

The Parietal Lobes

H. P. was a 28-year-old accountant who was planning his wedding with his fiancée when she noticed that he was making addition errors as he calculated the budget for their reception. At first, they joked about it, especially given his occupation, but in the following weeks H. P.'s problem with numbers became serious. In fact, he was no longer able to do a simple subtraction such as $30 - 19$ in which the solution requires "borrowing" 10 when subtracting 9 from 0.

At first, H. P. simply put it down to working too hard, but soon he began to have trouble reaching for objects. He was constantly knocking over his water glass, because his reach was clumsy and misdirected. He began confusing left and right and having difficulties reading. Some of the words appeared to be backward or upside down, and he could not make sense of them.

Finally, when H. P. visited a neurologist for testing, it was obvious that something was seriously wrong. Indeed something was: he had a fast-growing tumor in his left parietal lobe. Unfortunately, the tumor was extremely virulent and, within a couple of months, he died.

The parietal cortex processes and integrates somatosensory and visual information, especially with regard to the control of movement. In this chapter, we first describe the anatomy of the parietal lobes and then present a theoretical model of parietal-lobe organization. Next, we consider the major somatosensory symptoms of parietal injury, survey the most commonly observed disorders of the posterior parietal region, and conclude the chapter with a survey of behavioral tests that reliably predict brain injury.

Anatomy of the Parietal Lobes

H. P.'s symptoms are typical of left parietal injury and illustrative of the curious pattern of symptoms that have proved a challenge for neuropsychologists to understand. Part of the challenge is that these symptoms are difficult to

demonstrate in animals. Common laboratory animals such as rats and cats have very modest parietal “lobes,” and, although monkeys with parietal damage show many symptoms similar to those seen in human patients, symptoms related to language or cognition are difficult to study in monkeys. Furthermore, the parietal lobes in the human brain have evolved to a much larger size, which might imply that humans will show some symptoms not seen in monkeys.

Subdivisions of the Parietal Cortex

The parietal lobe is the region of cerebral cortex between the frontal and occipital lobes, underlying the parietal bone at the roof of the skull. This area is roughly demarcated anteriorly by the central fissure, ventrally by the Sylvian fissure, dorsally by the cingulate gyrus, and posteriorly by the parieto-occipital sulcus (Figure 14.1A). The principal regions of the parietal lobe include the postcentral gyrus (Brodmann’s areas 1, 2, and 3), the superior parietal lobule (areas 5 and 7), the parietal operculum (area 43), the supramarginal gyrus (area 40), and the angular gyrus (area 39) (Figure 14.1A and B).

Together, the supramarginal gyrus and angular gyrus are often referred to as the inferior parietal lobe. The parietal lobe can be divided into two functional zones: an anterior zone including areas 1, 2, 3, and

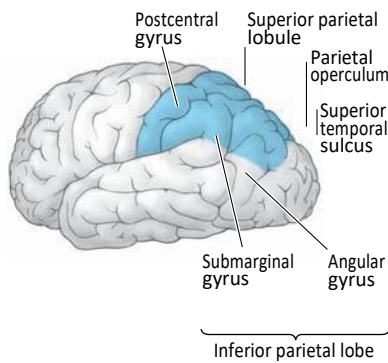
43; and a posterior zone, which includes the remaining areas. The anterior zone is the somatosensory cortex; the posterior zone is referred to as the **posterior parietal cortex**.

The parietal lobes have undergone a major expansion in the course of human evolution, largely in the inferior parietal region. This increase in size has made comparisons of various areas in the human brain with those in the monkey brain confusing, especially because Brodmann did not identify areas 39 and 40 in the monkey. Whether monkeys actually have regions homologous to areas 39 and 40 is debatable. One solution to this

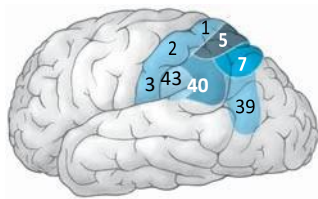
problem is to consult another anatomist, Constantin von Economo.

On von Economo’s maps, in which parietal areas are called PA (parietal) the parietal lobe.

(A) Major parietal lobe gyri and sulci



(B) Brodmann’s cytoarchitectonic regions



(C) von Economo’s cytoarchitectonic regions

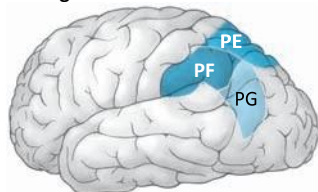


Figure 14.1 Gross anatomy of

area A), PB, and so forth, are three posterior parietal areas (PE, PF, PG) that von Economo described in both humans and monkeys (Figure 14.1C). If we use this system, area PF is equivalent to area 7b and PE to area 5 in Felleman and van Essen's flat map of cortical areas in the macaque (see Figure 10.17).

Similarly, area PG in the monkey includes areas 7a, VIP, LIP, IPG, PP, MSTc, and MSTp. These PG areas are primarily visual (see Chapter 15).

An area of significant expansion in the human brain appears to consist of the polymodal parts of area PG and the adjoining polymodal cortex in the superior temporal sulcus. (Polymodal cells are those that receive inputs from more than one sensory modality.) Those in PG respond to both somatosensory and visual inputs, whereas those in the superior temporal sulcus (the third visual pathway discussed in Chapter 13) respond to various combinations of auditory, visual, and

somatosensory inputs.

The increase in size of area PG and the superior temporal sulcus is especially interesting because this region is anatomically asymmetrical in the human brain (see Figure 11.1). This asymmetry may be due to a much larger area PG (and possibly superior temporal sulcus) on the right than on the left. If PG has a visual function and is larger in humans, especially in the right hemisphere, then we might expect unique visual symptoms after right parietal le-

sions, which is indeed the case. Note, however, that PG is also larger on the left in the human than in the monkey, which would lead us to expect humans to have unique deficits after left hemisphere lesions. This, too, is the case.

Connections of the Parietal Cortex

The anterior parietal cortex has rather straightforward connections, which are illustrated in Felleman and van Essen's hierarchy (see Figure 10.19). There are projections from the primary somatosensory cortex to area PE, which has a tactile recognition function, as well as to motor areas, including the primary motor cortex (area 4) and the supplementary motor and premotor regions. The motor connections must be important for providing sensory information about limb position in the control of movement (see Chapter 9).

Although more than 100 inputs and outputs of areas 5 and 7 in the monkey (PE, PF, and PG) have been described (see Figure 10.19), a few basic principles will summarize the connections diagrammed in Figure 14.2:

1. Area PE (Brodmann's area 5) is basically a somatosensory area, receiving most of its connections from the primary somatosensory cortex (areas 1, 2, and 3). Its cortical outputs are to the primary motor cortex (area 4) and to the supplementary motor (SMA) and premotor (6 and 8) regions, as well as to PF. Area PE therefore plays some role in guiding movement by providing information about

limb position.

2. Area PF (area 7b) has a heavy somatosensory input from the primary cortex (areas 1, 2, and 3) through area PE. It also receives inputs from the motor and premotor cortex and a small visual input

through area PG. Its efferent connections are similar to those of area PE, and these connections presumably provide some elaboration of similar information for the motor systems.

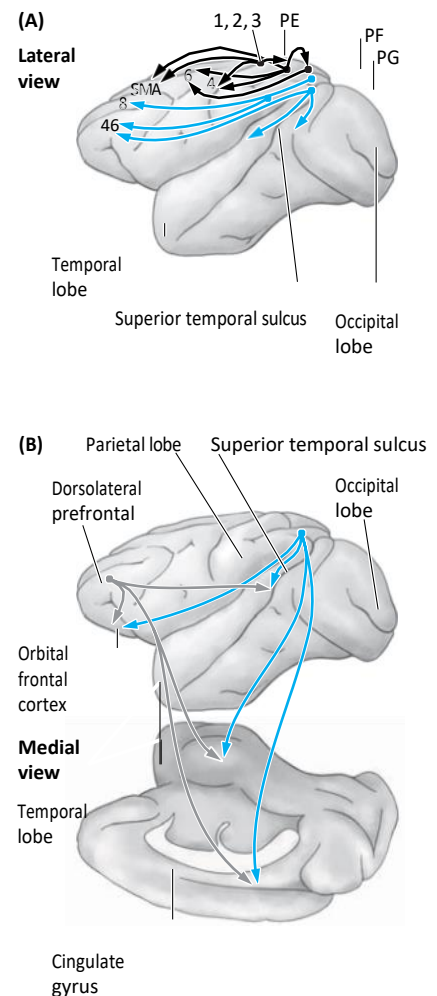
3. Area PG (area 7b and visual areas) receives more-complex connections including visual, somesthetic, proprioceptive (internal

stimuli), auditory, vestibular (balance), oculomotor (eye movement), and cingulate (motivational?). This region was described by MacDonald Critchley as the "parieto-temporo-occipital crossroads," which is apparent from the connectivity. It seems likely that its function corresponds to this intermodal mixing. Area PG is part of the dorsal stream discussed in Chapter 13. It is assumed to have a role in controlling spatially guided behavior with respect to visual and tactile information.

4. There is a close relation between the posterior parietal connections and the prefrontal cortex (especially area 46). Thus, there are connections between the posterior parietal cortex (PG and PF) and the

dorsolateral prefrontal region. Additionally, both the prefrontal and

the posterior parietal regions project to the same areas of the paralimbic cortex and the temporal cortex as well as to the hippocampus and various subcortical regions. These connections emphasize a close functional



relation between the prefrontal cortex and the parietal cortex. This relation probably has an

important role in the control of spatially guided behavior.

Figure 14.2 Connections of the parietal lobe. (A) The major cortical-cortical projections of the parietal lobe. (B) The posterior parietal and dorsolateral prefrontal projections to cingulate, orbital frontal, and temporal regions.

A Theory of Parietal-Lobe Function

If we consider the anterior (somatosensory) and posterior parietal zones as functionally distinct regions, we can identify two independent contributions of the parietal lobes. The anterior zone processes somatic sensations and perceptions; the posterior zone is specialized primarily for integrating sensory input from the somatic and visual regions and, to a lesser extent, from other sensory regions, mostly for the control of movement. We are concerned here mostly with the function of the posterior parietal zone; the anterior zone's somatosensory functions were discussed in Chapter 8.

Imagine that you are having dinner with a friend in a restaurant. You are confronted with a set of cutlery, some dishes, a basket of bread, a glass of water, perhaps a glass of wine or a cup of coffee, a napkin, and of course your companion. Seemingly without effort you select various utensils and foods as you chat with your friend.

If we analyze what is required to do all these things, however, we see that your brain is faced with several complex tasks. For example, you must reach and correctly grasp a glass or cup or fork or piece of bread. Each of those movements is directed toward a different place and requires a different hand posture and or limb movement or both. Your eyes and head must be directed toward various places in space, and you must coordinate the movements of your limbs and your head to get food to your mouth.

Furthermore, you must attend to certain objects and ignore others. (You do not take your companion's fork or drink.) You also must attend to the conversation with your friend and ignore other conversations around you. When you eat items from your plate, you must choose which one you want and select the correct utensil. It would be inappropriate to try to eat your peas with a knife. You must also make movements in the correct order. For example, you must cut your food before picking it up. Similarly, when you choose a bit of bread you must pick up a knife, get some butter, place the butter on the bread, and then eat the bread.

As we think about how the brain can manage these tasks, it seems obvious that there must be some sort of internal representation of the location of different objects around us, a sort of map in the brain of where things are. Furthermore, we assume that the map must be common to all our senses because we can move without apparent effort from visual to auditory to tactile information. On the basis of clinical observations of patients with parietal injury, it has been widely believed for nearly 60 years that the parietal lobe plays a central role in the creation of this brain map. But what is the map?

The commonly held introspective view is that real space must be mapped topographically because that is how it appears to us. That is, we take it for granted that the world around us is as we perceive it, and thus that the brain must employ some sort of unified spatial map. (This view is a form of the binding problem discussed in Chapter 10.) Unfortunately, there is very little evidence for the existence of such a map in the brain. Rather, it seems likely that there is no single map, but a series of representations of space, which vary in two ways. First, different representations are used for different behavioral needs. Second, representations of space vary from simple ones, which are ap-

plicable to the control of simple movements, to abstract ones, which may represent information such as topographical knowledge. We consider each of these aspects of brain maps in turn.

Uses of Spatial Information

Goodale and Milner emphasize that spatial information about the location of objects in the world is needed both to direct actions at those objects and to assign meaning and significance to them. In this sense, spatial information is simply another property of visual information, much like form, motion, and color. However, just as form is coded in more than one way in visual processing, so is spatial information. The critical factor for both form and space is how the information is to be used.

Recall that form recognition is of two basic types: one is for object recognition and the other is for the guidance of movement. Spatial information can be thought of in the same way.

Object Recognition

The spatial information needed to determine the relations between objects, independent of what the subject's behavior might be, is very different from the spatial information needed to guide eye, head, or limb movements to objects. In the latter case, the visuomotor control must be viewer centered — that is, the location of an object and its local orientation and motion must be determined relative to the viewer. Furthermore, because the eyes, head, limbs, and body are constantly moving, computations about orientation, motion, and location must take place every time we wish to undertake an action. Details of object characteristics, such as color, are irrelevant to visuomotor guidance of the viewer-centered movements. That is, a detailed visual representation is not needed to guide hand action.

Milner suggests that the brain operates on a “need to know” basis. Having too much information may be counterproductive for any given system. In contrast with the viewer-centered system, the object-centered system must be concerned with such properties of objects as size, shape, color, and relative location so that the objects can be recognized when they are encountered in different visual contexts or from different vantage points. In this case, the details of the objects themselves (color, shape) are important. Knowing where the red cup is relative to the green one requires identifying each of them.

The temporal lobe codes relational properties of objects. Part of this control is probably in the polymodal region of the superior temporal sulcus, and another part is in the hippocampal formation. We return to the role of the temporal cortex in Chapter 15.

Guidance of Movement

The posterior parietal cortex has a role in the viewer-centered system. To accommodate the many different types of viewer-centered movements (eyes, head, limbs, body, and combinations of them) requires separate control systems. Consider, for example, that the control of the eyes is based on the optical axis of the eye, whereas the control of the limbs is probably based on the positions of the shoulders and hips. These movements are of very different types.

We have seen many visual areas in the posterior parietal region and multiple projections from the posterior parietal regions to the motor structures for the eyes (frontal eye fields, area 8) and limbs (premotor and supplementary motor). There also are connections to the prefrontal region (area 46) that have a role in short-term memory of the location of events in space.

The role of the posterior parietal region in visuomotor guidance is confirmed by the results of studies of neurons in the posterior parietal lobe of monkeys. The activity of these neurons depends on the concurrent behavior of the animal with respect to visual stimulation. In fact, most neurons in the posterior parietal region are active both during sensory input *and* during movement. For example, some cells show only weak responses to stationary visual stimuli but, if the animal makes an active eye or arm movement toward the stimulus *or even if it just shifts its attention to the object*, the discharge of these cells is strongly enhanced.

Some cells are active when a monkey manipulates an object and also respond to the structural features of the object, such as size and orientation. That is, the neurons are sensitive to the features of an object that determine the posture of the hand during manipulation.

A characteristic common to all the posterior parietal neurons is their responsiveness to movements of the eyes and to the location of the eye in its socket. When cells are stimulated at the optimum spot in their receptive fields, they discharge at the highest rate when the eyes are in a particular position. This discharge appears to signal the size of the eye movement, or **saccade**, necessary to move the visual target to the fovea of the retina.

In other words, these cells detect visual information and then move the eye to get the fine vision of the fovea to examine it. A curious aspect of many posterior parietal eye-movement cells is that they are particularly responsive to visual stimuli that are behaviorally relevant, such as a cue signaling the availability of a reward. This responsiveness has been interpreted as suggesting that these cells are affected by the “motivational” characteristics of information.

Stein summarized the responses of posterior parietal neurons by emphasizing that they all have two important characteristics in common. First, they receive combinations of sensory, motivational, and related motor inputs. Second, their discharge is enhanced when the animal attends to a target or makes a movement toward it. These neurons therefore are well suited to transforming the necessary sensory information into commands for directing attention and guiding motor output.

It is not possible to study the activity of single cells in the human posterior parietal region, but event-related potentials (ERPs) in response to visual stimuli can be recorded. Thus, when a stimulus is presented in one visual field, activation would be expected in the opposite hemisphere, which receives information from the contralateral visual field. Stephen Hillyard showed that, when a visual stimulus is presented, there is a large negative wave from about 100 to 200 ms later in the posterior parietal region. The wave is larger than that seen in the occipital cortex and is largest in the hemisphere contralateral to the stimulus.

Two interesting characteristics of these waves are reminiscent of neurons in monkeys. First, if a subject is asked to pay attention to a particular spot in one visual field, the ERP is largest when the stimulus is presented there rather than

elsewhere. Second, there is a large parietal response between 100 and 200 ms before eye movements. Pere Roland also showed that, when subjects direct their attention to visual targets, blood flow increases preferentially in the posterior parietal region.

Taken together, the results of electrophysiological and blood-flow studies in monkeys and humans support the general idea that the posterior parietal region plays a significant role in directing movements in space and in detecting stimuli in space. We can predict, therefore, that posterior parietal lesions impair the guidance of movements and perhaps the detection of sensory events.

The role of the superior parietal cortex in the control of eye movements has important implications for PET studies of visual processing. Recall from Chapter 13 that Haxby and colleagues showed an increase in blood flow in the posterior parietal cortex when subjects identified different spatial locations. This finding was taken as evidence that the dorsal stream of processing deals with “spatial processing.”

One difficulty with this interpretation, however, is that, when people solve spatial tasks, they move their eyes. The increased PET activation, therefore, could be due to the movement of the eyes, rather than to the processing of *where* the target actually is in space. Indeed, it has been demonstrated that, when people solve problems in which they must rotate objects mentally, they move their eyes back and forth. These saccades may indicate the ongoing activity of parietal circuits, but they also present a problem for PET studies. Thus there is a practical difficulty in constructing watertight experimental designs in brain-imaging studies.

The Complexity of Spatial Information

The second aspect of spatial representation is complexity. The control of limb or eye movements is concrete and relatively simple, but other types of viewer-centered representations are far more complex. For example, the concept of “left” and “right” is viewer centered but need not require movement. Patients, such as H. P., with posterior parietal lesions are impaired at distinguishing left from right. But there are spatial relations that are even more complex. For example, you can visualize objects and manipulate these mental images spatially as was done in the experiments described in the Snapshot on page 352. Patients with posterior parietal lesions are impaired at mental manipulations, such as those illustrated in Figure B in the Snapshot.

It seems likely that the ability to manipulate objects mentally is an extension of the ability to manipulate objects with the hands. Thus, mental manipulation is really just an elaboration of the neural control of actual manipulation, much as visual imagery is an elaboration of the neural record of actual visual input. The actual location of the cells taking part in mental manipulation is not known, but one guess is that it includes the temporoparietal polysensory regions that show such significant expansion in the human brain. (These regions constitute the third stream of processing illustrated in Figure 13.5.) This idea is speculative but based on the knowledge that this region is larger in the right hemisphere and that larger deficits in mental “spatial tasks” follow right-hemisphere lesions.

S N A P S H O T

Snapshot

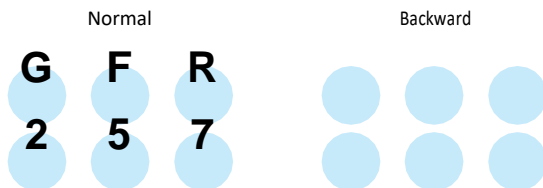
Measuring Parietal-Lobe Activation During Mental Rotation

To determine whether the posterior parietal cortex shows functional activation during a mental-rotation task, Alivisatos and Petrides used PET to measure regional blood flow during two different test conditions. Subjects

were first presented with letters or numbers and asked merely to press one key in response to a number and a different key in response to a letter. Their responses established a baseline level of activation, or control condition, for the experiment.

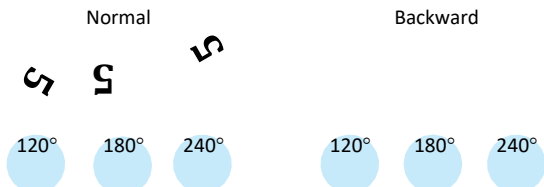
In the mirror-image test condition, subjects were presented letters or numbers either in the “normal” or backward, “mirror-image” orientation, as shown in Figure A. Their task was to press a different key to indicate each ori-

(A) Mirror-image test condition



(A) The alphanumeric stimuli used in their “normal” form (left) and in their “backward” form (right).

(B) Mental-rotation test condition



(B) An example of the rotation of the stimuli in the mental-rotation task.

entation. In the mental-rotation test, subjects were presented with the same stimuli, but in different orientations, as shown in Figure B. The subjects were required to make the same normal-versus-backward discrimination as in the mirror-image condition.

To determine whether the mirror-image or mental-rotation

tasks activated the parietal lobe, the baseline discrimination was subtracted from each test. Both tasks increased activation in the parietal cortex on the left and in a slightly more posterior temporal region on the left (Figure C). In addition, bilateral activation of the posterior temporal cortex was recorded. When the activation in the mirror-image condition was subtracted from that in the mental-rotation condition, the right hemisphere activation in the parietal and temporal lobes was no longer significant. Evidently both

Other Aspects of Parietal Function

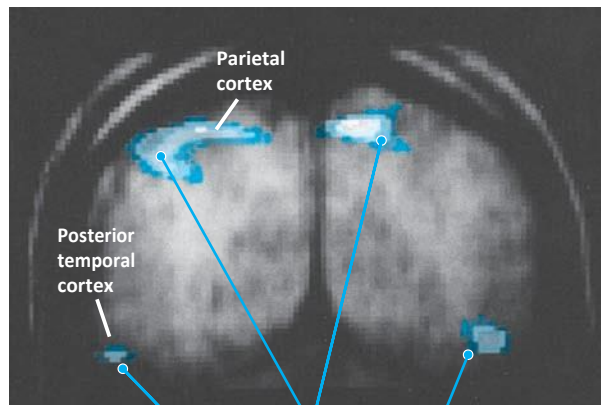
Three parietal-lobe symptoms do not fit obviously into a simple view of the parietal lobe as a visuomotor control center. These symptoms include difficulties with arithmetic, certain aspects of language, and movement sequences — deficits encountered in H. P.’s case.

Luria proposed that mathematics and arithmetic have a quasi-spatial nature analogous to the mental manipulation of concrete shapes but entailing abstract symbols. For example, addition and subtraction have spatial properties that are

important to calculating a correct solution. Consider the problem of subtracting 25 from 52. The “2” and “5” occupy different positions and have different meanings in the two numbers. There must be a “borrowing” from the 10’s column in 52 in order to subtract, and so on.

From this perspective, the reason that parietal-lobe patients such as H. P. experience **acalculia** (an inability to do arithmetic) stems from the spatial na-

(C) Average brain scan



After subtracting baseline discrimination from each test, both tasks increased activation in the parietal cortex and posterior temporal cortex.

(C) PET reactivity in the parietal cortex during mental rotation.

tasks require the same right parietal and temporal activation, whereas there was something different about the left parietal involvement in the two tasks.

The left difference is likely related to the increased difficulty in identifying alpha-numeric stimuli when they are rotated, which makes sense given that the left hemisphere is dominant for verbal processing. One puzzle, however, is that, in a parallel study, these researchers found that making similar manipulations with abstract stimuli produced a similar pattern of activation, even though the stimuli were not verbal. This finding suggests that the left parietal cortex has a role in active mental transformations of stimuli, regardless of the content of the stimulus material.

Notably, parietal activation in the two hemispheres is not in the same location, as you can see in the MRI in Figure C. The activation on the left is more rostral and inferior (area 40) than the activation on the right, which is more posterior and superior (area 7). This difference suggests that each hemisphere contributes a different type of processing to mental manipulation.

(B. Alivisatos and M. Petrides. Functional activation of the human brain during mental rotation. *Neuropsychologia* 35:111–118, 1997.)

ture of the task. Indeed, if parietal-lobe patients are given simple problems such as $6 - 4$, they usually solve them because the spatial demands are few. Even when the problems are somewhat more difficult, such as $984 - 23$, the patients have little problem. When more-complex manipulations, such as borrowing, must be made, however, the patients' abilities to do arithmetic break down, as in $983 - 24$. Thus, arithmetic operations may depend on the polysensory tissue at the left temporoparietal junction.

Language has many of the same demands as arithmetic. The words "tap" and "pat" have the same letters, but the spatial organization is different. Similarly, the phrases "my son's wife" and "my wife's son" have identical words but very different meanings. These observations have led Luria and others to suggest that language can be seen as quasispatial. Patients such as H. P. may have a clear understanding of individual elements, but they are unable to

understand the whole when the syntax becomes important. This ability, too, may depend on the polysensory region at the temporoparietal junction.

The deficit in organizing individual elements of behavior can be seen not only in language but in movement as well. People with parietal-lobe injuries have difficulty in copying sequences of movements, a problem that we shall return to shortly.

In summary, the posterior parietal lobe controls the visuomotor guidance of movements in egocentric (that is, viewer-centered) space. This control is most obvious in regard to reaching and to eye movements needed to grasp or manipulate objects. The eye movements are important, because they allow the visual system to attend to particular sensory cues in the environment. The polymodal region of the posterior parietal cortex is also important in various aspects of “mental space,” ranging from arithmetic and reading to the mental rotation and manipulation of visual images to sequencing movements.

Somatosensory Symptoms of Parietal-Lobe Lesions

In this section, we consider the somatosensory symptoms associated with damage to the postcentral gyrus (see Figure 14.1A and areas 1, 2, and 3 in Figure 14.1B) and the adjacent cortex (areas PE and PF in Figure 14.1C).

Somatosensory Thresholds

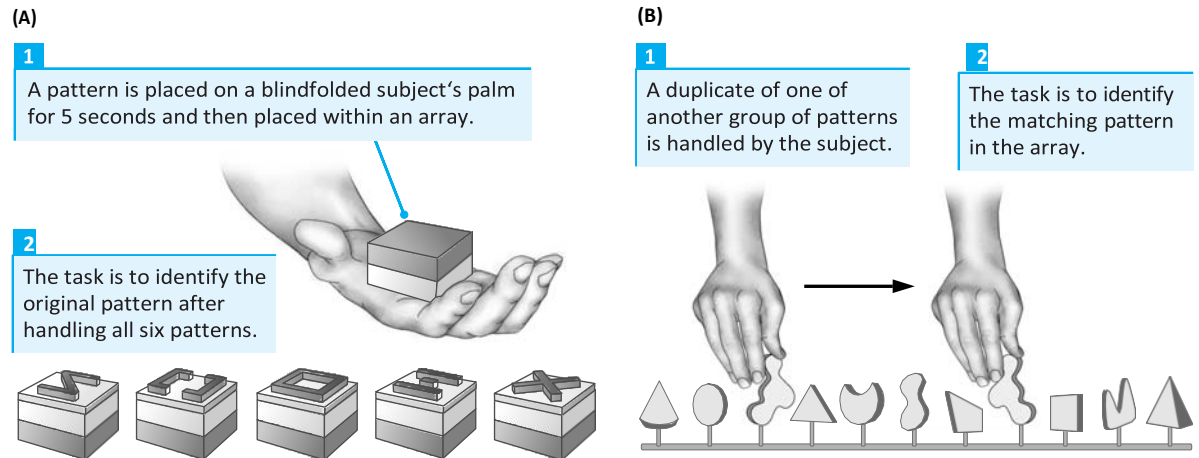
Damage to the postcentral gyrus is typically associated with marked changes in somatosensory thresholds. The most thorough studies of these changes were done by Josephine Semmes and her colleagues on World War II veterans with missile wounds to the brain and by Suzanne Corkin and her coworkers on patients who had undergone cortical surgery for the relief of epilepsy.

Both research groups found that lesions of the postcentral gyrus produced abnormally high sensory thresholds, impaired position sense, and deficits in stereognosis (tactile perception). For example, in the Corkin study, patients performed poorly at detecting a light touch to the skin (pressure sensitivity), at determining if they were touched by one or two sharp points (two-point threshold), and at localizing points of touch on the skin on the side of the body contralateral to the lesion. If blindfolded, the patients also had difficulty in reporting whether the fingers of the contralateral hand were passively moved.

Lesions of the postcentral gyrus may also produce a symptom that Luria called **afferent paresis**. Movements of the fingers are clumsy because the person has lost the necessary feedback about their exact position.

Somatoperceptual Disorders

The presence of normal somatosensory thresholds does not preclude the possibility of other types of somatosensory abnormalities. First, there is **astereognosis** (from the Greek *stereo*, meaning “solid”), which is the inability to



recognize the nature of an object by touch. This disturbance can be demonstrated in tests of tactile appreciation of object qualities, illustrated in Figure 14.3. In these tests, objects are placed on the palms of blindfolded subjects or the subjects are told to handle shapes. The task is to match the original shape or object to one of several alternatives solely on the basis of tactile information.

A second somatoperceptual disorder, **simultaneous extinction**, can be demonstrated only by special testing procedures. The logic of this test is that a person is ordinarily confronted by an environment in which many sensory stimuli impinge simultaneously, yet the person is able to distinguish and perceive each of these individual sensory impressions. Thus, a task that presents stimuli one at a time represents an unnatural situation that may underestimate sensory disturbances or miss them altogether.

To offer more-complicated sensory stimulation, two tactile stimuli are presented simultaneously to the same or different body parts. The objective of such double simultaneous stimulation is to uncover those situations in which both stimuli would be reported if applied singly, but only one would be reported if both were applied together, as illustrated in Figure 14.4. A failure to report one stimulus is usually called **extinction** and is most commonly associated with damage to the somatic secondary cortex (areas PE and PF), especially in the right parietal lobe.

Figure 14.3 Tests for tactile appreciation of objects. (A) A pattern is placed on the blindfolded subject's palm for 5 seconds and then placed within the array. The task is to handle all six patterns and identify which of them is the original pattern. (B) The subject handles a duplicate of one of the patterns. The task is to identify, again by handling, the matching pattern in the array. (After Teuber, 1978.)

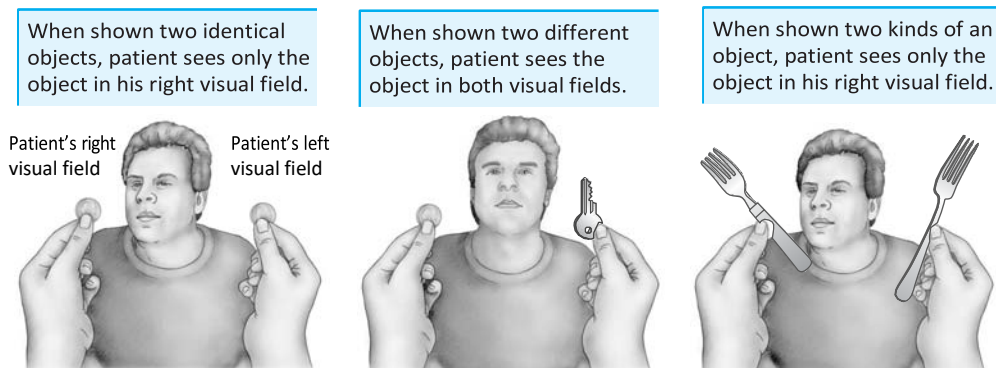


Figure 14.4 Testing for extinction in a stroke patient. The patient responds differently, depending on whether objects in the left and right visual fields are similar or different.

Blind Touch

Evidence that patients can identify the location of a visual stimulus even though they deny “seeing” it was presented in Chapter 13. Jacques Paillard and his colleagues reported the case of a woman who appears to have a tactile analogue of blindsight. This woman had a large lesion of areas PE, PF, and some of PG, resulting in a complete anesthesia of the right side of the body so severe that she was likely to cut or burn herself without being aware of it. Nevertheless, she was able to point with her left hand to locations on her right hand where she had been touched, even though she failed to report feeling the touch.

Although reported in a single case, the phenomenon is clearly reminiscent of blindsight. The presence of a tactile analogue of blindsight is important because it suggests the existence of two tactile systems — one specialized for detection and the other for localization. Such specialization may be a general feature of sensory system organization.

Somatosensory Agnosias

There are two major types of somatosensory agnosias: astereognosis (see the preceding discussion of somatoperceptual disorders) and **asomatognosia** — the loss of knowledge or sense of one’s own body and bodily condition. Although astereognosis is essentially a disorder of tactile appreciation (see Figure 14.3), it is included here because it is often described clinically simply as an agnosia.

Asomatognosia is one of the most curious of all agnosias. It is an almost unbelievable syndrome — until you actually observe it. The varieties of asomatognosias include **anosognosia**, the unawareness or denial of illness; **anosodiaphoria**, indifference to illness; **autopagnosia**, an inability to localize and name body parts; and **asymbolia for pain**, the absence of normal reactions to pain, such as reflexive withdrawal from a painful stimulus.

Asomatognosias may affect one or both sides of the body, although most commonly the left side, as a result of lesions in the right hemisphere. An exception comprises the autopagnosias, which usually result from lesions of the left parietal cortex. The most common autopagnosia is **finger agnosia**, a condition in which a person is unable either to point to the various fingers of either hand or show them to an examiner. A curious relation exists between finger agnosia and dyscalculia (difficulty in performing arithmetic operations). When children learn arithmetic, they normally use their fingers to count. We might predict that children who are unable to use their fingers to count, such as those with finger agnosia, would have difficulty learning arithmetic. In fact, children with a condition known as spina bifida have finger agnosia and have been found to be terrible at arithmetic.

Symptoms of Posterior Parietal Damage

The clinical literature describes a bewildering array of symptoms of posterior parietal injury. We will restrict our consideration here to the most commonly observed disorders.

Balint's Syndrome

In 1909, R. Balint described a patient whose bilateral parietal lesion was associated with rather peculiar visual symptoms. The patient had full visual fields and could recognize, use, and name objects, pictures, and colors normally. Nevertheless, he had three unusual symptoms:

1. Although he spontaneously looked straight ahead, when an array of stimuli was placed in front of him, he directed his gaze 35° to 40° to the right and perceived only what was lying in that direction. Thus, he could move his eyes but could not fixate on specific visual stimuli.
2. When his attention was directed toward an object, he did not notice other stimuli. With urging, he could identify other stimuli placed before him, but he quickly relapsed into his former neglect. Balint concluded that the patient's field of attention was limited to one object at a time, a disorder that made reading very difficult because each letter was perceived separately. (This disorder is often referred to as *simultagnosia*.)
3. The patient had a severe deficit in reaching under visual guidance. Balint described this symptom as *optic ataxia*. He noted that the patient could still make accurate movements directed toward the body, presumably by using tactile or proprioceptive information, but could not make visually guided movements.

Although Balint's syndrome is quite rare, optic ataxia is a common symptom of posterior parietal lesions and can develop after unilateral lesions. Consider the following description of a patient of Damasio and Benton:

She consistently misreached for targets located in the nearby space, such as pencils, cigarettes, matches, ashtrays and cutlery. Usually she underreached by 2 to 5 inches, and then explored, by tact [touch], the surface path leading to the target. This exploration, performed in one or two groping attempts, was often successful and led straight to the object. Occasionally, however, the hand would again misreach, this time on the side of the target and beyond it. Another quick tactualy guided correction would then place the hand in contact with the object. In striking contrast to the above difficulties was the performance of movements which did not require visual guidance, such as buttoning and unbuttoning of garments, bringing a cigarette to the mouth, or pointing to some part of her body. These movements were smooth, quick and on target. (Damasio and Benton, 1979, p. 171)

The deficits in eye gaze and visually guided reaching are most likely to result from lesions in the superior parietal region (area PE). Optic ataxia does not accompany lesions in the inferior parietal region, suggesting a clear functional dissociation of the two posterior parietal regions.

Contralateral Neglect and Other Symptoms of Right Parietal Lesions

Critchley remarked in his 1953 textbook on the parietal lobes that the symptoms of parietal lesions differ widely – one patient showing only a few abnormal signs that are mild in nature but another showing an intricate clinical

picture with elaborate symptoms. What causes this diversity is still not known. We must keep this uncertainty in mind as we consider the symptoms of right parietal lesions because the range and severity of symptoms varies widely among individual patients.

Contralateral Neglect

A perceptual disorder subsequent to right parietal lesions was described by John Hughlings-Jackson in 1874. Not until the 1940s, however, was the effect of right parietal lesions clearly defined by Alan Paterson and Oliver Zangwill. A classic paper by John McFie and Zangwill, published in 1960, reviewed much of the previous work and described several symptoms of right parietal lesions, which are illustrated in the following patient.

Mr. P., a 67-year-old man, had suffered a right parietal stroke. At the time of our first seeing him (24 hours after admission), he had no visual-field defect or paresis. He did, however, have a variety of other symptoms:

- Mr. P. neglected the left side of his body and of the world. When asked to lift up his arms, he failed to lift his left arm but could do so if one took his arm and asked him to lift it. When asked to draw a clock face, he crowded all the numbers onto the right side of the clock. When asked to read compound words such as “ice cream” and “football,” he read “cream” and “ball.” When he dressed, he did not attempt to put on the left side of his clothing (a form of dressing apraxia), and when he shaved, he shaved only the right side of his face. He ignored tactile sensation on the left side of his body. Finally, he appeared unaware that anything was wrong with him and was uncertain about what all the fuss was about (anosagnosia). Collectively, these symptoms are referred to as **contralateral neglect**.
- He was impaired at combining blocks to form designs (constructional apraxia) and was generally impaired at drawing freehand with either hand, copying drawings, or cutting out paper figures. When drawing, he often added extra strokes in an effort to make the pictures correct, but the drawings generally lacked accurate spatial relations. In fact, it is common for patients showing neglect to fail to complete the left side of the drawing, as illustrated in Figure 14.5.
- Mr. P. had a topographical disability, being unable to draw maps of well-known regions from memory. He attempted to draw a map of his neighborhood, but it was badly distorted with respect to directions, the spatial arrangement of landmarks, and distances. Despite all these disturbances, Mr. P. knew where he was and what day it was, and he could recognize his family’s faces. He also had good language functions: he could talk, read, and write normally.

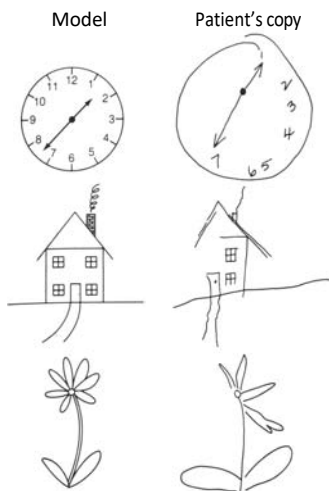


Figure 14.5 Drawings copied by a patient with contralateral neglect. (From F. E. Bloom and A. Lazerson. *Brain, Mind, and Behavior*, 2ded. New York: W. H. Freeman and Company, p. 300. Copyright © 1988.)

Contralateral neglect as observed in Mr. P. is one of the most fascinating symptoms of brain dysfunction. Neglect occurs in visual, auditory, and somesthetic (somatosensory) stimulation on the side of the body or space or both opposite the lesion. Neglect may be accompanied by denial of the deficit.

Recovery passes through two stages. **Allesthesia** is characterized by the person’s beginning to respond to stimuli on the neglected side as if the stimuli were on the unlesioned side. The person responds and orients to visual, tactile, or auditory stimuli on the left side of the body as if they were on the right.

The second stage of recovery, noted earlier, is simultaneous extinction (see Figure 14.4). The person responds to stimuli on the hitherto neglected side unless both sides are stimulated simultaneously, in which case he or she notices only the stimulation on the side ipsilateral to the lesion.

Neglect presents obstacles to understanding. What is the location of the lesion that produces this effect? Figure 14.6A is a composite drawing of the region damaged (as inferred from brain scans) in 13 patients with neglect as described by Ken Heilman and Robert Watson. The area of most overlap (Figure 14.6B) among the lesions was the right inferior parietal lobule.

Note, however, that contralateral neglect is occasionally observed subsequent to lesions to the frontal lobe and cingulate cortex, as well as to subcortical structures including the superior colliculus and lateral hypothalamus. What is not clear is whether the same phenomenon results from lesions in these various locations.

Why does neglect occur? The two main theories argue that neglect is caused by either (1) defective sensation or perception or (2) defective attention

or orientation. The strongest argument favoring the theory of defective sensation or perception is that a lesion to the parietal lobes, which receive input from all the sensory regions, can disturb the integration of sensation into perception. Derek Denny-Brown and Robert Chambers termed this function *morphosynthesis* and its disruption *amorphosynthesis*.

A current elaboration of this theory proposes that neglect follows a right parietal lesion because the integration of the spatial properties of stimuli becomes disturbed. As a result, although stimuli are perceived, their location is uncertain to the nervous system and they are consequently ignored. The neglect is thought to be unilateral because, in the absence of right-hemisphere function, the left hemisphere is assumed to be capable of some rudimentary spatial synthesis that prevents neglect of the right side of the world. This rudimentary spatial ability cannot compensate, however, for the many other behavioral deficits resulting from right parietal lesions.

Critchley and, later, others suggested that neglect results from defective attention or orientation; that is, an inability to attend to input that has in fact been registered. This suggestion was elaborated most recently by Heilman and Watson. They propose that neglect is manifested by a defect in orienting to stimuli; the defect results from the disruption of a system whose function is to “arouse” the person when new sensory stimulation is present.

Object Recognition

Elizabeth Warrington and her colleagues described another common symptom of right-parietal-lobe lesion: although able to recognize objects shown in familiar views, patients having these lesions are badly impaired at recognizing objects shown in unfamiliar views (Figure 14.7). Warrington concluded

that the deficit is not in forming a gestalt, or concept—in this case, (A) of “bucket”—but rather in perceptual classification—the mechanism for categorizing information as being part of the idea “bucket.” Such allocation can be seen as a type of a spatial matching in which the common view of an object must be rotated spatially to match the novel view. Warrington and Taylor suggested that the focus for this deficit is roughly the right inferior parietal lobule, the same region proposed as the locus of contralateral neglect (see Figure 14.6B).

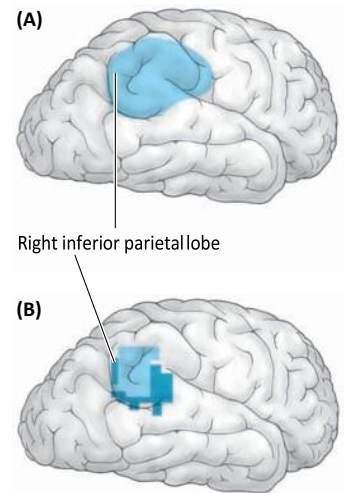
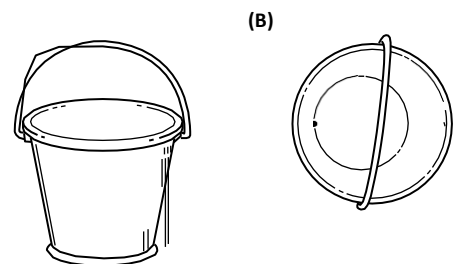


Figure 14.6 The locus of right parietal symptoms. (A) Composite map of the region damaged (inferred from brain scans) in 13 patients with contralateral neglect as described by Heilman and Watson. The area of greatest overlap is the right inferior parietal lobule. (B) Composite outline of the region of overlap among lesions producing deficits in Warrington and Taylor’s test of recognition of objects seen in unfamiliar views. The lightly shaded region is the area of maximal overlap. Note the locational similarity between parts A and B.

Figure 14.7 Drawing of a bucket in (A) familiar and (B) unfamiliar views. Patients with right parietal lesions have difficulty in recognizing objects in unfamiliar views, such as that shown in part B.



The Gerstmann Syndrome and Other Left Parietal Symptoms

In 1924, Josef Gerstmann described a patient with an unusual disorder subsequent to a left parietal stroke: finger agnosia, an asomatognosia described earlier in the chapter. Gerstmann's patient was unable to name or indicate recognition of the fingers on either hand. This symptom aroused considerable interest, and, in the ensuing years, three other symptoms were reported to accompany finger agnosia: right-left confusion, **agraphia** (inability to write), and acalculia. These four symptoms collectively became known as the Gerstmann syndrome.

Gerstmann and others argued that these symptoms accompany a circumscribed lesion in the left parietal lobe, roughly corresponding to the angular gyrus (area PG). If these four symptoms arose as a group, the patient was said to demonstrate the Gerstmann syndrome, and the lesions could be localized in the angular gyrus (see Figure 14.1A). The Gerstmann syndrome is a doubtful diagnostic tool in routine investigations, but all the symptoms can be associated with left parietal lesions.

Various other symptoms of left parietal lesions are illustrated in the following case history. On 24 August 1975, S. S., an 11-year-old boy, suddenly had a seizure, which was characterized by twitching on the right side of the body, particularly the arm and face. He was given anticonvulsant medication and was free of symptoms until 16 September 1975, when he began to write upside down and backward. S. S. was immediately referred to a neurologist, who diagnosed a left parietal malignant astrocytoma. Careful neuropsychological assessment revealed a number of symptoms characteristic of left parietal lesions:

- *Disturbed language function.* S. S. was unable to write even his name (agraphia), had serious difficulties in reading (dyslexia), and spoke slowly and deliberately, making many errors in grammar (dysphasia).
- *Apraxia.* S. S. was unable to combine blocks to form designs and had difficulty learning a sequence of novel movements of the limbs (see the next subsection).
- *Dyscalculia.* He was very poor at mental arithmetic and could not solve even simple additions and subtractions.
- *Recall.* He had an especially low digit span, being able to master the immediate recall of only three digits, whether they were presented orally or visually.
- *Right-left discrimination.* He was totally unable to distinguish left from right, responding at random on all tests of this ability.
- *Right hemianopia.* Probably because his tumor had damaged the geniculostriate connections, as S. S.'s tumor progressed, movement of the right side of his body became disturbed as the tumor placed pressure on the frontal lobe.

By the end of October 1975 S. S. died; neither surgery nor drug therapy could stop the growth of the tumor. The symptoms that S. S. exhibited resemble those of other patients whom we have seen with left parietal lesions, including H. P., whose story begins this chapter. Curiously, S. S. did not have finger agnosia, one of the Gerstmann symptoms, illustrating the point that even very large lesions do not produce the same effects in every patient.

Apraxia and the Parietal Lobe

Apraxia is a disorder of movement in which loss of skilled movement is not caused by weakness, an inability to move, abnormal muscle tone or posture, intellectual deterioration, poor comprehension, or other disorders of movement such as tremor. Among the many types of apraxia, we shall focus on two: ideomotor apraxia and constructional apraxia.

In ideomotor apraxia, patients are unable to copy movements or to make gestures (for example, to wave “hello”). Patients with left posterior parietal lesions often present ideomotor apraxia. Kimura showed that the deficits in such patients can be quantified by asking them to copy a series of arm movements such as those illustrated in Figure 14.8A. Patients with left-parietal-lobe lesions are grossly impaired at this task, whereas people with right-parietal-lobe lesions perform the task normally. We return to ideomotor apraxia in Chapter 22.

In constructional apraxia, a visuomotor disorder, spatial organization is disordered. Patients with constructional apraxia cannot assemble a puzzle, build a tree house, draw a picture, or copy a series of facial movements (Figure 14.8B). Constructional apraxia can develop after injury to either parietal lobe, although debate over whether the symptoms are the same after left- and right-side lesions is considerable (see the review by Benton). Nonetheless, constructional apraxia often accompanies posterior parietal lesions.

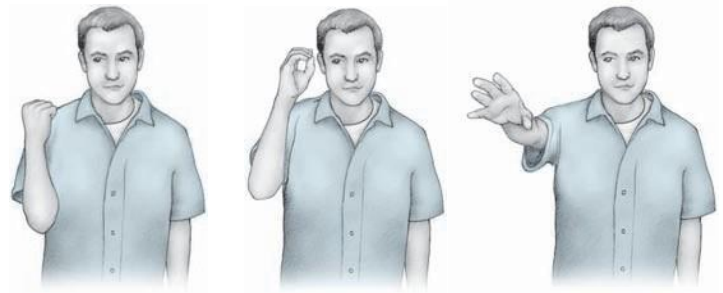
You can view both ideomotor and constructional apraxia as disturbances of movement that result from a disruption of the parietofrontal connections that control movement. Mountcastle proposed that the posterior parietal cortex receives afferent signals not only of the tactile and visual representation of the world but also of the position and movement of the body. He proposed that the region uses this information to function as “a command apparatus for operation of the limbs, hands, and eyes within immediate extrapersonal space.”

Thus, the parietal lobe not only integrates sensory and spatial information to allow accurate movements in space but also functions to direct or guide movements in the immediate vicinity of the body. Both ideomotor and constructional apraxia can be seen as examples of a dysfunction in this guidance system.

Drawing

Although drawing deficits are known to arise subsequent to lesions in either hemisphere, the deficits in drawing are generally believed to be greater after damage to the right hemisphere than after damage to the left, and the right parietal damage is believed to have the greatest influence on drawing ability.

(A) Serial arm-movement copying test



(B) Serial facial-movement copying test



Figure 14.8 Testing for apraxia.

(A) Sample items from a serial arm-movement copying test. To assess ideomotor apraxia, subjects are asked to copy each movement in the series as accurately as they can. (B) Sample items from a serial facial-movement copying test used to assess constructional apraxia.

This conclusion is consistent with the general idea that the right hemisphere plays a dominant role in spatial abilities, but it may not be correct. Rather, it appears that disturbances in drawing differ, depending on whether the lesion is in the right or the left hemisphere. For example, Kimura and Faust asked a large sample of patients to draw a house and a man. Apraxic or aphasic left-hemisphere patients did very poorly, producing fewer recognizable drawings and fewer lines than did right-hemisphere patients. In contrast, right-hemisphere patients tended to omit details from the left side of their drawings and to rotate the drawings on the page.

In sum, drawing is a complex behavior that may require verbal as well as nonverbal (for example, spatial) processes. If asked to draw a bicycle, many people will make a mental checklist of items to include (fenders, spokes, chain, and so on). In the absence of language, we would expect such people to draw less-complete bicycles. Further, if patients are apraxic, there is likely to be a deficit in making the required movements. Similarly, the parts of a bicycle have a particular spatial organization. If spatial organization is poor, the drawing is likely to be distorted.

Spatial Attention

As we move about the world, we are confronted with a vast array of sensory information that cannot possibly all be treated equally by the nervous system. Thus, the brain must select certain information to process. Consider, for example, the sensory overload to which we are subjected when we stop to chat with an old friend in a department store. Several other people may be around, and there will certainly be displays of various items to purchase, competing sounds (others talking, music, cash registers), novel odors, and so on. Nonetheless, we can orient to a small sample of the incoming information and ignore most of the other input. In fact, we may focus to the exclusion of other, potentially more important information. Cognitive psychologists refer to this orienting of the sensory systems as *selective attention*. Thus, we are said to attend to particular stimuli.

Posner proposed that one function of the parietal cortex is to allow attention to shift from one stimulus to another, a process that he calls **disengagement**. Consider our earlier example of dining with a friend. As we eat, we shift from peas to bread to wine. We are disengaging each time that we shift from one food to another. One aspect of this disengagement is that we must reset our visuomotor guidance system to form the appropriate movements for the next target. We can extend this idea to mental manipulation of objects and spatial information, too: we must reset the system for the next operation. We return to the problem of selective attention in Chapter 22.

Disorders of Spatial Cognition

We use the term “spatial cognition” to refer to a broad category of abilities that require mentally using or manipulating spatial properties of stimuli, including the ability to mentally manipulate images of objects and maps. The mental-rotation tasks illustrated in Figures 12.2 and 21.11 provide good examples. Another is the ability to follow an upside-down map.

There is little doubt that posterior lesions, most likely including the region of PG and the polymodal cortex of the superior temporal sulcus, produce deficits in mental-rotation and map-reading tasks. Although it is widely assumed in the neuropsychological literature that the right hemisphere is “spatial” and that deficits in spatial cognition should thus result from right posterior lesions, the clinical evidence is far from convincing. Indeed, there is little doubt that both left- and right-hemisphere lesions produce deficits in spatial-cognition tasks.

The emerging view, however, is that left- and right-hemisphere lesions have different effects on the performance of spatial cognition. For example, Corballis suggested that mental rotation requires two different operations: (1) the mental imaging of the stimulus and (2) the manipulation of the image. Newcombe and Ratcliff suggested that the left-hemisphere deficit may result from an inability to generate an appropriate mental image. In Chapter 13, we saw that visual-imaging deficits result from left occipital lesions. In contrast, the right-hemisphere deficit may be due to an inability to perform operations on this mental image.

Deficits in the ability to use topographical information are more likely to be associated with damage to the right hemisphere than to the left. Such disorders include the loss of memory of familiar surroundings, the inability to locate items such as countries or cities on a map, and the inability to find one’s way about the environment. Not surprisingly, such deficits are likely to be associated with other visual deficits (such as contralateral neglect or visual agnosia), but patients have been described with relatively specific disorders of topographical orientation.

Emillio de Renzi concluded that injury to the right posterior hemisphere is a prerequisite for such disorders. Newcombe and Ratcliff noted that such disorders are often associated with injury to the right posterior cerebral artery and thus are likely to include right occipitotemporal and right hippocampal regions. When the parietal cortex is affected, it is most likely to be the inferior part, probably including area PG and the superior temporal sulcus.

Left and Right Parietal Lobes Compared

In their classic paper, McFie and Zangwill compared the symptoms of patients with left or right parietal lesions. Although they found some overlapping symptoms, the asymmetry was clear (Table 14.1). In addition, as noted earlier, ideomotor apraxia is more likely to be associated with left parietal lesions.

A puzzling feature of the McFie and Zangwill study noted in Table 14.1 is that lesions to the two hemispheres produce some overlapping symptoms, despite the clear asymmetry. The results of neuropsychological studies tend to emphasize the asymmetry of lesion effects, but the overlapping symptoms are important theoretically. Indeed, as noted earlier, both constructional apraxia and disorders of spatial cognition are poorly lateralized. Many theories of hemispheric asymmetry discussed in Chapter 11 do not predict such ambiguity

Table 14.1 Effects of left- and right-parietal-lobe lesions compared

	PERCENTAGE OF SUBJECTS WITH DEFICIT*	
	Left (%)	Right(%)
Unilateral neglect	13	67
Dressing disability	13	67
Cube counting	0	86
Paper cutting	0	90
Topographical loss	13	50
Right-left discrimination	63	0
Weigl’s sorting test	83	6

*Note the small but significant overlap in symptoms of left and right lesions. Source: Based on data presented by McFie and Zangwill, 1960.

in symptom localization and tend to assume far greater dissociation of lesion effects than is actually observed.

One explanation for the overlapping symptoms relates to the concept of preferred cognitive mode, introduced in Chapter 11. There it was noted that many problems can be solved by using either a verbal cognitive mode or a spatial nonverbal cognitive mode. Genetic, maturational, and environmental factors may predispose people to use different cognitive modes. For example, a complex spatial problem, such as reading an upside-down map, can be solved either directly, by “spatial cognition” (the directions to travel are intuited spatially) or indirectly, by “verbal cognition” (the spatial information is encoded into words and the problem is solved by being “talked” through step by step).

People who are highly verbal prefer the verbal mode even when it is less efficient; we expect lesions of the left parietal lobe in these people to disturb functions that ordinarily are disrupted preferentially by right parietal lesions. Little direct evidence favors this explanation of functional overlap, but it is a provocative idea that accounts in part for individual differences as well as for the apparent functional overlap revealed by the results of lesion studies.

Major Symptoms and Their Assessment

Table 14.2 summarizes the major symptoms of parietal-lobe lesions. Damage to the anterior parietal cortex, including area PE, produces deficits in various somatosensory functions. Damage to the posterior parietal regions produces most of the other disorders.

Table 14.2 Summary of major symptoms of parietal-lobe damage

Symptom	Most probable lesion site	Basic reference
Disorders of tactile function	Areas 1, 2, 3	Semmes et al., 1960 Corkin et al., 1970
Tactile agnosia	Area PE	Hecaen and Albert, 1978 Brown, 1972
Defects in eye movement	Areas PE, PF	Tyler, 1968
Misreaching	Areas 5, 7	Damasio and Benton, 1979
Manipulation of objects	Areas PF, PG	Pause et al., 1989
Apraxia	Areas PF, PG, left	Heilman and Rothi, 1993 Kimura, 1980
Constructional apraxia	Area PG	Benton, 1990
Acalculia	Areas PG, STS*	Levin et al., 1993
Impaired cross-modal matching	Areas PG, STS	Butters and Brody, 1968
Contralateral neglect	Area PG right	Heilman et al., 1993
Impaired object recognition	Area PG right	Warrington and Taylor, 1973
Disorders of body image	Area PE?	Benton and Sivan, 1993
Right-left confusion	Areas PF, PG	Semmes et al., 1960
Disorders of spatial ability	Areas PE, PG	Newcombe and Ratcliff, 1990
Disorders of drawing	Area PG	Warrington et al., 1966 Kimura and Faust, 1987

*STS, superior temporal sulcus.

Table 14.2 also lists the regions most likely to be associated with the deficits, but few studies clearly demonstrate anatomical dissociations of such deficits. A major difficulty in dissociating the regions is that natural lesions rarely respect anatomical boundaries and affect only the neocortex. And, in contrast with the frontal and temporal lobes, which are often implicated in epilepsy and thus may be removed surgically, the parietal lobe is rarely epileptogenic, and so surgical removal is rare, as is the opportunity for follow-up research.

Clinical Neuropsychological Assessment

As we have seen, restricted lesions of the parietal cortex produce a wide variety of behavioral changes. Behavioral tests used to evaluate brain damage in neurologically verified cases could be logically employed to predict the locus and extent of damage or dysfunction in new cases. (See Chapter 28 for more detail on the rationale of neuropsychological assessment.) This section briefly summarizes the behavioral tests that have proved sensitive and valid predictors of brain injury (Table 14.3). Although these tests do not assess all the symptoms of parietal injury, they do evaluate a broad range of parietal-lobe functions. It would be highly unusual for a person to perform normally on all these tests yet show other symptoms of parietal-lobe damage. In addition to these tests, Howard Goodglass and Edith Kaplan describe a good series of tests in their “parietal lobe battery.”

Table 14.3 Standardized clinical neuropsychological tests for parietal-lobe damage

Function	Test	Basic reference
Somatosensory threshold	Two-point discrimination	Corkin et al., 1970
Tactile form recognition	Seguin-Goddard Form Board Tactile patterns	Teuber and Weinstein, 1954 Benton et al., 1983
Contralateral neglect	Line bisection	Schenkenberg et al., 1980
Visual perception	Gollin Incomplete Figures Mooney Closure	Warrington and Rabin, 1970 Milner, 1980
Spatial relations	Right-left differentiation	Benton et al., 1983
Language		
Speech comprehension	Token	de Renzi and Faglioni, 1978
Reading comprehension	Token	
Apraxia	Kimura Box	Kimura, 1977

Note: These standardized tests have been validated on large samples of patients with known localized brain damage.

Somatosensory Threshold

Recall that subsequent to lesions of the postcentral gyrus, the somatosensory threshold increases on the contralateral side of the body. The two-point discrimination test requires the blindfolded subject to report whether he or she felt one or two points touch the skin (usually on the face or on the palm of the hand). The distance between the points is at first very large (say, 3 cm) and is gradually reduced until the subject can no longer perceive two points. In extreme cases, the process is reversed: the distance must be increased to find when the subject first perceives two points.

Tactile Form Recognition

In the Seguin-Goddard Form Board test, the blindfolded subject manipulates 10 blocks of different shapes (star, triangle, and so forth) and attempts to place them in similarly shaped holes on a form board. When the test is completed, the form board and blocks are removed and the subject is asked to draw the board from memory. The precise locus of the lesion producing deficits on this test is controversial, and no claims have been proved. Nevertheless, the results of research on tactile performance in monkeys with parietal lesions indicate that blindfolded tactile recognition is probably sensitive to lesions of areas PE and PF, whereas, in humans, the drawing part – a test of both memory and cross-modal matching – is probably sensitive to lesions in area PG.

Contralateral Neglect

A variety of tests have been devised, but we favor the line-bisection test by Schenkenberg and colleagues because it is particularly sensitive. In this test, the subject is asked to mark the middle of each of a set of 20 lines. Each line is a different length and is located at a different position on the page – some left of center, some in the middle, and some right of center. Patients showing contralateral neglect typically fail to mark the lines on the left side of the page.

Visual Perception

Visual perceptual capacity is easily assessed by either the Mooney Closure Test or the Gollin Incomplete-Figures Test. In both tasks, a series of incomplete representations of faces or objects is presented, and the subject must combine the elements to form a gestalt and identify the picture. These tests are especially sensitive to damage at the right parietotemporal junction, presumably in regions of the ventral visual stream.

Spatial Relations

In the right–left differentiation test, a series of drawings of hands, feet, ears, and so on, are presented in different orientations (upside down, rear view, and so forth), and the subject’s task is to indicate whether the drawing is of the left or the right body part. In a verbal variant of this test, subjects are read a series of commands (for example, “Touch your right ear with your left hand”) that are to be carried out. Both tests are very sensitive to left-parietal-lobe damage, but caution is advised, because subjects with left-frontal-lobe damage also are often impaired at these tasks.

Language

The Token Test is an easily administered test of language comprehension. Twenty tokens – four shapes (large and small circles, large and small squares) in each of five colors (white, black, yellow, green, red) – are placed in front of the subject. The test begins with simple tasks (for example, touching the white circle) and becomes progressively more difficult (for example, touching the large yellow circle and the large green square).

A Token Test of reading comprehension can also be given by having the subject read the instructions out loud and then carry them out. We have not considered language a function of the parietal lobe, but the posterior speech

zone borders on area PG. Thus, injuries affecting PG often include temporal speech-related cortex, and aphasia is observed.

Apraxia

It is unfortunate that there are no standardized tests of apraxia analogous to the token test for aphasia. However, the Kimura box test (Figure 14.9) is probably the best test currently available. The subject is required to make consecutive movements of pushing a button with the index finger, pulling a handle with four fingers, and pressing a bar with the thumb. This test is done very poorly by apraxics, and many of them appear unable to perform this very simple series of movements even with extensive practice.

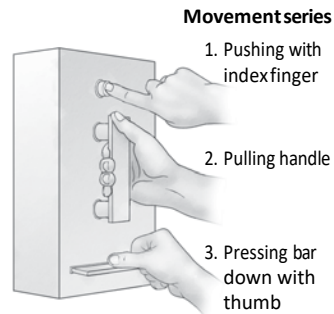


Figure 14.9 Kimura box test. Subjects are required to learn the movement series that consists of pushing the top button with the index finger, pulling the handle with four fingers, and pressing a bar with the thumb. Apraxic subjects are impaired at this task, and they may be unable to learn it at all.

Summary

The parietal lobe can be divided into three functional zones for somatosensory processes, movement, and spatial cognition. The most anterior zones primarily take part in somatosensory functions. The superior parietal region primarily controls the visual guidance of movements of the hands and fingers, limbs, head, and eyes. This region has expanded in humans to include regions controlling not only the actual manipulation of objects but also the mental manipulation of objects. Movements around the body, or in the imagination, necessarily include the space around the body and the object. Thus, the posterior parietal region can be conceived of as having a “spatial” function, although the precise nature of this spatial function is far from clear.

The inferior parietal region has a role in processes related to spatial cognition and in what have been described as quasi-spatial processes, such as are used in arithmetic and reading.

Damage to the somatosensory regions of the parietal lobe produces deficits in tactile functions, ranging from simple somatosensation to the recognition of objects by touch.

Posterior parietal-lobe injury interferes with the visual guidance of hand and limb movements. Thus, for left parietal injury, there may be limb apraxias; whereas, for right parietal injury, constructional apraxias may result. Left parietal injury also produces a range of cognitive symptoms including deficits in arithmetic and writing; right parietal injury produces a complementary range of symptoms including contralateral neglect and various deficits in spatial cognition.

Neuropsychological analyses of parietal-lobe functions utilize tests that are sensitive to discrete parietal-lobe injuries. Such tests include the assessment of tactile functioning, visual guidance of movement, and cognitive functions such as spatial orientation, including both the copying of complex geometric figures and mental rotation.