

What does Capgras syndrome teach us about the brain itself? One lesson involves the fact that many different parts of the brain are needed for even the simplest achievement. In order to recognize your father, for example, one part of your brain needs to store the factual memory of what he looks like. Another part of the brain is responsible for analyzing the visual input you receive when looking at a face. Yet another brain area has the job of comparing this now-analyzed input to the factual information provided from memory, to determine whether there's a match. Another site provides the emotional evaluation of the input. A different site presumably assembles the data from all these other sites—and registers the fact that the face being inspected does match the factual recollection of your father's face, and also produces a warm sense of familiarity.

Usually, all these brain areas work together, allowing the recognition of your father's face to go smoothly forward. If they don't work together—that is, if coordination among these areas is disrupted—yet another area works to make sure you offer reasonable hypotheses about this disconnect, and not zany ones. (In other words, if your father looks less familiar to you on some occasion, you're likely to explain this by saying, "I guess he must have gotten new glasses" rather than "I bet he's been replaced by a robot.")

Unmistakably, this apparently easy task—seeing your father and recognizing who he is—requires multiple brain areas. The same is true of most tasks, and in this way Capgras syndrome illustrates this crucial aspect of brain function.

### TEST YOURSELF

1. What are the symptoms of Capgras syndrome, and why do they suggest a two-part explanation for how you recognize faces?

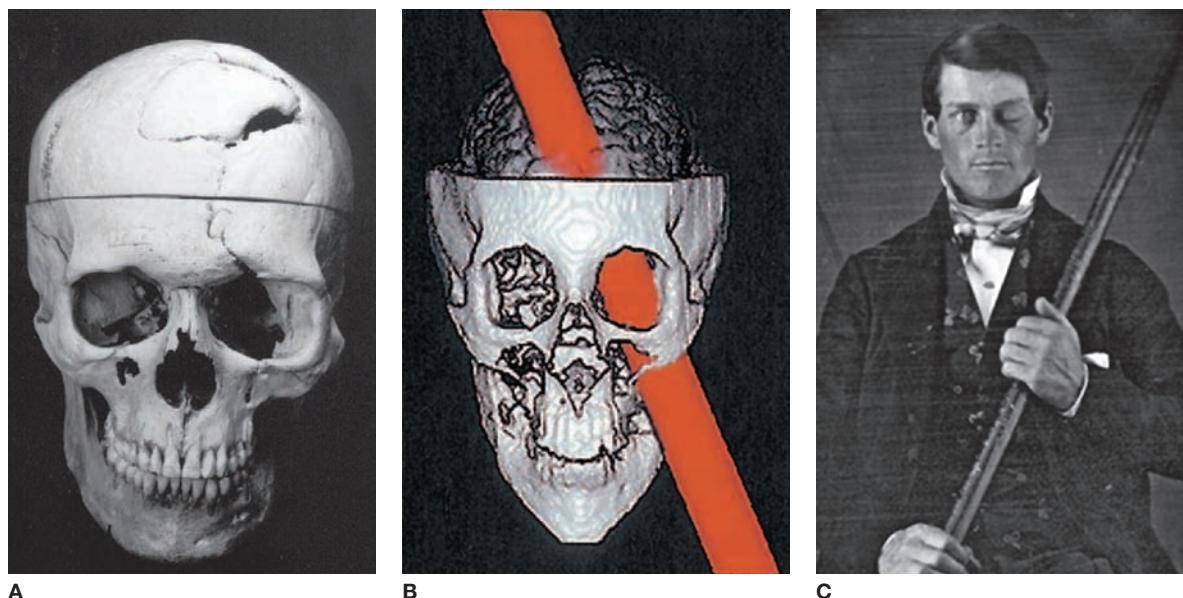
## The Study of the Brain

In order to discuss Capgras syndrome, we needed to refer to different brain areas and had to rely on several different research techniques. In this way, the syndrome also illustrates another point—that this is a domain in which we need some technical foundations before we can develop our theories. Let's start building those foundations.

The human brain weighs (on average) a bit more than 3 pounds (roughly 1.4 kg), with male brains weighing about 10% more than female brains (Hartmann, Ramseier, Gudat, Mihatsch, & Polasek, 1994). The brain is roughly the size of a small melon, yet this compact structure has been estimated to contain 86 billion nerve cells (Azevedo et al., 2009). Each of these cells is connected to 10,000 or so others—for a total of roughly 860 trillion connections. The brain also contains a huge number of *glial cells*, and we'll have more to say about all of these individual cells later on in the chapter. For now, though, how should we begin our study of this densely packed, incredibly complex organ?

One place to start is with a simple fact we've already met: that different parts of the brain perform different jobs. Scientists have known this fact about the brain for many years, thanks to clinical evidence showing that the symptoms produced by brain damage depend heavily on the location of the damage. In 1848, for example, a horrible construction accident caused Phineas Gage to suffer damage in the frontmost part of his brain

**FIGURE 2.4 PHINEAS GAGE**



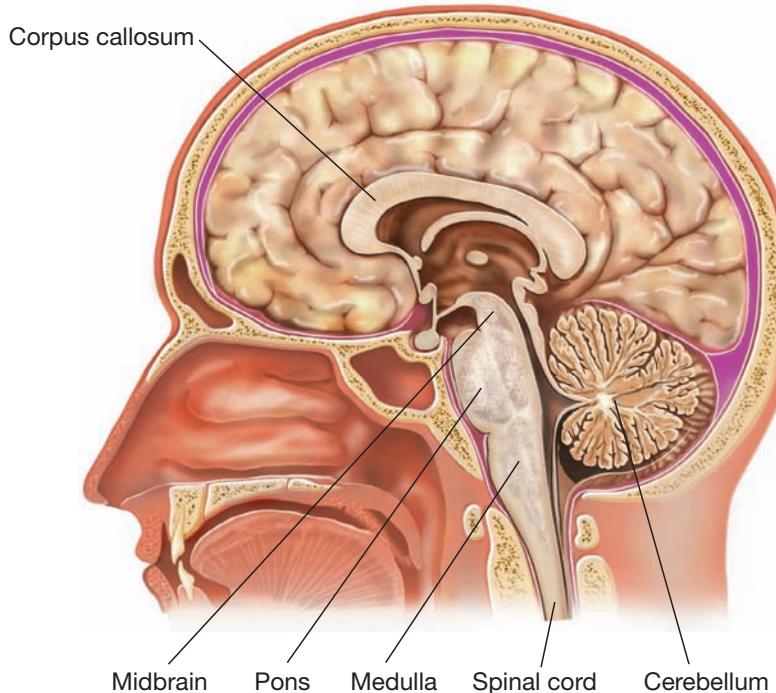
Phineas Gage was working as a construction foreman when some blasting powder misfired and launched a piece of iron into his cheek and through the front part of his brain. Remarkably, Gage survived and continued to live a fairly normal life, but his pattern of intellectual and emotional impairments provide valuable cues about the function of the brain's frontal lobes. Panel A is a photo of Gage's skull; the drawing in Panel B depicts the iron bar's path as it blasted through his head. Panel C is an actual photograph of Gage, and he's holding the bar that went through his brain!

(see Figure 2.4), and this damage led to severe personality and emotional problems. In 1861, physician Paul Broca noted that damage in a different location, on the left side of the brain, led to a disruption of language skills. In 1911, Édouard Claparède (1911/1951) reported his observations with patients who suffered from profound memory loss produced by damage in still another part of the brain.

Clearly, therefore, we need to understand brain functioning with reference to brain anatomy. Where was the damage that Gage suffered? Where was the damage in Broca's patients or Claparède's? In this section, we fill in some basics of brain anatomy.

### Hindbrain, Midbrain, Forebrain

The human brain is divided into three main structures: the hindbrain, the midbrain, and the forebrain. The **hindbrain** is located at the very top of the spinal cord and includes structures crucial for controlling key life



### GROSS ANATOMY OF A BRAIN SHOWING BRAIN STEM

The pons and medulla are part of the hindbrain. The medulla controls vital functions such as breathing and heart rate. The pons (Latin for “bridge”) is the main connection between the cerebellum and the rest of the brain.

functions. It’s here, for example, that the rhythm of heartbeats and the rhythm of breathing are regulated. The hindbrain also plays an essential role in maintaining the body’s overall tone. Specifically, the hindbrain helps maintain the body’s posture and balance; it also helps control the brain’s level of alertness.

The largest area of the hindbrain is the **cerebellum**. For many years, investigators believed this structure’s main role was in the coordination of bodily movements and balance. Research indicates, however, that the cerebellum plays various other roles and that damage to this organ can cause problems in spatial reasoning, in discriminating sounds, and in integrating the input received from various sensory systems (Bower & Parsons, 2003).

The **midbrain** has several functions. It plays an important part in coordinating movements, including the precise movements of the eyes as they explore the visual world. Also in the midbrain are circuits that relay auditory information from the ears to the areas in the forebrain where this information is processed and interpreted. Still other structures in the midbrain help to regulate the experience of pain.

For our purposes, though, the most interesting brain region (and, in humans, the largest region) is the **forebrain**. Drawings of the brain (like the one shown in Figure 2.2) show little other than the forebrain, because this structure surrounds (and so hides from view) the entire midbrain and most of the hindbrain. Of course, only the outer surface of the forebrain—the **cortex**—is visible in such pictures. In general, the word “cortex” (from the Latin word for “tree bark”) refers to an organ’s outer surface, and many organs each have their own cortex; what’s visible in the drawing, then, is the *cerebral cortex*.

The cortex is just a thin covering on the outer surface of the forebrain; on average, it’s a mere 3 mm thick. Nonetheless, there’s a great deal of cortical tissue; by some estimates, the cortex makes up 80% of the human brain. This considerable volume is made possible by the fact that the cerebral cortex, thin as it is, consists of a large sheet of tissue. If stretched out flat, it would cover more than 300 square inches, or roughly 2,000 cm<sup>2</sup>. (For comparison, this is an area roughly 20% greater than the area covered by an extra-large—18 inch, or 46 cm—pizza.) But the cortex isn’t stretched flat; instead, it’s crumpled up and jammed into the limited space inside the skull. It’s this crumpling that produces the brain’s most obvious visual feature—the wrinkles, or **convolutions**, that cover the brain’s outer surface.

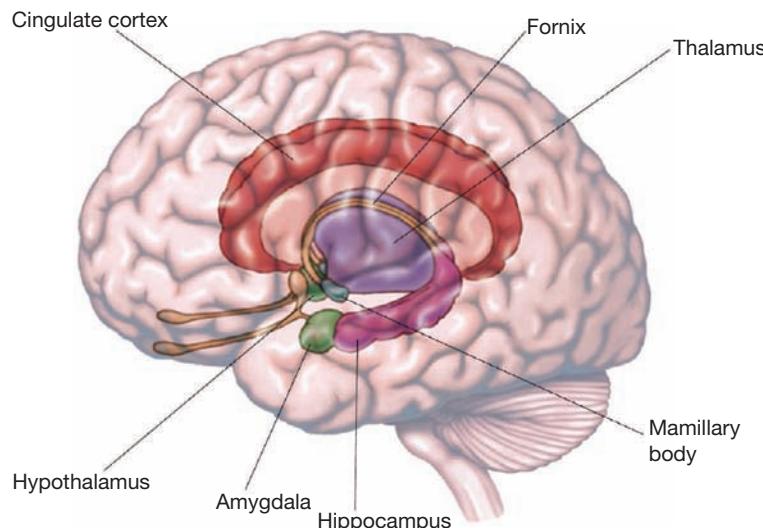
Some of the “valleys” between the wrinkles are actually deep grooves that divide the brain into different sections. The deepest groove is the **longitudinal fissure**, running from the front of the brain to the back, which separates the left **cerebral hemisphere** from the right. Other fissures divide the cortex in each hemisphere into four lobes (again, look back at Figure 2.2), and these are named after the bones that cover them—bones that, as a group, make up the skull. The **frontal lobes** form the front of the brain, right behind the forehead. The **central fissure** divides the frontal lobes on each side of the brain from the **parietal lobes**, the brain’s topmost part. The bottom edge of the frontal lobes is marked by the **lateral fissure**, and below it are the **temporal lobes**. Finally, at the very back of the brain, connected to the parietal and temporal lobes, are the **occipital lobes**.

## Subcortical Structures

Hidden from view, underneath the cortex, are several **subcortical structures**. One of these structures, the **thalamus**, acts as a relay station for nearly all the sensory information going to the cortex. Directly underneath the thalamus is the **hypothalamus**, a structure that plays a crucial role in controlling behaviors that serve specific biological needs—behaviors that include eating, drinking, and sexual activity.

Surrounding the thalamus and hypothalamus is another set of structures that form the **limbic system**. Included here is the amygdala, and close by is the **hippocampus**, both located underneath the cortex in the temporal lobe (plurals: amygdalae and hippocampi; see Figure 2.5). These structures

**FIGURE 2.5 THE LIMBIC SYSTEM AND THE HIPPOCAMPUS**



Color is used in this drawing to help you visualize the arrangement of these brain structures. Imagine that the cortex is semitransparent, allowing you to look into the brain to see the (subcortical) structures highlighted here. The limbic system includes a number of subcortical structures that play a crucial role in learning and memory and in emotional processing.

are essential for learning and memory, and the patient H.M., discussed in Chapter 1, developed his profound amnesia after surgeons removed large portions of these structures—strong confirmation of their role in the formation of new memories.

We mentioned earlier that the amygdala plays a key role in emotional processing, and this role is reflected in many findings. For example, presentation of frightful faces causes high levels of activity in the amygdala (Williams et al., 2006). Likewise, people ordinarily show more complete, longer-lasting memories for emotional events, compared to similar but emotionally flat events. This memory advantage for emotional events is especially pronounced in people who showed greater activation in the amygdala while they were witnessing the event in the first place. Conversely, the memory advantage for emotional events is diminished (and may not be observed at all) in people who (through sickness or injury) have suffered damage to the amygdala.

## Lateralization

Virtually all parts of the brain come in pairs, and so there is a hippocampus on the left side of the brain and another on the right, a left-side amygdala and a right-side one. The same is true for the cerebral cortex itself: There is a temporal cortex (i.e., a cortex of the temporal lobe) in the left hemisphere and another in the right, a left occipital cortex and a right one, and so on. In all cases, cortical and subcortical, the left and right structures in each pair have roughly the same shape and the same pattern of connections to other brain areas. Even so, there are differences in function between the left-side and right-side structures, with each left-hemisphere structure playing a somewhat different role from the corresponding right-hemisphere structure.

Let's remember, though, that the two halves of the brain work together—the functioning of one side is closely integrated with that of the other side. This integration is made possible by the **commissures**, thick bundles of fibers that carry information back and forth between the two hemispheres. The largest commissure is the **corpus callosum**, but several other structures also make sure that the two brain halves work as partners in almost all mental tasks.

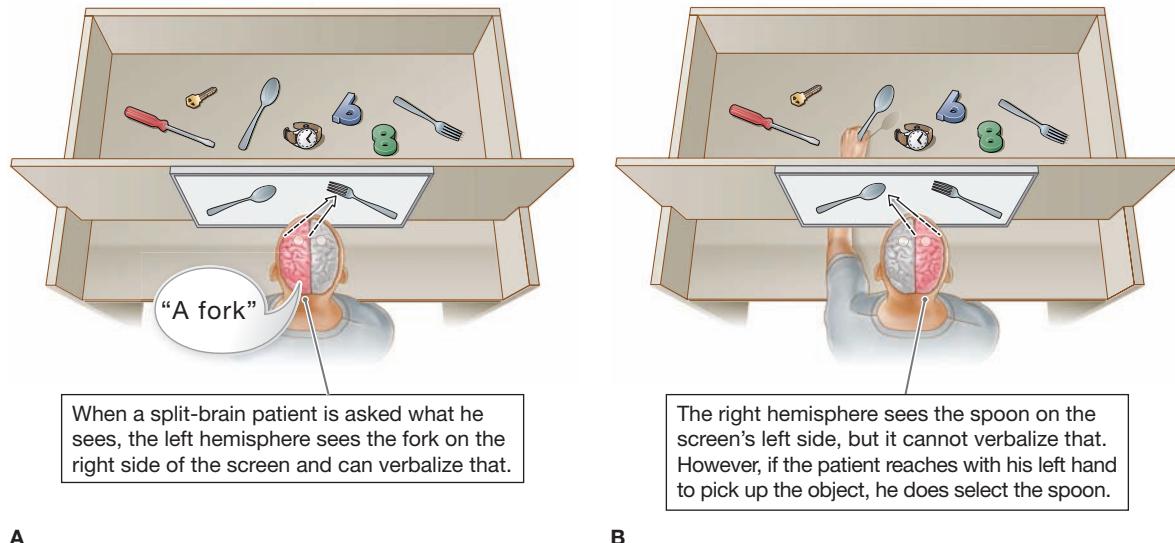
In certain cases, though, there are medical reasons to sever the corpus callosum and some of the other commissures. (For many years, this surgery was a last resort for extreme cases of epilepsy.) The person is then said to be a “split-brain patient”—still having both brain halves, but with communication between the halves severely limited. Research with these patients has taught us a great deal about the specialized function of the brain’s two hemispheres. It has provided evidence, for example, that many aspects of language processing are lodged in the left hemisphere, while the right hemisphere seems crucial for a number of tasks involving spatial judgment (see *Figure 2.6*).

However, it’s important not to overstate the contrast between the two brain halves, and it’s misleading to claim (as some people do) that we need to silence our “left-brain thinking” in order to be more creative, or that intuitions grow out of “right-brain thinking.” These claims do begin with a kernel of truth, because some elements of creativity depend on specialized processing in the right hemisphere (see, e.g., Kounios & Beeman, 2015). Even so, whether we’re examining creativity or any other capacity, the two halves of the brain have to work together, with each hemisphere making its own contribution to the overall performance. Therefore, “shutting down” or “silencing” one hemisphere, even if that were biologically possible, wouldn’t allow you new achievements, because the many complex, sophisticated skills we each display (including creativity, intuition, and more) depend on the whole brain. In other words, our hemispheres are not cerebral competitors, each trying to impose its style of thinking on the other. Instead, the hemispheres pool their specialized capacities to produce a seamlessly integrated, single mental self.

### TEST YOURSELF

2. What is the cerebral cortex?
3. What are the four major lobes of the forebrain?
4. Identify some of the functions of the hippocampus, the amygdala, and the corpus callosum.

**FIGURE 2.6 STUDYING SPLIT-BRAIN PATIENTS**



In this experiment, the patient is shown two pictures, one of a spoon and one of a fork (Panel A). If asked what he sees, his verbal response is controlled by the left hemisphere, which has seen only the fork (because it's in the right visual field). However, if asked to pick up the object shown in the picture, the patient—reaching with his left hand—picks up the spoon (Panel B). That happens because the left hand is controlled by the right hemisphere, and this hemisphere receives visual information from the left-hand side of the visual world.

## Sources of Evidence about the Brain

How can we learn about these various structures—and many others that we haven't named? Cognitive neuroscience relies on many types of evidence to study the brain and nervous system. Let's look at some of the options.

### Data from Neuropsychology

We've already encountered one form of evidence—the study of individuals who have suffered brain damage through accident, disease, or birth defect. The study of these cases generally falls within the domain of *neuropsychology*: the study of the brain's structures and how they relate to brain function. Within neuropsychology, the specialty of *clinical neuropsychology* seeks (among other goals) to understand the functioning of intact, undamaged brains by means of careful scrutiny of cases involving brain damage.

Data drawn from clinical neuropsychology will be important throughout this text. For now, though, we'll emphasize that the symptoms resulting from

brain damage depend on the site of the damage. A **lesion** (a specific area of damage) in the hippocampus produces memory problems but not language disorders; a lesion in the occipital cortex produces problems in vision but spares the other sensory modalities. Likewise, the consequences of brain lesions depend on which hemisphere is damaged. Damage to the left side of the frontal lobe, for example, is likely to produce a disruption of language use; damage to the right side of the frontal lobe generally doesn't have this effect. In obvious ways, then, these patterns confirm the claim that different brain areas perform different functions. In addition, these patterns provide a rich source of data that help us develop and test hypotheses about those functions.

## Data from Neuroimaging

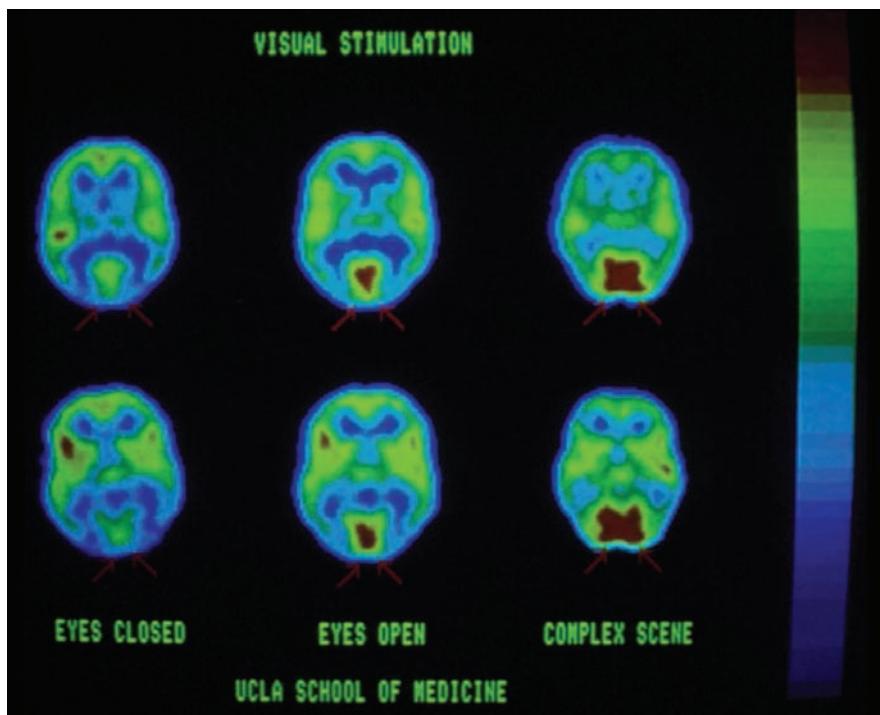
Further insights come from **neuroimaging techniques**. There are several types of neuroimaging, but they all produce precise, three-dimensional pictures of a living brain. Some neuroimaging procedures provide *structural* imaging, generating a detailed portrait of the shapes, sizes, and positions of the brain's components. Other procedures provide *functional* imaging, which tells us about activity levels throughout the brain.

For many years, **computerized axial tomography (CT scans)** was the primary tool for structural imaging, and **positron emission tomography (PET scans)** was used to study the brain's activity. CT scans rely on X-rays and so—in essence—provide a three-dimensional X-ray picture of the brain. PET scans, in contrast, start by introducing a tracer substance such as glucose into the patient's body; the molecules of this tracer have been tagged with a low dose of radioactivity, and the scan keeps track of this radioactivity, allowing us to tell which tissues are using more of the glucose (the body's main fuel) and which ones are using less.

For each type of scan, the primary data (X-rays or radioactive emissions) are collected by a bank of detectors placed around the person's head. A computer then compares the signals received by each of the detectors and uses this information to construct a three-dimensional map of the brain—a map of structures from a CT scan, and a map showing activity levels from a PET scan.

More recent studies have turned to two newer techniques, introduced earlier in the chapter. **Magnetic resonance imaging (MRI scans)** relies on the magnetic properties of the atoms that make up the brain tissue, and it yields fabulously detailed pictures of the brain. MRI scans provide structural images, but a closely related technique, **functional magnetic resonance imaging (fMRI scans)**, provides functional imaging. The fMRI scans measure the oxygen content in blood flowing through each region of the brain; this turns out to be an accurate index of the level of neural activity in that region. In this way, fMRI scans offer an incredibly precise picture of the brain's moment-by-moment activities.

The results of structural imaging (CT or MRI scans) are relatively stable, changing only if the person's brain structure changes (because of an injury, perhaps, or the growth of a tumor). The results of PET or fMRI scans, in contrast, are highly variable, because the results depend on what task the person is performing. We can therefore use these latter techniques to explore brain function—using fMRI scans, for example, to determine which brain sites are



## PET SCANS

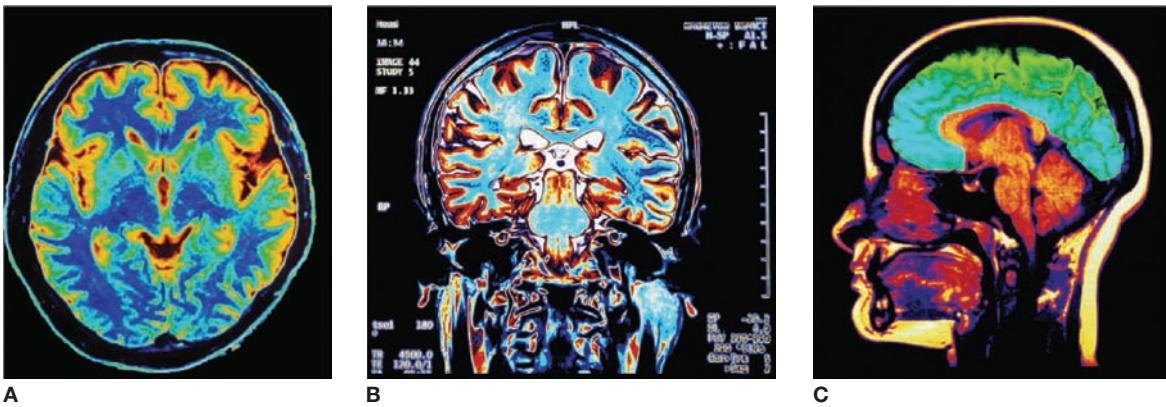
PET scans measure how much glucose (the brain's fuel) is being used at specific locations within the brain; this provides a measurement of each location's activity level at a certain moment in time. In the figure, the brain is viewed from above, with the front of the head at the top and the back of the head at the bottom. The various colors indicate relative activity levels (an actual brain is uniformly colored), using the palette shown on the right side of the figure. Dark blue indicates a low level of activity; red indicates a high level. And as the figure shows, visual processing involves increased activity in the occipital lobe.

especially activated when someone is making a moral judgment or trying to solve a logic problem. In this way, the neuroimaging data can provide crucial information about how these activities are made possible by specific patterns of functioning within the brain.

## Data from Electrical Recording

Neuroscientists have another technique in their toolkit: electrical recording of the brain's activity. To explain this point, though, we need to say a bit about how the brain functions. As mentioned earlier, the brain contains billions of nerve cells—called “neurons”—and it is the neurons that do the brain's main work. (We'll say more about these cells later in the chapter.) Neurons vary in their functioning, but for the most part they communicate with one another via chemical signals called “neurotransmitters.” Once a neuron is “activated,” it releases the transmitter, and this chemical can then activate (or, in some cases, *de-activate*) other, adjacent neurons. The adjacent neurons “receive” this chemical signal and, in turn, send their own signal onward to other neurons.

Let's be clear, though, that the process we just described is communication *between* neurons: One neuron releases the transmitter substance, and this activates (or *de-activates*) another neuron. But there's also communication *within* each neuron. The reason, basically, is that neurons have an “input” end and an “output” end. The “input” end is the portion of the neuron that's most sensitive to neurotransmitters; this is where the signal from other neurons is received. The “output” end is the portion that releases neurotransmitters, sending the



## MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging produces magnificently detailed pictures of the brain. Panel A shows an “axial view”—a “slice” of the brain viewed from the top of the head (the front of the head is at the top of the image). Clearly visible is the longitudinal fissure, which divides the left cerebral hemisphere from the right. Panel B, a “coronal view,” shows a slice of the brain viewed from the front. Again, the separation of the two hemispheres is clearly visible, as are some of the commissures linking the two brain halves. Panel C, a “sagittal view,” shows a slice of the brain viewed from the side. Here, many of the structures in the limbic system (see Figure 2.5) are easily seen.

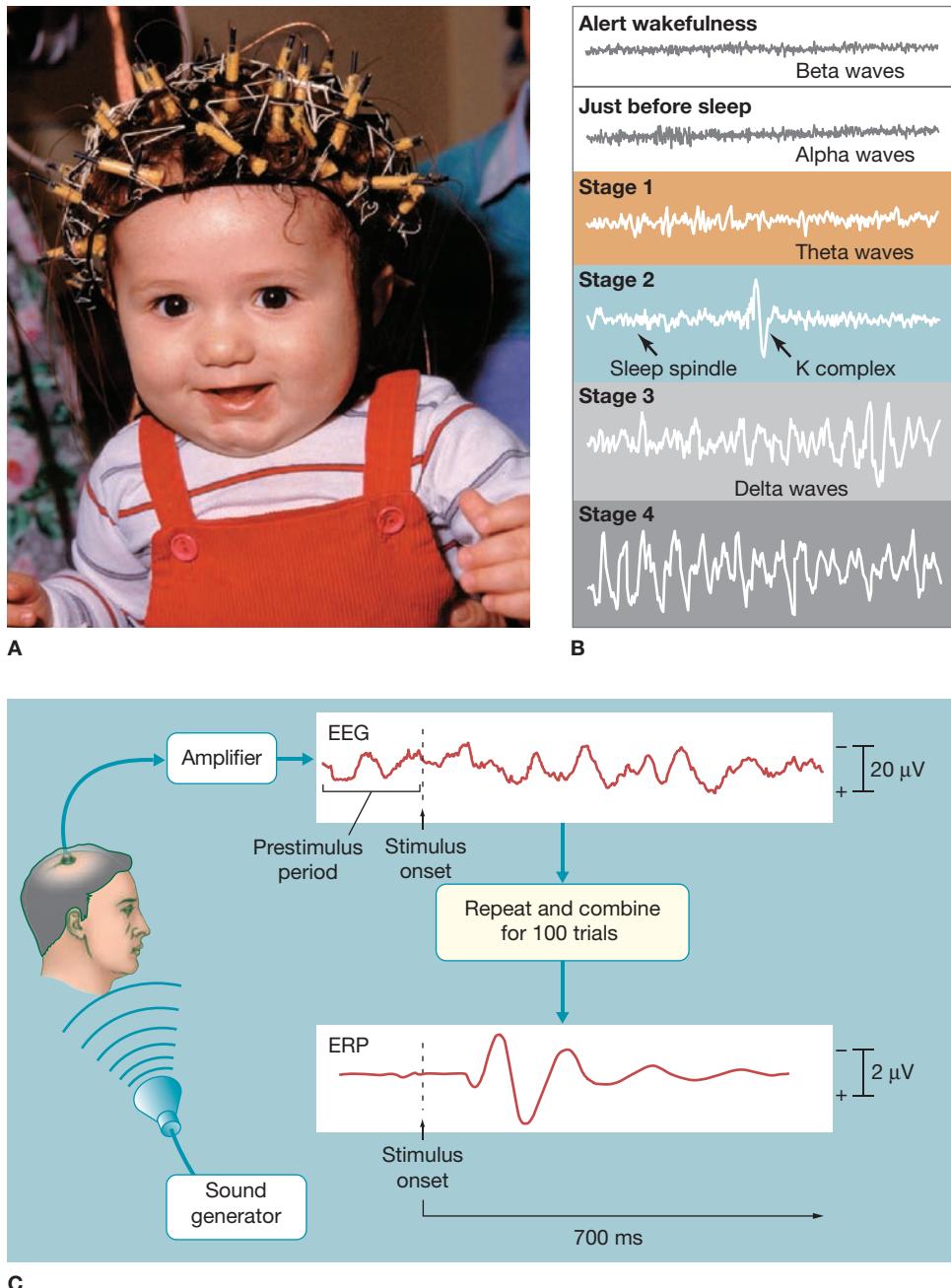
signal on to other neurons. These two ends can sometimes be far apart. (For example, some neurons in the body run from the base of the spine down to the toes; for these cells, the input and output ends might be a full meter apart.) The question, then, is how neurons get a signal from one end of the cell to the other.

The answer involves an electrical pulse, made possible by a flow of charged atoms (ions) in and out of the neuron (again, we’ll say more about this process later in the chapter). The amount of electrical current involved in this ion flow is tiny; but many millions of neurons are active at the same time, and the current generated by all of them together is strong enough to be detected by sensitive electrodes placed on the surface of the scalp. This is the basis for *electroencephalography*—a recording of voltage changes occurring at the scalp that reflect activity in the brain underneath. This procedure generates an *electroencephalogram (EEG)*—a recording of the brain’s electrical activity.

Often, EEGs are used to study broad rhythms in the brain’s activity. For example, an *alpha rhythm* (with the activity level rising and falling seven to ten times per second) can usually be detected in the brain of someone who is awake but calm and relaxed; a *delta rhythm* (with the activity rising and falling roughly one to four times per second) is observed when someone is deeply asleep. A much faster *gamma rhythm* (between 30 and 80 cycles per second) has received a lot of research attention, with a suggestion that this rhythm plays a key role in creating conscious awareness (e.g., Crick & Koch, 1990; Dehaene, 2014).

Sometimes, though, we want to know about the electrical activity in the brain over a shorter period—for example, when the brain is responding to a specific input or a particular stimulus. In this case, we measure changes in the EEG in the brief periods just before, during, and after the event. These changes are referred to as *event-related potentials* (see Figure 2.7).

**FIGURE 2.7 RECORDING THE BRAIN'S ELECTRICAL ACTIVITY**



To record the brain's electrical signals, researchers generally use a cap that has electrodes attached to it. The procedure is easy and entirely safe—it can even be used to measure brain signals in a baby (Panel A). In some procedures, researchers measure recurrent rhythms in the brain's activity, including rhythms that distinguish the stages of sleep (Panel B). In other procedures, they measure brain activity produced in response to a single event—such as the presentation of a well-defined stimulus (Panel C).

## The Power of Combining Techniques

Each of the research tools we've described has strengths and weaknesses. CT scans and MRI data tell us about the shape and size of brain structures, but they tell nothing about the activity levels within these structures. PET scans and fMRI studies do tell us about brain activity, and they can locate the activity rather precisely (within a millimeter or two). But these techniques are less precise about *when* the activity took place. For example, fMRI data summarize the brain's activity over a period of several seconds and cannot indicate when exactly, within this time window, the activity took place. EEG data give more precise information about timing but are much weaker in indicating *where* the activity took place.

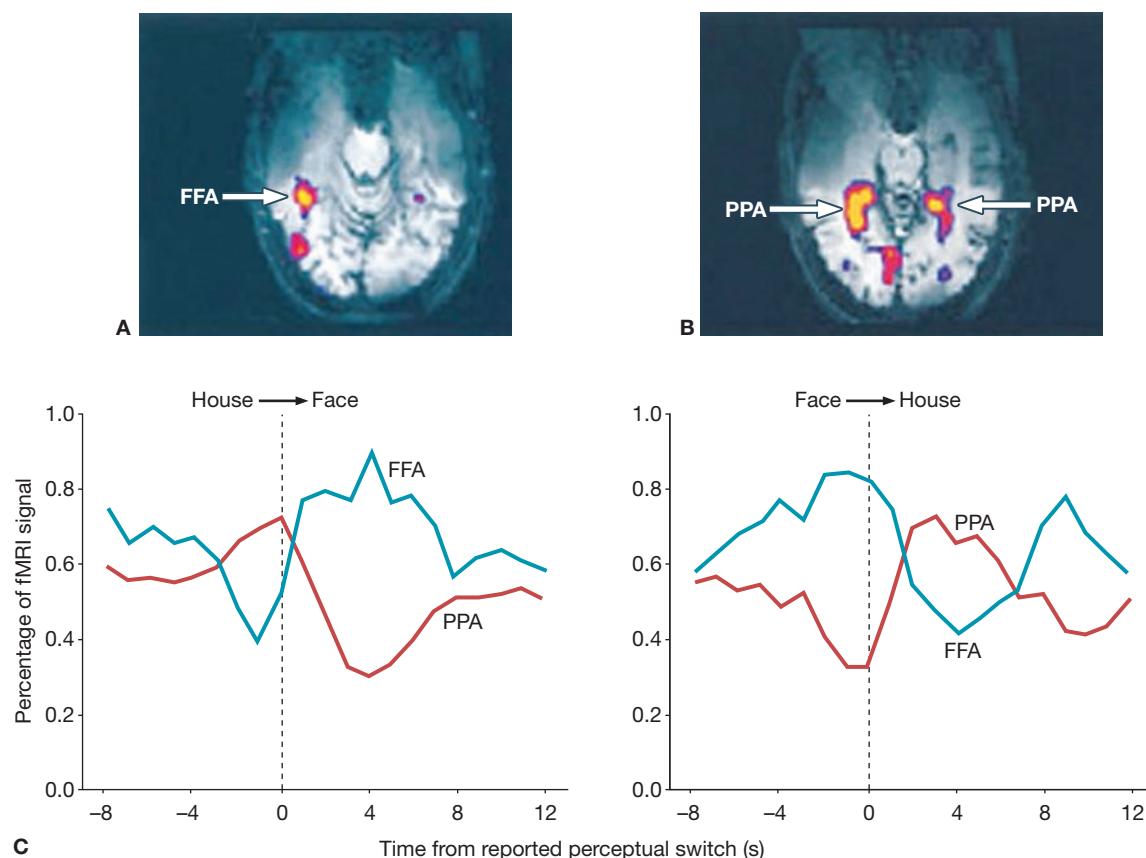
Researchers deal with these limitations by means of a strategy commonly used in science: We seek data from multiple sources, so that the strengths of one technique can make up for the shortcomings of another. As a result, some studies combine EEG recordings with fMRI scans, with the EEGs telling us when certain events took place in the brain, and the scans telling us where the activity took place. Likewise, some studies combine fMRI scans with CT data, so that findings about brain activation can be linked to a detailed portrait of the person's brain anatomy.

Researchers also face another complication: the fact that many of the techniques described so far provide *correlational data*. To understand the concern here, let's look at an example. A brain area called the **fusiform face area (FFA)** is especially active whenever a face is being perceived (see [Figure 2.8](#))—and so there is a correlation between a mental activity (perceiving a face) and a pattern of brain activity. Does this mean the FFA is needed for face perception? A different possibility is that the FFA activation may just be a by-product of face perception and doesn't play a crucial role. As an analogy, think about the fact that a car's speedometer becomes “more activated” (i.e., shows a higher value) whenever the car goes faster. That doesn't mean that the speedometer *causes* the speed or *is necessary* for the speed. The car would go just as fast and would, for many purposes, perform just as well if the speedometer were removed. The speedometer's state, in other words, is correlated with the car's speed but in no sense causes (or promotes, or is needed for) the car's speed.

In the same way, neuroimaging data can tell us that a brain area's activity is correlated with a particular function, but we need other data to determine whether the brain site plays a role in *causing* (or supporting, or allowing) that function. In many cases, those other data come from the study of brain lesions. If damage to a brain site disrupts a particular function, it's an indication that the site does play some role in supporting that function. (And, in fact, the FFA does play an important role in face recognition.)

Also helpful here is a technique called **transcranial magnetic stimulation (TMS)**. This technique creates a series of strong magnetic pulses at a specific location on the scalp, and these pulses activate the neurons directly underneath this scalp area (Helmuth, 2001). TMS can thus be used as a means of

**FIGURE 2.8 BRAIN ACTIVITY AND AWARENESS**



Panel A shows an fMRI scan of a subject looking at faces. Activation levels are high in the fusiform face area (FFA), an area that is apparently more responsive to faces than to other visual stimuli. Panel B shows a scan of the same subject looking at pictures of places; now, activity levels are high in the parahippocampal place area (PPA). Panel C compares the activity in these two areas when the subject has a picture of a face in front of one eye and a picture of a house in front of the other eye. When the viewer's perception shifts from the house to the face, activation increases in the FFA. When the viewer's perception shifts from the face to the house, PPA activation increases. In this way, the activation level reflects what the subject is aware of, and not just the pattern of incoming stimulation.

(AFTER TONG, NAKAYAMA, VAUGHAN, & KANWISHER, 1998)

asking what happens if we stimulate certain neurons. In addition, because this stimulation *disrupts* the ordinary function of these neurons, it produces a (temporary) lesion—allowing us to identify, in essence, what functions are compromised when a particular bit of brain tissue is briefly “turned off.” In these ways, the results of a TMS procedure can provide crucial information about the functional role of that brain area.

## Localization of Function

Drawing on the techniques we have described, neuroscientists have learned a great deal about the function of specific brain structures. This type of research effort is referred to as the **localization of function**, an effort (to put it crudely) aimed at figuring out what's happening where within the brain.

Localization data are useful in many ways. For example, think back to the discussion of Capgras syndrome earlier in this chapter. Brain scans told us that people with this syndrome have damaged amygdalae, but how is this damage related to the symptoms of the syndrome? More broadly, what problems does a damaged amygdala create? To tackle these questions, we rely on localization of function—in particular, on data showing that the amygdala is involved in many tasks involving emotional appraisal. This combination of points helped us to build (and test) our claims about this syndrome and, in general, claims about the role of emotion within the ordinary experience of “familiarity.”

### TEST YOURSELF

5. What is the difference between *structural imaging* of the brain and *functional imaging*? What techniques are used for each?
6. What do we gain from *combining* different methods in studying the brain?
7. What is meant by the phrase “localization of function”?

As a different illustration, consider the experience of calling up a “mental picture” before the “mind’s eye.” We’ll have more to say about this experience in Chapter 11, but we can already ask: How much does this experience have in common with ordinary seeing—that is, the processes that unfold when we place a real picture before someone’s eyes? As it turns out, localization data reveal enormous overlap between the brain structures needed for these two activities (visualizing and actual vision), telling us immediately that these activities do have a great deal in common (see Figure 2.9). So, again, we build on localization—this time to identify how exactly two mental activities are related to each other.

## The Cerebral Cortex

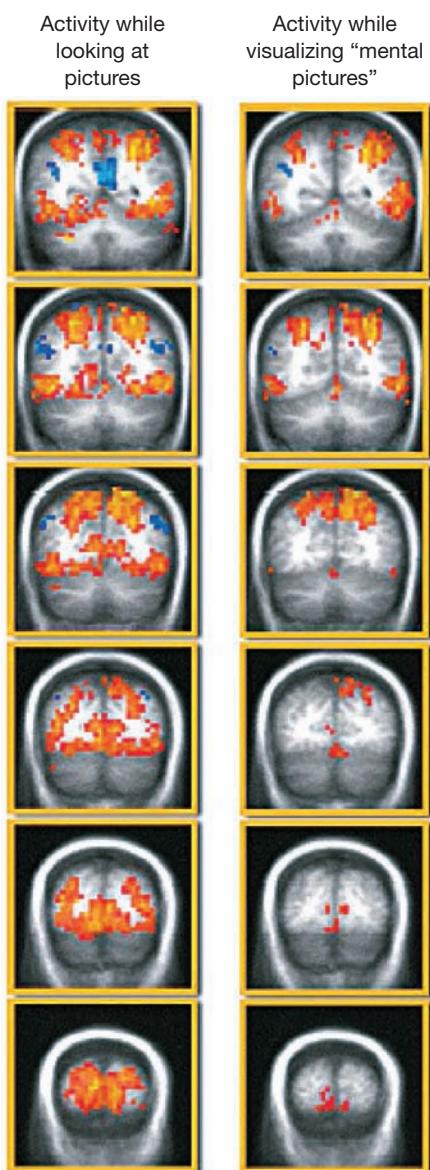
As we’ve noted, the largest portion of the human brain is the cerebral cortex—the thin layer of tissue covering the cerebrum. This is the region in which an enormous amount of information processing takes place, and so, for many topics, it is the brain region of greatest interest for cognitive psychologists.

The cortex includes many distinct regions, each with its own function, but these regions are traditionally divided into three categories. *Motor areas* contain brain tissue crucial for organizing and controlling bodily movements. *Sensory areas* contain tissue essential for organizing and analyzing the information received from the senses. *Association areas* support many functions, including the essential (but not well-defined) human activity we call “thinking.”

### Motor Areas

Certain regions of the cerebral cortex serve as the “departure points” for signals leaving the cortex and controlling muscle movement. Other areas are the “arrival points” for information coming from the eyes, ears, and

## FIGURE 2.9 A PORTRAIT OF THE BRAIN AT WORK



These fMRI images show different “slices” through the living brain, revealing levels of activity in different brain sites. Regions that are more active are shown in yellow, orange, and red; lower activity levels are indicated in blue. The first column shows brain activity while a person is making judgments about simple pictures. The second column shows brain activity while the person is making the same sorts of judgments about “mental pictures,” visualized before the “mind’s eye.”

other sense organs. In both cases, these areas are called “primary projection areas,” with the departure points known as the **primary motor projection areas** and the arrival points contained in regions known as the **primary sensory projection areas**.

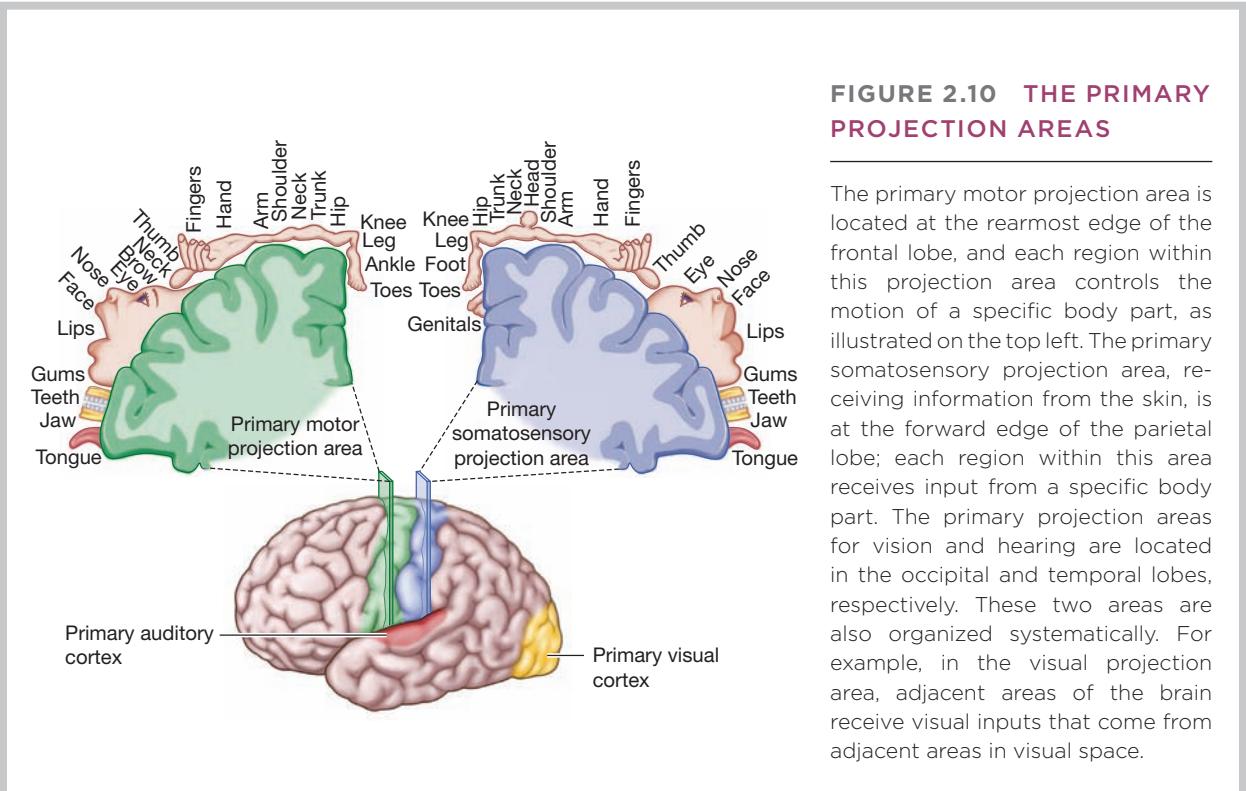
Evidence for the motor projection area comes from studies in which investigators apply mild electrical current to this area in anesthetized animals. This stimulation often produces specific movements, so that current applied to one site causes a movement of the left front leg, while current applied to a different site causes the ears to prick up. These movements show a pattern of **contralateral control**, with stimulation to the left hemisphere leading to movements on the right side of the body, and vice versa.

Why are these areas called “projection areas”? The term is borrowed from mathematics and from the discipline of map making, because these areas seem to form “maps” of the external world, with particular positions on the cortex corresponding to particular parts of the body or particular locations in space. In the human brain, the map that constitutes the motor projection area is located on a strip of tissue toward the rear of the frontal lobe, and the pattern of mapping is illustrated in **Figure 2.10**. In this illustration, a drawing of a person has been overlaid on a depiction of the brain, with each part of the little person positioned on top of the brain area that controls its movement. The figure shows that areas of the body that we can move with great precision (e.g., fingers and lips) have a lot of cortical area devoted to them; areas of the body over which we have less control (e.g., the shoulder and the back) receive less cortical coverage.

## Sensory Areas

Information arriving from the skin senses (your sense of touch or your sense of temperature) is projected to a region in the parietal lobe, just behind the motor projection area. This is labeled the “somatosensory” area in Figure 2.10. If a patient’s brain is stimulated in this region (with electrical current or touch), the patient will typically report a tingling sensation in a specific part of the body. Figure 2.10 also shows the region (in the temporal lobes) that functions as the primary projection area for hearing (the “auditory” area). If the brain is directly stimulated here, the patient will hear clicks, buzzes, and hums. An area in the occipital lobes is the primary projection area for vision; stimulation here causes the patient to see flashes of light or visual patterns.

The sensory projection areas differ from each other in important ways, but they also have features in common—and they’re features that parallel the attributes of the motor projection area. First, each of these areas provides a “map” of the sensory environment. In the somatosensory area, each part of the body’s surface is represented by its own region on the cortex; areas of the body that are near to each other are typically represented by similarly nearby areas in the brain. In the visual area, each region of visual space has its own cortical representation, and adjacent areas of visual space are usually represented by adjacent brain sites. In the auditory projection area, different



**FIGURE 2.10 THE PRIMARY PROJECTION AREAS**

The primary motor projection area is located at the rearmost edge of the frontal lobe, and each region within this projection area controls the motion of a specific body part, as illustrated on the top left. The primary somatosensory projection area, receiving information from the skin, is at the forward edge of the parietal lobe; each region within this area receives input from a specific body part. The primary projection areas for vision and hearing are located in the occipital and temporal lobes, respectively. These two areas are also organized systematically. For example, in the visual projection area, adjacent areas of the brain receive visual inputs that come from adjacent areas in visual space.

frequencies of sound have their own cortical sites, and adjacent brain sites are responsive to adjacent frequencies.

Second, in each of these sensory maps, the assignment of cortical space is governed by function, not by anatomical proportions. In the parietal lobes, parts of the body that aren't very discriminating with regard to touch—even if they're physically large—get relatively little cortical area. Other, more sensitive areas of the body (the lips, tongue, and fingers) get much more space. In the occipital lobes, more cortical surface is devoted to the fovea, the part of the eyeball that is most sensitive to detail. (For more on the fovea, see Chapter 3.) And in the auditory areas, some frequencies of sound get more cerebral coverage than others. It's surely no coincidence that these "advantaged" frequencies are those essential for the perception of speech.

Finally, we also find evidence here of contralateral connections. The somatosensory area in the left hemisphere, for example, receives its main input from the right side of the body; the corresponding area in the right hemisphere receives its input from the left side of the body. Likewise for the visual projection areas, although here the projection is not contralateral with regard to body parts. Instead, it's contralateral with regard to physical space. Specifically, the visual projection area in the right hemisphere receives information from both the left eye and the right, but the information it receives corresponds to the left half of visual space (i.e., all of the things



### THE SENSORY HOMUNCULUS

An artist's rendition of what a man would look like if his appearance were proportional to the area allotted by the somatosensory cortex to his various body parts.

### TEST YOURSELF

8. What is a projection area in the brain? What's the role of the motor projection area? The sensory projection area?
9. What does it mean to say that the brain relies on "contralateral" connections?

visible to your left when you're looking straight ahead). The reverse is true for the visual area in the left hemisphere. It receives information from both eyes, but from only the right half of visual space. The pattern of contralateral organization is also evident—although not as clear-cut—for the auditory cortex, with roughly 60% of the nerve fibers from each ear sending their information to the opposite side of the brain.

## Association Areas

The areas described so far, both motor and sensory, make up only a small part of the human cerebral cortex—roughly 25%. The remaining cortical areas are traditionally referred to as the **association cortex**. This terminology is falling out of use, however, partly because this large volume of brain tissue can be subdivided further on both functional and anatomical grounds. These subdivisions are perhaps best revealed by the diversity of symptoms that result if the cortex is damaged in one or another specific location. For example, some lesions in the frontal lobe produce **apraxias**, disturbances in the initiation or organization of voluntary action. Other lesions (generally in the occipital cortex, or in the rearmost part of the parietal lobe) lead to **agnosias**, disruptions in the ability to identify familiar objects. Agnosias usually affect one modality only—so a patient with visual agnosia, for example, can recognize a fork by touching it but not by looking at it. A patient with auditory agnosia, by contrast, might be unable to identify familiar voices but might still recognize the face of the person speaking.

Still other lesions (usually in the parietal lobe) produce **neglect syndrome**, in which the individual seems to ignore half of the visual world. A patient afflicted with this syndrome will shave only half of his face and eat food from only half of his plate. If asked to read the word “parties,” he will read “ties,” and so on.

Damage in other areas causes still other symptoms. We mentioned earlier that lesions in areas near the lateral fissure (the deep groove that separates the frontal and temporal lobes) can result in disruption to language capacities, a problem referred to as **aphasia**.

Finally, damage to the frontmost part of the frontal lobe, the prefrontal area, causes problems in planning and implementing strategies. In some cases, patients with damage here show problems in inhibiting their own behaviors, relying on habit even in situations for which habit is inappropriate. Frontal lobe damage can also (as we mentioned in our discussion of Capgras syndrome) lead to a variety of confusions, such as whether a remembered episode actually happened or was simply imagined.

We'll discuss more about these diagnostic categories—aphasia, agnosia, neglect, and more—in upcoming chapters, where we'll consider these disorders in the context of other things that are known about object recognition, attention, and so on. Our point for the moment, though, is simple: These clinical patterns make it clear that the so-called association cortex contains many subregions, each specialized for a particular function, but with all of the subregions working together in virtually all aspects of our daily lives.

# Brain Cells

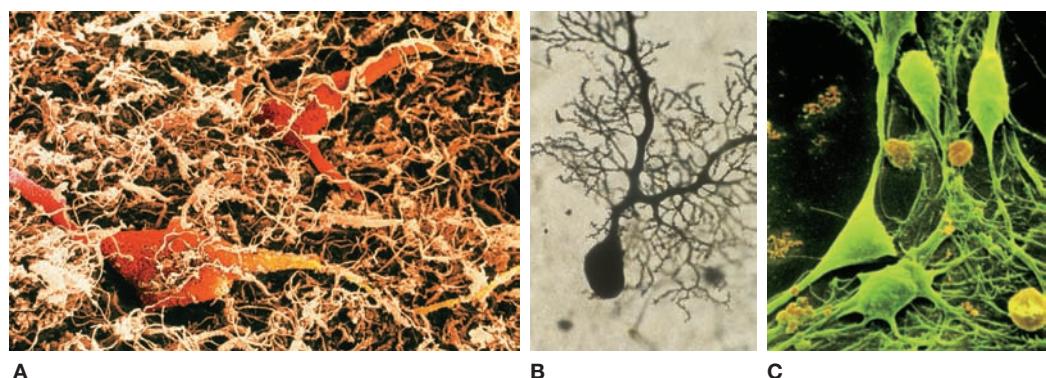
Our brief tour so far has described some of the large-scale structures in the brain. For many purposes, though, we need to zoom in for a closer look, in order to see how the brain's functions are actually carried out.

## Neurons and Glia

We've already mentioned that the human brain contains many billions of **neurons** and a comparable number of **glia**. The glia perform many functions. They help to guide the development of the nervous system in the fetus and young infant; they support repairs if the nervous system is damaged; they also control the flow of nutrients to the neurons. Specialized glial cells also provide a layer of electrical insulation surrounding parts of some neurons; this insulation dramatically increases the speed with which neurons can send their signals. (We'll return to this point in a moment.) Finally, some research suggests the glia may also constitute their own signaling system within the brain, separate from the information flow provided by the neurons (e.g., Bullock et al, 2005; Gallo & Chitajullu, 2001).

There is no question, though, that the main flow of information through the brain—from the sense organs inward, from one part of the brain to the others, and then from the brain outward—is made possible by the neurons. Neurons come in many shapes and sizes (see **Figure 2.11**), but in general, neurons have three major parts. The **cell body** is the portion of the cell that contains the neuron's nucleus and all the elements needed for the normal metabolic activities of the cell. The **dendrites** are usually the

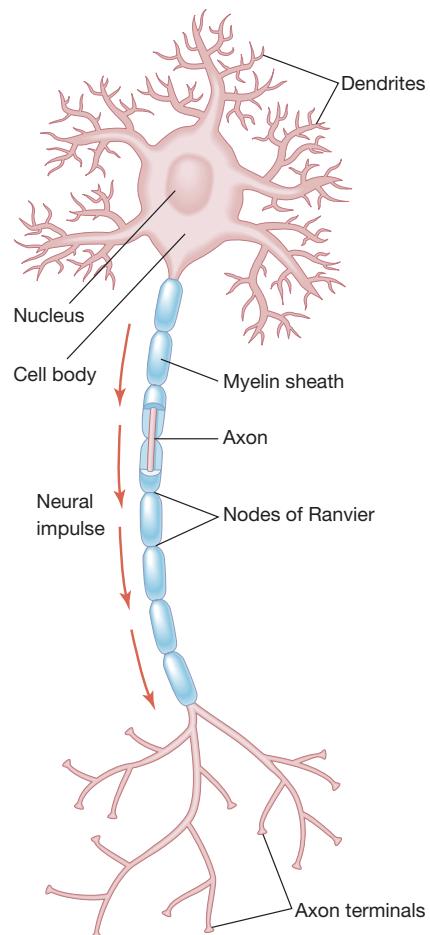
**FIGURE 2.11 NEURONS**



Panel A shows neurons from the spinal cord (stained in red); Panel B shows neurons from the cerebellum; Panel C shows neurons from the cerebral cortex.

“input” side of the neuron, receiving signals from many other neurons. In most neurons, the dendrites are heavily branched, like a thick and tangled bush. The **axon** is the “output” side of the neuron; it sends neural impulses to other neurons (see **Figure 2.12**). Axons can vary enormously in length—the giraffe, for example, has neurons with axons that run the full length of its neck.

**FIGURE 2.12 REGIONS OF THE NEURON**



Most neurons have three identifiable regions. The *dendrites* are the part of the neuron that usually detects incoming signals. The *cell body* contains the metabolic machinery that sustains the cell. The *axon* is the part of the neuron that transmits a signal to another location. When the cell fires, neurotransmitters are released from the terminal endings at the tip of the axon. The myelin sheath is created by glial cells that wrap around the axons of many neurons. The gaps in between the myelin cells are called the *nodes of Ranvier*.



Of the many drugs that influence the brain, one is readily available and often consumed: alcohol. Alcohol influences the entire brain, and even at low levels of intoxication we can detect alcohol's effects—for example, with measures of motor skills or response time.

Alcohol's effects are more visible, though, in some functions than in others, and so someone who's quite intoxicated can perform many activities at a fairly normal level. However, alcohol has a strong impact on activities that depend on the brain's prefrontal cortex. This is the brain region that's essential for the mind's *executive function*—the system that allows you to control your thoughts and behaviors. (We'll say more about executive function in upcoming chapters.) As a result, alcohol undercuts your ability to resist temptation or to overcome habit. Impairments in executive function also erode your ability to make thoughtful decisions and draw sensible conclusions.

In addition, alcohol can produce impairments in memory, including "alcoholic blackouts." So-called fragmentary blackouts, in which the person remembers some bits of an experience but not others, are actually quite common. In one study, college students were asked: "Have you ever awoken after a night of drinking not able to remember things that you did or places where you went?" More than half of the students indicated that, yes, this had happened to them at some point; 40% reported they'd had a blackout within the previous year.

How drunk do you have to be in order to experience a blackout? Many authorities point to a blood alcohol level of 0.25 (roughly nine or ten drinks for someone of average weight), but other

factors also matter. For example, blackouts are more common if you become drunk rapidly—as when you drink on an empty stomach, or when you gulp alcohol rather than sipping it.

Let's combine these points about blackouts, though, with our earlier observation about alcohol's uneven effects. It's possible for someone to be quite drunk, and therefore suffer an alcoholic blackout, even if the person seemed alert and coherent during the drunken episode. To see some of the serious problems this can cause, consider a pattern that often emerges in cases involving allegations of sexual assault. Victims of assault sometimes report that they have little or no memory of the sexual encounter; they therefore assume they were barely conscious during the event and surely incapable of giving consent. But is this assumption correct?

The answer is complex. If someone was drunk enough to end up with a blackout, then that person was probably impaired to a degree that would interfere with decision making—and so the person could not have given legitimate, meaningful consent. But, even so, the person might have been functioning in a way that seemed mostly normal (able to converse, to move around) and may even have expressed consent in words or actions.

In this situation, then, the complainant is correct in saying that he or she couldn't have given (and therefore didn't give) meaningful consent, but the accused person can legitimately say that he or she perceived that there was consent. We can debate how best to judge these situations, but surely the best path forward is to avoid this sort of circumstance—by drinking only in safe settings or by keeping a strict limit on your drinking.

## The Synapse

We've mentioned that communication from one neuron to the next is generally made possible by a chemical signal: When a neuron has been sufficiently stimulated, it releases a minute quantity of a **neurotransmitter**. The molecules of this substance drift across the tiny gap between neurons and latch on to the dendrites of the adjacent cell. If the dendrites receive enough of this substance, the next neuron will "fire," and so the signal will be sent along to other neurons.

Notice, then, that neurons usually don't touch each other directly. Instead, at the end of the axon there is a gap separating each neuron from the next. This entire site—the end of the axon, plus the gap, plus the receiving membrane of the next neuron—is called a **synapse**. The space between the neurons is the *synaptic gap*. The bit of the neuron that releases the transmitter into this gap is the **presynaptic membrane**, and the bit of the neuron on the other side of the gap, affected by the transmitters, is the **postsynaptic membrane**.

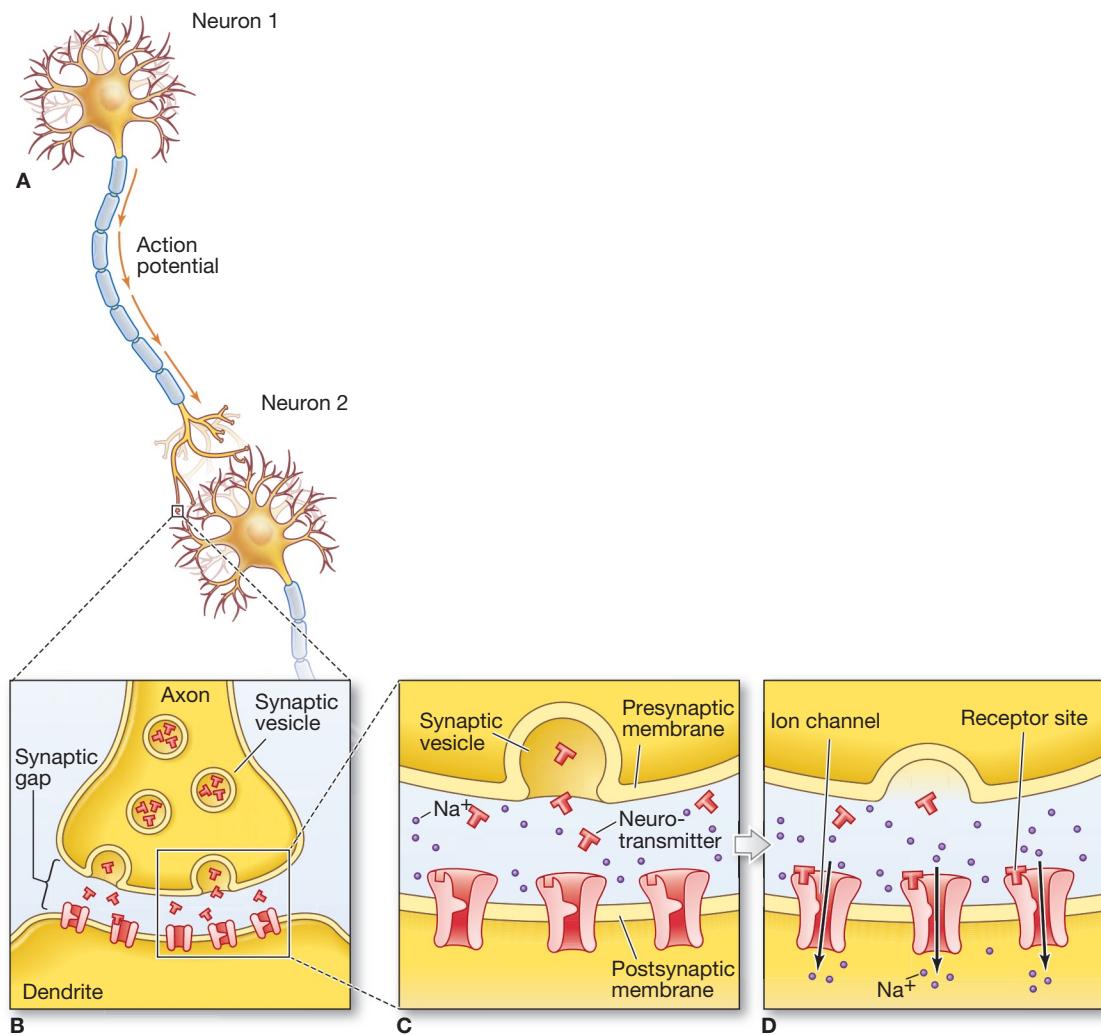
When the neurotransmitters arrive at the postsynaptic membrane, they cause changes in this membrane that enable certain ions to flow into and out of the postsynaptic cell (see Figure 2.13). If these ionic flows are relatively small, then the postsynaptic cell quickly recovers and the ions are transported back to where they were initially. But if the ionic flows are large enough, they trigger a response in the postsynaptic cell. In formal terms, if the incoming signal reaches the postsynaptic cell's **threshold**, then the cell fires. That is, it produces an **action potential**—a signal that moves down its axon, which in turn causes the release of neurotransmitters at the next synapse, potentially causing the next cell to fire.

In some neurons, the action potential moves down the axon at a relatively slow speed. For other neurons, specialized glial cells are wrapped around the axon, creating a layer of insulation called the **myelin sheath** (see Figure 2.12). Because of the myelin, ions can flow in or out of the axon only at the gaps between the myelin cells. As a result, the signal traveling down the axon has to "jump" from gap to gap, and this greatly increases the speed at which the signal is transmitted. For neurons without myelin, the signal travels at speeds below 10 m/s; for "myelinated" neurons, the speed can be ten times faster.

Overall, let's emphasize four points about this sequence of events. First, let's note once again that neurons depend on two different forms of information flow. Communication from one neuron to the next is (for most neurons) mediated by a chemical signal. In contrast, communication from one end of the neuron to the other (usually from the dendrites down the length of the axon) is made possible by an electrical signal, created by the flow of ions in and out of the cell.

Second, the postsynaptic neuron's initial response can vary in size; the incoming signal can cause a small ionic flow or a large one. Crucially, though, once these inputs reach the postsynaptic neuron's firing threshold, there's no variability in the response—either a signal is sent down the axon or it is not. If the signal is sent, it is always of the same magnitude, a fact referred to as

**FIGURE 2.13 SCHEMATIC VIEW OF SYNAPTIC TRANSMISSION**



(Panel A) Neuron 1 transmits a message across the synaptic gap to Neuron 2. The neurotransmitters are initially stored in structures called “synaptic vesicles” (Panel B). When a signal travels down the axon, the vesicles are stimulated and some of them burst (Panel C), ejecting neurotransmitter molecules into the synaptic gap and toward the postsynaptic membrane (Panel D). Neurotransmitter molecules settle on receptor sites, ion channels open, and sodium ( $\text{Na}^+$ ) floods in.

the **all-or-none law**. Just as pounding on a car horn won't make the horn any louder, a stronger stimulus won't produce a stronger action potential. A neuron either fires or it doesn't; there's no in-between.

This does not mean, however, that neurons always send exactly the same information. A neuron can fire many times per second or only occasionally. A neuron can fire just once and then stop, or it can keep firing for an extended span. But, even so, each individual response by the neuron is always the same size.

Third, we should also note that the brain relies on many different neurotransmitters. By some counts, a hundred transmitters have been catalogued so far, and this diversity enables the brain to send a variety of different messages. Some transmitters have the effect of stimulating subsequent neurons; some do the opposite and *inhibit* other neurons. Some transmitters play an essential role in learning and memory; others play a key role in regulating the level of arousal in the brain; still others influence motivation and emotion.

Fourth, let's be clear about the central role of the synapse. The synaptic gap is actually quite small—roughly 20 to 30 nanometers across. (For contrast's sake, the diameter of a human hair is roughly 80,000 nanometers.) Even so, transmission across this gap slows down the neuronal signal, but this is a tiny price to pay for the advantages created by this mode of signaling: Each neuron receives information from (i.e., has synapses with) many other neurons, and this allows the “receiving” neuron to integrate information from many sources. This pattern of many neurons feeding into one also makes it possible for a neuron to “compare” signals and to adjust its response to one input according to the signal arriving from a different input. In addition, communication at the synapse is *adjustable*. This means that the strength of a synaptic connection can be altered by experience, and this adjustment is crucial for the process of *learning*—the storage of new knowledge and new skills within the nervous system.

### TEST YOURSELF

10. What are glia? What are dendrites? What is an axon? What is a synapse?
11. What does it mean to say that neurons rely on two different forms of information flow, one chemical and one electrical?

## Coding

This discussion of individual neurons leads to a further question: How do these microscopic nerve cells manage to represent a specific idea or a specific content? Let's say that right now you're thinking about your favorite song. How is this information represented by neurons? The issue here is referred to as **coding**, and there are many options for what the neurons' “code” might be (Gallistel, 2017). As one option, we might imagine that a specific group of neurons somehow represents “favorite song,” so that whenever you're thinking about the song, it's precisely these neurons that are activated. Or, as a different option, the song might be represented by a broad *pattern* of neuronal activity. If so, “favorite song” might be represented in the brain by something like “Neuron X firing strongly while Neuron Y is firing weakly and Neuron Z is not firing at all” (and so on for thousands of other neurons). Note that within this scheme the same neurons might be involved in the representation of other sounds, but with different patterns. So—to continue our example—Neuron X might also be involved in the representation of the sound of a car