The Parietal Lobes

Portraits: Varieties of Spatial Information

HP, a 28-year-old accountant, 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 HP’s problem with numbers became serious. In fact, he was no longer able to perform a simple subtraction, such as 30 minus 19, in which the solution requires ‘borrowing’ 10 when subtracting 9 from 0. At first, HP 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. Simple manipulations, such as playing with the Rubik’s cube puzzle pictured here, would have become difficult if not impossible for HP. He began confusing left and right and having difficulties reading. Some of the words appeared backward or upside down to him, and he could not make sense of them.

Finally, when HP 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, HP 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 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

HP’s symptoms described in the portrait at the beginning of this chapter are typical of left parietal injury and illustrate 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 region of the cerebral cortex lies between the frontal and the 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 lateral (Sylvian) fissure, dorsally by the cingulate gyri, and posteriorly by the parietal-occipital sulcus. The principal regions of the parietal lobe, mapped in Figure 14.1A and B, include the postcentral gyrus (Brodmann’s areas 1, 2, and 3), the superior parietal lobules (areas 5 and 7), the parietal operculum (area 41), the supramarginal gyrus (area 40), and the angular gyrus (area 39).

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 41; 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 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 10.18). Similarly, area PG in the monkey includes areas 7a, VIP, LIP, IPG, PP, MST, 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 (STS). (Recall from Chapters 10 and 13 that polymodal cells receive inputs from more than one sensory modality) Those in PG respond to both somatosensory and visual inputs, whereas those in the STS (the third visual pathway discussed in Chapter 13) respond to various combinations of auditory, visual, and somatosensory inputs.

The increase in the 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 STS) 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 lesions, which is indeed the case. Note, however, that PG is also larger on the left in the human
Connections of the Parietal Cortex

The anterior parietal cortex has rather straightforward connections, which are illustrated in Fillemann and van Essen’s hierarchy (see Figure 10.19). There are projections from the primary somatosensory cortex (area 3-1-2) to area PE (area 5), 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 (area 6). 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 14.3:

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 input from the primary somatosensory 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 (skin sensations), proprioceptive (internal stimuli), auditory, vestibular (balance), ocularmotor (eye movement), and circulatory (motivational?). MacDonald Critchley described area PG as the "parieto-temporo-occipital crossroads," which is apparent from the connectivity. Its function likely corresponds to this intermodal mixing. Area PG, which is part of the dorsal stream, 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 (except 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.

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. The anterior zone’s somatosensory functions were discussed in Chapter 8; we are concerned here mostly with the functions of the posterior parietal zone.

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 these movements is directed toward a different place and requires a different hand posture, 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 wine, for example.) 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 what one you want to
use and select the correct utensil. It would be inappropriate to try to eat your
peas by using 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, some sort of inter-
nal representation of the location of different objects around us seems obvious—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 ob-
servations of patients with parietal injury, it has been widely believed for more
than 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 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 bind-
ing problem discussed in Chapter 10.)
Unfortunately, very little evidence supports the existence of such a map in
the brain. Likely, rather than a single map, there are a series of representations
of space that vary in two ways. First, different representations are used for dif-
erent behavioral needs. Second, representations of space vary from simple
ones, which are applicable to the control of simple movements, to abstract
ones, which may represent information such as topographic knowledge. We
consider each of these aspects of brain maps in turn.

Uses of Spatial Information
David Milner and Melvin Goodale emphasize that spatial information about
the location of objects in the world is needed both to direct actions at those ob-
jects and to assign meaning and significance to them. In this sense, spatial in-
formation 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 vi-
sual 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 recogni-
tion and the other is for guiding movements. Spatial information can be thought
of in the same way.

Object Recognition
The spatial information needed to determine the relations between objects, in-
dependent 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 lo-
cation of an object and its local orientation and motion must be determined rel-
atively 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 char-
acteristics, 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
so much information may be counterproductive for any given system. In con-
trast with the viewer-centered system, the object-centered system must be con-
cerned 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 viewpoint positions. 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 cup.
The temporal lobe codes relational properties of objects. Part of this con-
trol is probably in the polyvocal region of the superior temporal sulcus, and
another part is in the hippocampal formation. We return to the role of the tem-
poral cortex in Chapter 15.

Guidance of Movement
The posterior parietal cortex has a role in the viewer-centered system. To ac-
commodate 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 examples are very different types of movements.

We have considered 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 (see Figure 14.3).
The role of the posterior parietal region in visuomotor guidance is confir-
med by the results of single-cell studies in the posterior parietal lobes of
monkeys. The activity of these neurons depends on the concurrent behavior of
an animal with respect to visual stimulation. In fact, most neurons in the pos-
terior parietal region are active both during sensory input and during move-
ment. 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 respond to
the structural features of the object, such as size and orientation, as well. That
is, the neurons are sensitive to the features of an object that determine the pos-
ture of the hand during manipulation.
A characteristic common to all the posterior parietal neurons is their re-
sponsiveness to movements of the eyes and to the location of the eyes 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 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 behaviorally relevant visual stimuli, such as a cue signaling the availability of a reward. This responsiveness has been interpreted to suggest that these cells are affected by the "motivational" characteristics of information.

John 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 an animal attends to a target or makes a movement toward it. These neurons are therefore well suited to transforming the necessary sensory information into commands for directing attention and guiding motor output.

Although the activity of single cells in the human posterior parietal region cannot be studied, event-related potentials (ERPs) in response to visual stimuli can be recorded (see Figure 6.8). 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. Per Roland also showed that, when subjects direct their attention to visual targets, blood flow increases preferentially in the posterior parietal region.

Taked 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 (much as in FPD, as is presented in the Portrait at the beginning of this chapter) 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 James Haxby and his colleagues found 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."

A difficulty with this interpretation, however, is that, when people solve spatial tasks, they move their eyes. The increased PET activation, therefore, may not be due to the movement of the eyes rather than to processing the location of the target in space. Indeed, 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 problems for PET studies: the construction of experimental designs in brain-imaging studies presents a practical difficulty.

**Sensory-motor Transformation**

When we move toward objects, we must integrate the movements of different body parts (eyes, body, arm, and so forth) with the sensory feedback of what movements are actually being made (the effector copy) and the plans to make the movements. As we move, the locations of our body parts change and must constantly be updated so that we can make future movements smoothly. These neural calculations are called *sensory-motor transformation*. Cells in the posterior parietal cortex produce both the movement-related and the sensory-related signals to make these transformations.

Another aspect of sensory-motor transformation is movement planning. Although less is known about the role of the parietal cortex in planning, Richard Andersen and his colleagues have shown that area PRP is active when a subject is preparing and executing a movement (see Figure 14.2). Importantly, PRP is coding not the limb variables required to make the movement but rather the desired goal of the movement. Thus, the goal of grasping a cup, for example, is coded rather than the details of the movements toward the cup.

Andersen's group devised novel experiments with monkeys in which they decoded from neural activity the animals' intentions to reach to position a cursor on a screen, as illustrated in Figure 14.4. Monkeys were trained to make a series of reaches to touch different locations on a computer screen (Figure 14.4A). The cell activity was analyzed to determine which activity was associated with movement to each location. The monkeys were instructed with a briefly flashed cue to plan to execute a reach to different locations but without making a movement.

The cellular activity was compared with activity associated with actual movements to the requested target, and, if it was the same as in an actual movement, the monkeys were rewarded with a drop of juice in the mouth and a visual feedback showing the correct location (Figure 14.4B). The authors had to use this approach because they could not simply say to the monkeys "think about reaching to the target." Rather, they had to devise a way for the monkeys to show that they were thinking about reaching to targets.

This type of study is potentially very important, because it means that paralyzed people can use mental activity to move prosthetics. In principle, an array of electrodes could be implanted over the PRP, and the recorded activity could be used to move the mechanical devices. The implications of such advances go well beyond limb movements. Implants over speech areas might allow a verbal readout of thoughts, thus bypassing cumbersome letter boards and spelling systems.
programs. Similarly, one could ask patients questions and have them move a cursor to identify the correct answers, thus gaining access to a wide variety of their thoughts and even emotions.

**Spatial Navigation**
When we travel in the real world, we can take the correct route subconsciously, making the correct turns at choice points until we reach our destination. To do so, we must have some type of "cognitive spatial map" in our brains, as well as a mental map of what we do at each spatial location. The internal map is sometimes referred to as "route knowledge."

This route knowledge is unlikely to be located in a single place in the brain. Findings from both lesion and neuroimaging studies in humans suggest that the medial prefrontal cortex (MPC), which includes the parietal cortex ventral to the PRR in Figure 14.2B as well as the adjacent posterior cingulate cortex (Figure 21.2), takes part. Neurons in the dorsal visual stream could be expected to participate in route knowledge, insofar as we must make specific visually guided movements at specific locations in our journey. To explore this idea, Nobuya Sato and colleagues trained monkeys to perform a navigation task in a virtual environment.

Three-quarters of the cells in the MPR showed responses associated with a specific movement at a specific location. The same movement in a different location did not activate the cells. Thus, like the cells in PRR that control the planning of limb movements to locations, the cells in MPR control only body movements to specific locations. When the authors inactivated the MPR pharmacologically, the monkeys became lost and failed to navigate correctly. Thus, the monkeys acted like human patients with medial parietal lesions who often become lost. We return to this problem in Chapter 21.

**The Complexity of Spatial Information**
The first aspect of our theory of parietal-lobe function considers the uses of spatial information for object recognition and for guiding movement. The second aspect of spatial representation is complexity. The control of limb or eye movement 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 with posterior parietal lesions, such as H.P., are impaired at distinguishing left from right.

Other spatial relations are even more complex. For example, you can visualize objects and manipulate these mental images spatially, as described in the Snapshot. Patients with posterior parietal lesions are impaired at mental manipulations. The ability to manipulate objects mentally is likely 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.

**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 by H.P.

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**SNAPSHOT**

**White-Matter Organization and Spatial Cognition**

The ability to imagine object transformations is a fundamental aspect of spatial cognition. Everyday activities, such as constructional tasks (say, putting a bookshelf together), require an ability to manipulate pieces both mentally and physically. Studies of lesion patients and noninvasive imaging reveal that mental transformations such as object rotation are carried out by the posterior parietal cortex. Humans vary substantially in their capacity to perform mental object transformations, however, and there is a significant sex difference favoring males in such tasks (see Chapter 12). This intersubject variation could result from differences in cognitive strategy or in the ability to maintain a representation in memory as it is manipulated, but the variation could also be related to differences in the underlying neuropsychiatry. Thomas Waller and his colleagues hypothesized that the anatomical difference could be in white-matter organization, which would correspond to connectivity of the posterior parietal region.

To determine the role of white-matter differences in mental rotation, the researchers gave male subjects the difficult mental rotation task illustrated in Figure A. As expected, they found considerable intersubject variability, despite controlling for spatial short-term memory ability. They used MRI to characterize the white-matter organization in the posterior parietal cortex. As shown in Figure B, there was a tight relation between mental spatial rotation proficiency and white-matter organization near the anterior part of the intraparietal sulcus.

This anatomical measure provides an indirect measure of brain organization because it includes a variety of factors such as myelination, axon diameter and density, and fiber crossing. Nonetheless, the results support the general idea that the details of neuroanatomical organization are related to individual differences in cognitive abilities.

Whether such differences are purely genetic or are influenced by experience remains to be determined. Similarly, because the investigators studied only males, we do not yet know if sex differences in mental rotation are related to differences in white-matter organization in the posterior parietal cortex.


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Alexander Luria proposed that mathematics and arithmetic have a quasi-spatial nature analogous to the mental manipulation of concrete shapes but entail 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 J.P. experience acalculia (an inability to do arithmetic) stems from the spatial nature of the task. Indeed, if parietal-lobe patients are given simple problems such as 6 minus 4, they usually solve them because the spatial demands are few. Even patients have little problem. When more-complex manipulations, such as lining, are made, however, as in 983 minus 24, the patients' arithmetic abilities break down. Thus, arithmetic operations may depend on the sensory tissue at the left temporoparietal junction.

Language has many of the same demands as arithmetic. The words “gap” and “par” 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 quasi-spatial. Patients such as J.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 polymodal 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 gyri (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 Josefine 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 sensitvity). When described in Chapter 8), and at localizing points of touch on the palm 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.

**Somatosensory Disorders**

The presence of somatosensory thresholds does not preclude the possibility of other types of somatosensory abnormalities. First, there is astereognosis (from the Greek terms, meaning “solid”), which is the inability to recognize the nature of an object by touch. This disturbance can be demonstrated in tests of tactile perception of object qualities, illustrated in Figure 14.5. 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 somatosensory disorder, simultaneous extinction, can be demonstrated only by a special testing procedure. 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 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 14.6. 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 14.5](image-url)

*Tests for Tactile Perception of Objects* Somatosensory abnormalities such as astereognosis can be identified by such tests. (After Teuber, 1978.)

![Figure 14.6](image-url)

*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 Paillal 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 area 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: acognosis (see the preceding discussion of somatosensory disorders) and anosagnosia—the loss of knowledge or sense of one’s own body and bodily condition. Although acognosis is essentially a disorder of tactile perception (see Figure 14.5), it is included here because it is often described clinically simply as agnosia.

Anosagnosia is one of the most curious of all agnosias. It is an almost unbelievable syndrome—until you actually observe it. The varieties of anosagnosia include anosognosia, an unawareness or denial of illness; anosodiaphoria, indifference to illness; autognosia, 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.

Anosagnosia 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 autognosias, which usually result from lesions of the left parietal cortex. The most common autognosia is finger agnosia, a condition in which a person is unable 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, 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, Rezső Balint described a patient whose bilateral parietal lesion was associated with rather peculiar visual symptoms. The patient had full visual fields and could recognize, see, 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, described in Chapter 13, 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 (see Chapter 13). 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 Antonio Damasio and Arthur 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 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 tactually 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
McDonald 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 McGuie and Zangwill, published in 1966, 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:

- He 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 (anosognosia). 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 freely with either hand, at copying drawings, or at 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, patients showing neglect commonly fail to complete the left side of the drawing, as illustrated in Figure 14.7.

- Mr. P. had a topographic 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.

Contralateral neglect as observed in Mr. P. is one of the most fascinating symptoms of brain dysfunction. Neglect occurs in visual, auditory, and somesthetic stimulation on the side of the body or space or both body and space opposite the lesion. Neglect may be accompanied by denial of the deficit. Recovery passes through two stages. *Aloesthesia* is characterized by a person's beginning to respond to stimuli on the neglected side as if the stimuli were on the undamaged 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 14.6). 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 14.8A is a composite drawing of the region damaged (as inferred from brain scans) in 13 patients with neglect as described by Kenneth Heilman and Robert Watson. A recent review by Argye Hillis concludes that both the right intraparietal sulcus (roughly dividing PF and PF') and the right angular gyrus are necessary for contralateral neglect. Furthermore, Neil Muggleton and his colleagues used transcranial magnetic stimulation over these regions to induce neglect in intact subjects.

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 arise? 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 *morphopsathyenia* and its disruption *anopsathyenia*.

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.

Crichtley 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. Heilman and Watson elaborated on this suggestion. They proposed that neglect is manifested by a defect in orienting to stimuli; the defect results from the disruption of a system whose function is to "around" 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 14.9). Warrington concluded that the deficit is not in forming a gestalt, or concept—in this case, 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 cortical neglect (see Figure 14.8B).

The Gerstmann Syndrome and Other Left Parietal Syndromes
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. The Gerstmann syndrome is a doubleblind 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 August 24, 1973, S.S., an 11-year-old boy, suddenly had a seizure that 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 September 16, 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).
- Agraphia. 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).
- Agraphia. 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 geniculo-cortical 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 exhibited by S.S. resemble those of other patients whom we have seen with left parietal lesions, including ELP, 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 the 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. Doreen Kimura showed that the deficits in such patients can be quantified by asking the patients to copy a series of arm movements such as those illustrated in Figure 14.10A. Patients with left-parietal-lobe lesions are greatly 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 14.10B). 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 parieto-frontal connections that control movement. Vernon Mountcastle proposed that the posterior parietal cortex receives afferent signals not only of the tactile and visual representations 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. 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, disturbances in drawing appear to differ, depending on whether the lesion is in the right or the left hemisphere.

For example, Kitamura and Faust asked a large sample of patients to draw a house and a man. Apractic 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, spoked, chain, and so on). In the absence of language, we would expect such people to draw less-complete bicycles. Further, if patients are apractic, 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, all of which cannot possibly 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 talk with an old friend in a department store. Several other people may be nearby, 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.

Michael 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 we shift from one food to another.

An aspect of disengagement is that we must reset our visuomotor guidance systems to form the appropriate movements for the next target. We can extend this idea to the 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 12.1 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 PG region 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, Michael Corballis suggested that mental rotation requires two different operations: (1) the mental imaging of the stimulus and (2) the manipulation of the image. Fred Newcombe and Graham Ratcliffe suggested that the left-hemisphere deficit may result from an inability to generate an appropriate mental image. As discussed in Chapter 13, 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 topographic 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 in one's environment. Not surprisingly, such deficits are likely to be associated with other visual deficits (such as contralateral neglect or visual agnosia), but specific disorders of topographic orientation have been described for some patients.

Emilio de Renzi concluded that injury to the right posterior hemisphere is a prerequisite for such disorders. Newcombe and Ratcliffe noted that such disorientations are often associated with injury to the right posterior cerebral artery and are thus likely to include the right occipitotemporal and right hippocampal region. 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 is 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 constructed apraxia and disorders of spatial cognition are poorly lateralized. Many theories of hemispheric asymmetry, discussed in Chapter 11, do not predict such ambiguity 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, where we note that many problems can be solved by using either a verbal cognitive mode or a spatial nonverbal cognitive mode. Generic, mnestic, and environmental factors may predispose people to use different cognitive modes. For example, you might solve a complex spatial problem, such as reading an upside-down map, 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 disfigure 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.

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

<table>
<thead>
<tr>
<th>PERCENTAGE OF SUBJECTS WITH DEFICIT*</th>
<th>Left (%)</th>
<th>Right (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unilateral neglect</td>
<td>13</td>
<td>67</td>
</tr>
<tr>
<td>Dressing disability</td>
<td>13</td>
<td>67</td>
</tr>
<tr>
<td>Cube counting</td>
<td>0</td>
<td>86</td>
</tr>
<tr>
<td>Paper cutting</td>
<td>0</td>
<td>90</td>
</tr>
<tr>
<td>Tegaphralic loss</td>
<td>13</td>
<td>56</td>
</tr>
<tr>
<td>Right-left confusion</td>
<td>62</td>
<td>0</td>
</tr>
<tr>
<td>Wegel's Sarting Test</td>
<td>83</td>
<td>6</td>
</tr>
</tbody>
</table>

*Note the small but significant overlap in symptoms of left and right lesions.

Source: Based on data presented by McFie and Zangwill, 1961.

## 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 region produces most of the other disorders.

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. Additionally, 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.

### Table 14.2 Summary of major symptoms of parietal-lobe damage

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Most probable lesion site</th>
<th>Basic reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deficits of tactile function</td>
<td>Areas 1, 2, 3</td>
<td>Semmes et al., 1960</td>
</tr>
<tr>
<td>Gait apraxia</td>
<td>Area PE</td>
<td>McFie and Albert, 1978</td>
</tr>
<tr>
<td>Spatial neglect</td>
<td>Areas PE, PF</td>
<td>Tyler, 1968</td>
</tr>
<tr>
<td>Apraxia of objects</td>
<td>Areas PE, PG</td>
<td>Damasio and Benton, 1979</td>
</tr>
<tr>
<td>Apraxia of speech</td>
<td>Areas PE, PG, left</td>
<td>Heilman and Rothi, 1993</td>
</tr>
<tr>
<td>Constructual apraxia</td>
<td>Area PG</td>
<td>Kimura, 1989</td>
</tr>
<tr>
<td>Apraxia of hands</td>
<td>Area PG</td>
<td>Benton, 1990</td>
</tr>
<tr>
<td>Apraxia of trunk</td>
<td>Area PG, STS*</td>
<td>Levin et al., 1993</td>
</tr>
<tr>
<td>Apraxia of arm</td>
<td>Area PG, STS</td>
<td>Butters and Brody, 1968</td>
</tr>
<tr>
<td>Apraxia of body image</td>
<td>Area PG, STS</td>
<td>Heilman et al., 1993</td>
</tr>
<tr>
<td>Apraxia of left-right confusion</td>
<td>Area PG, right</td>
<td>Warrington and Taylor, 1973</td>
</tr>
<tr>
<td>Apraxia of spatial ability</td>
<td>Area PG, left</td>
<td>Benton and Shinn, 1993</td>
</tr>
<tr>
<td>Apraxia of drawing</td>
<td>Area PG, right</td>
<td>Semmes et al., 1960</td>
</tr>
<tr>
<td>Apraxia of drawing</td>
<td>Area PG</td>
<td>Newcombe and Ratcliff, 1990</td>
</tr>
</tbody>
</table>

STs, superior temporal sulci.

### 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. Although these tests, summarized in Table 14.3, 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

<table>
<thead>
<tr>
<th>Function</th>
<th>Test</th>
<th>Basic reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatosensory threshold</td>
<td>Two-point discrimination</td>
<td>Corin et al., 1970</td>
</tr>
<tr>
<td>Tactile form recognition</td>
<td>Seguin-Goldstein Form Board</td>
<td>Teuber and Wein, 1954</td>
</tr>
<tr>
<td></td>
<td>Board (tactile patterns)</td>
<td>Benton et al., 1983</td>
</tr>
<tr>
<td></td>
<td>Line bisection</td>
<td>Schaltenbrand et al., 1950</td>
</tr>
<tr>
<td></td>
<td>Gollin Incomplete Figures</td>
<td>Warrington and Robins, 1979</td>
</tr>
<tr>
<td></td>
<td>Mooney Closure</td>
<td>Milner, 1980</td>
</tr>
<tr>
<td></td>
<td>Right-left differentiation</td>
<td>Benton et al., 1983</td>
</tr>
<tr>
<td>Contralateral neglect</td>
<td>Token</td>
<td>de Renzi and Tagliani, 1978</td>
</tr>
<tr>
<td>Visual perception</td>
<td>Token</td>
<td>Kimura, 1977</td>
</tr>
<tr>
<td>Spatial relations</td>
<td>Token</td>
<td>Kimura Box</td>
</tr>
<tr>
<td>Language</td>
<td>Token</td>
<td></td>
</tr>
<tr>
<td>Speech comprehension</td>
<td>Token</td>
<td></td>
</tr>
<tr>
<td>Reading comprehension</td>
<td>Token</td>
<td></td>
</tr>
<tr>
<td>Apraxia</td>
<td>Kimura Box</td>
<td></td>
</tr>
</tbody>
</table>

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

Tactile Form Recognition

In the Seguin-Goldstein Form Board test, a 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 PFE, 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 for contralateral neglect have been devised, but we favor the line-bisection test by Thomas Schenkenberg and his 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 tests, a series of incomplete representations of faces or objects are 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 that are to be carried out (for example, "Touch your right ear with your left hand"). 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 a 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 for apraxia analogous to the Token Test for aphasia. However, the Kimura Box Test (Figure 14.11) is probably the best test currently available. Subjects are 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. Apraxics perform very poorly on this test, and many of them appear unable to perform this very simple series of movements even with extensive practice.

Summary

Anatomy of the Parietal Lobes

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 areas controlling not only the actual manipulation of objects but also the mental manipulation of objects.

Movement of 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.

A Theory of Parietal-Lobe Function

The hand can be considered the organ of the parietal lobe; so the parietal lobe has a primary function of guiding limb movements to place the hand in specific
spatial locations. The inferior parietal region also 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.

**Somatosensory Symptoms of Parietal-Lobe Lesions**

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.

**Symptoms of Posterior Parietal Damage**

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 apraxia may also 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 conical neglect and various deficits in spatial cognition.

**Major Symptoms and Their Assessment**

Neuropsychological analyses of parietal-lobe functional tests 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.

**References**


