS AND AMNESIA

The oldest methodology for examining the relation between memory and the brain is to study patients with brain damage. This is because examining patients with neuropsychological deficits does not require technology. However, researchers must first locate patients who have suffered brain damage 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, a foreperson on a railroad crew, was severely injured when a poorly timed dynamite blast shot a metal rod through his frontal lobe. Although he survived the accident and lived for many years afterward, 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 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. Because damage to an area causes deficits in a particular function, such as working memory, then it is thought that the brain area must play some role in that function, in this case, working memory.

Just over 100 years after Gage, in September 1953, a 27-year-old man known to science as H. M. underwent risky experimental surgery to alleviate symptoms of debilitating epilepsy. During the surgery, parts of his medial temporal lobe, including most of both of his hippocampi, were removed on both sides. As a direct result of the surgical procedure, H. M. suffered from strong anterograde amnesia—that is, a deficit in learning and memory.
retaining new information. He could not learn new facts, such as memorizing a phone number. He also suffered some relatively mild retrograde amnesia—that is, the loss of memory of events before the injury. Although this surgery has never been repeated on any other human being, H. M.’s memory was studied extensively for the next 50 years (Corkin, 2002). H. M. 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, with memories just barely different from those of people without brain damage, to very severe amnesia. 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 (B. A. Wilson, 2009).

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 H. M.—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.

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, few illegal drugs have as profound an effect on our nervous system as 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. Certain drugs do improve our alertness, influence how long we can stay awake and focused, and perhaps give us more time to learn. There are some data to suggest that nicotine enhances some cognitive processes, including memory (Valentine & Sofuglu, 2018). Nonetheless, nicotine has terrible health risks, so it should be avoided at all costs. However, to date, no other drug has been shown to improve memory efficiency in normal adults. On the other hand, there is no doubt that some drugs 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. Although these drugs reduce anxiety, they may also have a profound amnesic effect on some patients (Helmes & Østbye, 2015). Thus, anyone taking drugs such as Valium (diazepam), Atavan (lorazepam), or Xanax (alprazolam) must be aware of the potential deficits in learning while using these medications.

The only prescription drugs available to improve memory are cholinergics (Haense et al., 2012; McDaniel, Maier, & Einstein, 2002; Risacher et al., 2013). 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 the neurotransmitter acetylcholine, used by many memory circuits. 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. Aricept (donepezil) is now a commonly prescribed drug for patients with early Alzheimer’s or other forms of cognitive dementia—it allows some temporary improvement of memory performance and speech fluency (Adlimoghaddam, Neuendorff, Roy, & Albensi, 2018). In late Alzheimer’s, the improvement may be minimal and is often discontinued for medical reasons, but research still documents some improvement (Adlimoghaddam et al., 2018).

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, some research suggests that caffeine, although it may help people study by allowing them to remain awake longer, reduces the efficiency of learning (Mednick, Cai, Kanady, & Drummond, 2008). Caffeine may hurt learning by decreasing the number of items learned per unit of time; one may be able to study more hours but learn less during each of those hours. For example, if you could learn 20 new items of information per hour without caffeine, you might only be able to learn 18 new items per hour while using caffeine. But if caffeine allows you to study for three hours instead of two, the additional time would compensate for the lower efficiency.

On the herbal side, the leaves of the ginkgo tree have been used for 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 of this herb 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 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, however, that clearly interfere with memory, causing temporary amnesia. Benzodiazepines, such as diazepam (i.e., Valium), lorazepam (i.e., Ativan), triazolam, and midazolam, are the most commonly consumed drugs in the world because of their effects on anxiety, insomnia, and muscle relaxation (Kaplan, 2005; Risacher et al., 2013). 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, our knowledge of the world (Bacon, Schwartz, Paire-Ficout, & Izaute, 2007). The benzodiazepines that are 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 is 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
language. Variance in this gene is associated with differences in the structure of the occipital lobe, particularly an area associated with object identification (Uddén, Snijders, Fisher, & Hagoort, 2017). In addition to helping us understand the nature of human memory, it is likely that correlating neural function with genetic variation will have a great many practical applications, particularly in the treatment of neurological disorders.

**CASE EXAMPLE: THE NEUROSCIENCE OF FACE MEMORY**

Certain areas in the temporal lobe of the brain specialize in face recognition. In particular, the *fusiform face area (FFA)* in the inferior-temporal cortex appears to specialize in face recognition (Harvey & Burgund, 2012; Hasinki & Sederberg, 2016; Kanwisher, 2004). This area of the brain is selectively activated when people are looking at unfamiliar faces and when they are recognizing familiar faces (Hasinki & Sederberg, 2016). The specialized areas of the brain appear to be evolutionarily determined areas for face recognition; equivalent areas in monkeys are also maximally responsive to monkey faces. In addition to the FFA, an area of the occipital lobe has also been identified as crucial to face recognition. This area in the occipital lobe is known, aptly, as the *occipital face area (OFA)* (Large, Cavina-Pratesi, Vilis, & Culham, 2008). Both of these areas are activated during face recognition tasks in functional magnetic resonance imaging (fMRI) studies (Grill-Spector, Knouf, & Kanwisher, 2004), and damage to these areas is associated with deficits in face recognition.

Damage to the FFA results in a neuropsychological condition called **prosopagnosia**. Prosopagnosia, also called face blindness, is defined as an acquired deficit in recognizing familiar faces (see Duchaine & Yovel, 2015; Shlomo, DeGutis, D’Esposito, & Robertson, 2007). It usually results from damage to the FFA and surrounding areas in the temporal lobe, typically as the result of a stroke or another event that prevents oxygen from reaching these areas of the brain. People suffering from prosopagnosia lose the ability to recognize familiar faces. They know that faces are faces but can no longer distinguish faces of people that they know. This includes recognizing the faces of people they have known for a long time and learning to recognize the faces of new individuals. Most people can recognize a face regardless of context. You can recognize your father’s face, regardless of whether you see it in the newspaper, on the “fan-cam” at a sporting event, or in a family photo album. A patient with prosopagnosia cannot do this. If the damage is restricted to the FFA, the patient is still able to recognize familiar people by their voices, their particular way of walking, or other clues. For example, if the patient’s spouse always wears a trademark hat, recognition can occur by noting the hat. Also, patients with prosopagnosia can recognize objects other than faces.

There is also a rare condition called developmental prosopagnosia. Developmental prosopagnosia is a congenital deficit in face recognition. Developmental prosopagnosia appears early in life and is not associated with any particular brain trauma (Dalrymple, Elison, & Duchaine, 2017; Duchaine & Yovel, 2015). Patients with developmental prosopagnosia show normal vision and normal visual perception in all areas other than face recognition (Dalrymple et al., 2017). Typically, patients with developmental prosopagnosia have normal facial expression recognition and can identify features of faces, such as eyes and mouths, in non-face images.

**Fusiform face area (FFA):** A part of the brain in the inferior-temporal cortex that appears to specialize in face recognition.

**Occipital face area (OFA):** An area of the occipital lobe that has been identified as crucial to face recognition.

**Prosopagnosia:** An acquired deficit in face recognition and face identification caused by brain damage.

**Developmental prosopagnosia:** A congenital deficit in face recognition.
prosopagnosia can recognize some faces, particularly those of close family and friends. However, they are slower to recognize familiar faces and slower to learn new faces than a comparison sample of people without developmental prosopagnosia. Because developmental prosopagnosia is limited to face recognition, people who suffer from it can compensate by focusing on auditory features of a person in order to recognize that person. They can also rely on other visual processes to help them with face recognition. Bennetts, Butcher, Lander, Udale, and Bate (2015) showed that patients with developmental prosopagnosia showed better face recognition when those faces were moving than when they were stationary. This suggests that developmental prosopagnosia is not as debilitating as one might think, if patients learn compensatory strategies.

In summary, face memory seems to be an integrated process involving both areas in the occipital lobe, which we know are involved with the perception of faces, and areas in the temporal lobe, which we know are involved in recognizing complex objects and memory for those objects. Damage to these areas results in prosopagnosia, which is a selective deficit at recognizing and remembering human faces. Developmental prosopagnosia occurs when people have a deficit in facial recognition seemingly from birth. Most of us, however, are constantly using these regions of the brain as we negotiate the sea of human faces that surrounds us.

Summary

The cognitive psychology of memory is increasingly influenced by the neuroscience of memory; together these areas of research form the hybrid field known as cognitive neuroscience. Cognitive neuroscience 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 cellular level, the brain is composed of billions of neurons that talk to each other electrically. At higher levels, several key components of the brain are 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—disorders of memory. Neuroimaging studies reveal how these areas are active during memory processes. There are several common neuroscience techniques: PET scans, MRI and fMRI, MEG, EEG, TMS, tDCS, and DBS. With respect to neuroimaging, fMRI has become the state of the art in cognitive neuroscience.

The brain uses chemicals called neurotransmitters to bridge the synapses between cells. Neurotransmitter function can be influenced by drugs. Some drugs, such as benzodiazepines, interfere with memory processing. The search continues for drugs that can improve memory performance directly. A strong connection exists between some odors and certain strong autobiographical memories by way of the limbic system and between music and memory via many areas of the brain, especially the auditory cortex in the temporal lobes, the sensory cortex in the parietal lobes, and the prefrontal cortex in the frontal lobe. Imaging genetics examines the relation between variation in the human genome and variations in brain anatomy and function. The fusiform face area (FFA) is an area of the brain responsible for identifying individual faces, whereas the occipital face area (OFA) is responsible for identifying objects that are faces. Prosopagnosia is the failure to identify individual faces. It is caused by damage to the fusiform face area.
Key Terms

- action potential 38
- Alzheimer’s disease 36
- amnesia 44
- amygdala 44
- anterograde amnesia 55
- axon 38
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personal autobiographical memory. Most people can describe the relation of a particular smell to some salient event from their life (R. Herz, 2007; Schwartz & Krantz, 2019). 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 Marcel Proust (1928) described how the scent of a French pastry called a *madeleine* transported him back to his childhood in the south of France. 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 also has ties to emotion. The memories elicited by odor are usually emotional memories.

The neural reason for this strong connection among our sense of smell, emotion, and 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 (R. S. Herz, 2005).

**GENETIC APPROACHES**

One of the newest approaches to the study of memory and the human brain comes from relating specific genes to specific neural structures and both to memory performance (Mei et al., 2018). Despite its newness, this field is giving us a rich understanding of how genes code for the brain structures that affect memory and other cognitive processes. At present, more than 100 genes have been correlated with memory performance in human beings (Munoz, 2017). Although correlating gene variation with individual differences has been around for a bit longer, the correlation of gene variation with the results of neuroimaging studies is much newer. Indeed, the term **imaging genetics** is now used to describe the relation between genetic variation and variation in brain anatomy. This research requires “big data” analyses, comparing studies looking at variation in genes across many people and correlating with huge sets of neuroimaging data (Munoz, 2017). For example, Schulze, Vargha-Khadem, and Mishkin (2018) were interested in FOXP2 gene and the actions of working memory. The FOXP2 gene is a gene that has been correlated with the development of language and memory in humans. A mutated form of the FOXP2 gene is associated with specific language impairment, in which people have normal intelligence but deficits in the acquisition and use of language (Schulze et al., 2018). Working memory is a term that refers to the various systems that are responsible for holding information over the short term, a topic we will discuss at length in the next chapter. Schulze et al. showed that people with the mutation that causes specific language impairment also have deficits in working memory. Another gene, the CNTNAP2, is also associated with memory and
language. Variance in this gene is associated with differences in the structure of the occipital lobe, particularly an area associated with object identification (Uddén, Snijders, Fisher, & Hagoort, 2017). In addition to helping us understand the nature of human memory, it is likely that correlating neural function with genetic variation will have a great many practical applications, particularly in the treatment of neurological disorders.

**CASE EXAMPLE: THE NEUROSCIENCE OF FACE MEMORY**

Certain areas in the temporal lobe of the brain specialize in face recognition. In particular, the *fusiform face area (FFA)* in the inferior-temporal cortex appears to specialize in face recognition (Harvey & Burgund, 2012; Hasinski & Sederberg, 2016; Kanwisher, 2004). This area of the brain is selectively activated when people are looking at unfamiliar faces and when they are recognizing familiar faces (Hasinski & Sederberg, 2016). The specialized areas of the brain appear to be evolutionarily determined areas for face recognition; equivalent areas in monkeys are also maximally responsive to monkey faces. In addition to the FFA, an area of the occipital lobe has also been identified as crucial to face recognition. This area in the occipital lobe is known, aptly, as the *occipital face area (OFA)* (Large, Cavina-Pratesi, Vilis, & Culham, 2008). Both of these areas are activated during face recognition tasks in functional magnetic resonance imaging (fMRI) studies (Grill-Spector, Knouf, & Kanwisher, 2004), and damage to these areas is associated with deficits in face recognition.

Damage to the FFA results in a neuropsychological condition called *prosopagnosia*. Prosopagnosia, also called face blindness, is defined as an acquired deficit in recognizing familiar faces (see Duchaine & Yovel, 2015; Shlomo, DeGutis, D’Esposito, & Robertson, 2007). It usually results from damage to the FFA and surrounding areas in the temporal lobe, typically as the result of a stroke or another event that prevents oxygen from reaching these areas of the brain. People suffering from prosopagnosia lose the ability to recognize familiar faces. They know that faces are faces but can no longer distinguish faces of people that they know. This includes recognizing the faces of people they have known for a long time and learning to recognize the faces of new individuals. Most people can recognize a face regardless of context. You can recognize your father’s face, regardless of whether you see it in the newspaper, on the “fan-cam” at a sporting event, or in a family photo album. A patient with prosopagnosia cannot do this. If the damage is restricted to the FFA, the patient is still able to recognize familiar people by their voices, their particular way of walking, or other clues. For example, if the patient’s spouse always wears a trademark hat, recognition can occur by noting the hat. Also, patients with prosopagnosia can recognize objects other than faces.

There is also a rare condition called developmental prosopagnosia. Developmental prosopagnosia is a congenital deficit in face recognition. Developmental prosopagnosia appears early in life and is not associated with any particular brain trauma (Dalrymple, Elison, & Duchaine, 2017; Duchaine & Yovel, 2015). Patients with developmental prosopagnosia show normal vision and normal visual perception in all areas other than face recognition (Dalrymple et al., 2017). Typically, patients with developmental
prosopagnosia can recognize some faces, particularly those of close family and friends. However, they are slower to recognize familiar faces and slower to learn new faces than a comparison sample of people without developmental prosopagnosia. Because developmental prosopagnosia is limited to face recognition, people who suffer from it can compensate by focusing on auditory features of a person in order to recognize that person. They can also rely on other visual processes to help them with face recognition. Bennetts, Butcher, Lander, Udale, and Bate (2015) showed that patients with developmental prosopagnosia showed better face recognition when those faces were moving than when they were stationary. This suggests that developmental prosopagnosia is not as debilitating as one might think, if patients learn compensatory strategies.

In summary, face memory seems to be an integrated process involving both areas in the occipital lobe, which we know are involved with the perception of faces, and areas in the temporal lobe, which we know are involved in recognizing complex objects and memory for those objects. Damage to these areas results in prosopagnosia, which is a selective deficit at recognizing and remembering human faces. Developmental prosopagnosia occurs when people have a deficit in facial recognition seemingly from birth. Most of us, however, are constantly using these regions of the brain as we negotiate the sea of human faces that surrounds us.

Summary

The cognitive psychology of memory is increasingly influenced by the neuroscience of memory; together these areas of research form the hybrid field known as cognitive neuroscience. Cognitive neuroscience 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 cellular level, the brain is composed of billions of neurons that talk to each other electrically. At higher levels, several key components of the brain are 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—disorders of memory. Neuroimaging studies reveal how these areas are active during memory processes. There are several common neuroscience techniques: PET scans, MRI and fMRI, MEG, EEG, TMS, tDCS, and DBS. With respect to neuroimaging, fMRI has become the state of the art in cognitive neuroscience. The brain uses chemicals called neurotransmitters to bridge the synapses between cells. Neurotransmitter function can be influenced by drugs. Some drugs, such as benzodiazepines, interfere with memory processing. The search continues for drugs that can improve memory performance directly. A strong connection exists between some odors and certain strong autobiographical memories by way of the limbic system and between music and memory via many areas of the brain, especially the auditory cortex in the temporal lobes, the sensory cortex in the parietal lobes, and the prefrontal cortex in the frontal lobe. Imaging genetics examines the relation between variation in the human genome and variations in brain anatomy and function. The fusiform face area (FFA) is an area of the brain responsible for identifying individual faces, whereas the occipital face area (OFA) is responsible for identifying objects that are faces. Prosopagnosia is the failure to identify individual faces. It is caused by damage to the fusiform face area.
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