The posterior parietal cortex has long been implicated in spatial aspects of perception and behavior in primates. Deficits after lesion to this area in humans indicate that this cortical region is essential for accurate visually guided motor activity, for appreciation of the orientation of self within the extrapersonal space, for normal spatial perception, and for the operation of spatial aspects of attention. Lesions to homologous areas in monkeys produce a similar set of signs and symptoms to those seen in humans, and the available psychophysical literature suggests that visual-spatial processing mechanisms are likely to be similar in the two species. Thus, neurophysiologists use the monkey as an animal model for understanding cortical spatial processes in humans.

An important issue in cortical physiology is how space might be represented in regions of the brain, such as the inferior parietal lobule, which are important for spatial aspects of behavior. Visual information is gathered in retinal coordinates as a result of the focusing of visual images on the retinæ. The contralateral visual field is represented in several different areas of the central nervous system through an orderly retinotopic mapping of projections from retina to brain and between visual areas in the brain. However, a retinotopic coordinate frame for visual space is not useful for many aspects of motor behavior, particularly rapid eye and limb movements made without visual feedback. Such movements are made to
locations in space referenced to the body rather than the retina. At some point in the nervous system visual information must be converted into an ego-centered coordinate frame using eye and head position signals so that these ballistic aspects of motor activity can be made with accuracy within the extrapersonal space. The perceptual apparatus also appears to transform visual inputs to non-retinal spatial representations since our perceptions of visual space remain quite stable regardless of the fact that we make on the average three eye movements a second. Many of the defects that are seen with lesions to the inferior parietal lobule could be accounted for by a disruption in the transformation of retinal coordinate frames to spatial coordinate frames.

In this chapter I will first review some of the spatial defects that are found in humans with lesions to the inferior parietal lobule. This review is meant to be selective and indicate some of the features that neurophysiologists use as clues to designing experiments in animals aimed at increasing our understanding of the functions of the human cortex. Next I will briefly review the defects caused by lesions to this area in the monkey. We will then cover the anatomy of the area in man and monkey with particular emphasis on its relation to the visual system. Last, we will examine what has been learned of the role of this area in spatial processing from single unit recording experiments in monkeys.

**HUMAN LESIONS**

Damage to the inferior parietal lobule in humans produces visual defects that can be divided into two general categories: those that affect visual attention and those that affect spatial perception. Side-ness plays a role in the expression of this disorder in humans with right hemisphere lesions in right-handed individuals often causing more frequent and severe parietal lobe syndromes (Piercy, Hecaen & Ajuriaguerra, 1960; McFie & Zangwill, 1960; Critchley, 1953). However, some students of the parietal lobe believe that left-sided parietal lesions in the absence of aphasia and the agnosias can produce a syndrome similar to that seen with right-sided lesions (Denny-Brown & Banker, 1954; Paterson & Zangwill, 1944). Unilateral lesions can produce disturbances that are only contralateral, or particularly with right hemisphere lesions, bilateral.

*Attentional defects.* Patients with posterior parietal lesions can exhibit visual inattention or "neglect" which is often confined to the
hemifield contralateral to the lesion. A vivid example of a contralateral neglect is shown in Figure 3.1. The patient was a painter who was asked to paint self-portraits at various times over several months during recovery from a stroke. As illustrated in Figure 3.1, in the early portraits he completely ignored the contralateral side of his face in the paintings and it was only after several months of recovery that the portraits appeared normal.

The neglect can also extend to the body with patients exhibiting aspontaneity of the contralateral body half, difficulty in dressing and lack of grooming of the contralateral side. In severe cases patients will deny that the contralateral body half is theirs or will have distortions of the body image such as perceived supernumerary limbs (Denny-Brown & Banker, 1954; Brain, 1941; Hecaen, Penfield, Bertrand & Malmo, 1956; Hecaen & Ajuriaguerra, 1954; De Renzi, Faglioni & Scotti, 1970; Critchley, 1953). It has been argued that the neglect of the body and neglect in the visual hemifield are signs of a common origin (Brain, 1941; Hecaen et al., 1956).

Spatial defects. Spatial defects with posterior parietal lesions are wide ranging and include visual disorientation, defects in visual localization, constructional apraxia, disturbances in topographical relationships, and loss of spatial memories.

The defective localization of objects in space in the absence of visual object agnosia has been termed visual disorientation. This deficit can occur with normal visual acuity and without visual field defects (Brain, 1941; Semmes, Weinstein, Ghent & Teuber, 1963). Patients report that their environments appear “confused” or “jumbled,” and these spatial defects, when severe, can be more debilitating than blindness (Critchley, 1953). Such patients cannot judge the position of two objects in relation to one another (Holmes, 1919) and often are unable to notice two objects simultaneously (Holmes, 1919; Ettlinger, Warrington & Zangwill, 1957). Reports such as “when I look at one thing, the rest vanish” are common (Kinsbourne & Warrington, 1962). Patients are unable to attend to backgrounds (Semmes et al., 1963) or to apply uniform frames of reference (Paterson & Zangwill, 1944). They do not appreciate a figure as a spatially organized unit, and attention to any one part of it destroys the effect of the whole (Paterson & Zangwill, 1944).

Errors in visual localization following posterior parietal lesions are indicated by mistakes in pointing to visual targets (Paterson & Zangwill, 1944; Ratcliff & Davies-Jones, 1972; Holmes, 1919); these defects are strictly visuospatial and are not defects in reaching (Ratcliff & Davies-Jones, 1972). These deficits are generally confined
Figure 3.1. Self-portraits by German artist Anton Raderscheidt. These portraits were made at different times after a stroke that damaged the right parietal cortex. The portraits were made two months (upper left), three and a half months (upper right), six months (lower left) and nine months (lower right) after the lesion. Note that in the earlier portraits the side of the face contralateral to the lesion is profoundly neglected. The figure is reprinted from Wurtz, Goldberg & Robinson (1982); the self-portraits were originally published by Jung (1974).
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To the contralateral space for reaching with either limb. Other spatial defects occur in distance estimation (Paterson & Zangwill, 1944; Holmes, 1919), appreciation of the relative lengths and sizes of objects (Holmes, 1919), and the occasional loss of stereopsis (Carmon & Bechtoldt, 1969; Critchley, 1953; Riddoch, 1917).

Constructional apraxia refers to an inability to reproduce spatial relations in a model, as for instance, in drawing (Benson & Barton, 1970; Piercy et al., 1960; Mcfie & Zangwill, 1960; Paterson & Zangwill, 1944; Hecaen et al., 1956; Hecaen & Ajuriaguerra, 1954). Constructional defects after posterior parietal cortex lesions are often global and include abnormal representations of perspective and depth, abnormalities of relative size and articulation, and a piece-meal approach whereby the patient wanders through a drawing going from detail to detail in no coherent spatial frame. Some authors consider constructional apraxia to be a secondary result of a visuospatial defect (Paterson & Zangwill, 1944; Butters & Barton, 1970; Hecaen et al., 1956). Figure 3.2 shows an example of constructional apraxia in which a patient has been asked to model with blocks the structure in the left panel with the poor results pictured in the right panel.

Following posterior parietal lobe lesions disturbances of topographical relationships, as in route-finding (Teuber, 1963; Brain, 1941; Semmes et al., 1963; Hecaen et al., 1956; Holmes, 1919), are also found. Investigators believe that visuospatial defects contribute to these signs although defects in spatial memories also appear to play a role. The first account in the literature of a topographic memory deficit comes from Charcot's description in 1883 of a patient who, in a town previously well known to the patient, could no longer recognize even commonplace landmarks. The patient claimed he felt as if he were at sea in what was at one time a familiar world. Wilbrand (1887) also described a patient who could not call up remem-

Figure 3.2. Constructional apraxia. A patient with a left fronto-parietal metastatic tumor was asked to model the block construction on the left. The patient's copy on the right indicates a poor performance on this three-dimensional task. Figure from Critchley, 1953.
bered visual images of a topological or geographical nature. Those few topographic images the patient could generate from memory were profoundly spatially distorted. For instance, the patient imagined that the street lay just outside her parlor when actually a bedroom intervened and believed that articles of her furniture were in the street rather than in her home. Both of these patients had other visual memory defects as well (Charcot-Wilbrand syndrome); however, since that time many cases have been described in which the memory deficit is restricted to topographic-spatial memory (reviewed in De Renzi, 1982). Moreover, these memory deficits have been distinguished from perceptual deficits (Critchley, 1953).

Certain aspects of the hemi-neglect commonly found with parietal lesions seem to derive from the defect in spatial memory. In an illuminating experiment with parietal patients exhibiting contralateral neglect, Bisiach and Luzzatti (1978) asked these patients to describe from memory, landmarks bordering a square familiar to the patients, the Piazza del Duomo in Milan (Figure 3.3). They were first asked to imagine that they were standing on the steps of the cathedral at one end of the square; in this instance they described mostly those establishments on the side of the street ipsilateral to the lesion. Next they were asked to imagine that they were on the

Figure 3.3. A map of the Piazza del Duomo in Milan. Contralateral neglect is demonstrated in patient I.G. who, after suffering a right hemisphere stroke, was asked to recall from memory landmarks on the square from the two perspectives indicated in the figure (A and B). The numbered dark circles indicate the positions of landmarks she recalled from perspective A and the numbered dark squares the landmarks recalled from perspective B. The figure is modified from Bisiach and Luzzatti (1978).
other side of the square facing the cathedral; they then described mostly establishments on the other side of the street. In interpreting these results the investigators reasoned that spatial memories are stored bilaterally with half of space represented contralaterally in each hemisphere.

Parietal lesions not only eliminate old and deeply rooted spatial memories as in the previous examples, but also interfere with the formation of new spatial memories. In clinical tests it has been found that such patients require significantly larger numbers of trials to learn spatial tasks such as memorizing a path through a maze or tapping a sequence of blocks based on their spatial position (De Renzi, Faglioni & Previdi, 1977; De Renzi, Faglioni & Villa, 1977).

Since parietal lobe lesions affect all spatial memories including both old memories recorded prior to the lesion and the formation of new memories after the lesion, it is likely that the posterior parietal area either contains the topological memory traces or is necessary for their recall. The dual nature of this amnesia distinguishes the posterior parietal area from certain other classical memory regions such as the hippocampal formation, which appear to be involved in the consolidation of memory since lesions to these regions primarily affect the acquisition of new memories. These observations are consistent with Mishkin's (1982) proposal that memories are stored in association cortex by way of a consolidating action of hippocampal and related structures on these cortices.

MONKEY LESION EXPERIMENTS

Posterior parietal lesions in monkeys produce many visual-spatial deficits similar to those recorded in humans. Unilateral lesions or cooling of posterior parietal cortex produce animals that exhibit contralateral disuse of the body, neglect in the contralateral visual field, and contralateral extinction for visual stimuli.

A most extensively studied defect is that of impaired spatial localization as indicated by inaccuracy in reaching with the contralateral limb under visual guidance following unilateral lesions (LaMotte & Acuna, 1978; Ettlinger & Kalsbeck, 1962; Hartje & Ettlinger, 1974; Moffet, Ettlinger, Morton & Piercy, 1967; Ratcliff, Ridley & Ettlinger, 1977; Faugier-Grimaud, Frenois & Stein, 1978). This defect is confined to the contralateral limb for reaching in either hemifield. The ipsilateral limb can reach accurately to objects in either hemifield. Thus, the defect manifests itself as if spatial vision had been severed from the one limb but not the other.
Bilateral ablations of posterior parietal cortex produce severe deficits in visual-spatial orientation. Such animals cannot discriminate the spatial relation of two or more objects in the visual field (Mendoza & Thomas, 1975; Brody & Pribram, 1978; Pohl, 1973; Ungerleider & Brody, 1977). Monkeys with bilateral posterior parietal lesions also have topographic deficits as measured by their performance on route-following tasks (Petrides & Iversen, 1979) and exhibit visual extinction (Lynch & McLaren, 1984). It can be seen, then, that the posterior parietal cortex of the monkey, as in man, is important in visual-spatial perception.

ANATOMY

The cerebral hemispheres are divided by large sulci into six major lobes. These lobes can be further subdivided into cortical fields which are areas of the cortex that differ in their connections, internal structure, and functional roles. Many cortical fields can be further subdivided into repeating functional modules; these modules are often referred to as cortical columns.

The anterior aspect of the parietal lobe contains Brodmann's areas 3, 1, and 2. These areas are often collectively referred to as primary somatosensory cortex, although they are functionally and anatomically distinct cortical areas.

The posterior parietal cortex is subdivided into superior and inferior parietal lobules. The superior parietal lobule consists of somatosensory association cortex and the inferior parietal lobule consists of high order visual and somatosensory cortical fields. Brodmann's area 5 encompasses the superior parietal lobule and his area 7 the inferior parietal lobule (see Figure 3.4a). Area 7 was further subdivided into a medial (7a) and lateral (7b) field by Vogt and Vogt (1919). Further confirmation for these cytoarchitectural subdivisions came from von Bonin and Bailey (1947) whose PE cortical area roughly corresponds to area 5 and whose PG and PF correspond to 7a and 7b (Figure 3.4b).

The exact homologies of these cortical areas in the monkey with those in the human are unclear. Brodmann believed that the cellular architecture of area 7 of the inferior parietal lobule of the monkey was identical to the posterior-most aspect of the human superior parietal lobule with area 5 in man located more anteriorly in the superior parietal lobule (Figure 3.4c). He recognized two cortical areas in the human inferior parietal lobule, corresponding to the angular (area 39) and marginal gyri (area 40), which he believed had
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Figure 3.4. Cytoarchitectonics of the posterior parietal cortex. Lateral views of monkey and human cerebral cortices showing cytoarchitectural parcellations made by different anatomists of the posterior parietal cortex.

(a) Brodmann's (1905) parcellation of the monkey cortex (Cercopithecus).
(b) von Bonin and Bailey's (1947) parcellation of the monkey cortex (Macaca mulatta).
(c) Brodmann's (1907) classification of the human cortex.
(d) von Economo's (1929) map of the human posterior parietal cortex.

no homologies in the old world monkey. Von Bonin and Bailey criticized Brodmann's work in the monkey and asserted that their areas PG and PF of the monkey inferior parietal lobule were homologous to area PG and PF of the human (Von Economo, 1929) which encompass the inferior parietal lobule of man (Figure 3.4d). Support for von Bonin and Bailey's view comes from the observation that lesions of the inferior parietal lobule of monkeys produce visual-spatial defects and thus are more like the inferior parietal lesions in humans which also produce visual-spatial defects and less like superior parietal lesions in humans which tend to result in somatic disturbances.

In the past 15 years a great deal has been learned from anatomical and physiological experiments about the structure and function of the posterior parietal cortex in monkeys. I will emphasize the inferior parietal lobule and particularly the visual cortical fields within this brain area since the most progress has been made in
these areas. The use of visual rather than somatosensory paradigms has proven to be most productive since visual stimuli are more easily controlled and a good deal more is known about the earlier stages of processing in the visual system.

The inferior parietal lobule receives visual inputs from visual association cortical fields (so called extrastriate cortical fields to distinguish them from striate or primary visual cortex) and somatosensory inputs from somatosensory association cortex. Its major thalamic input is from the pulvinar. The inferior parietal lobule is part of a net of reciprocal interconnections between cortical fields concerned with some of the highest order cortical functions including the prefrontal cortex, cingulate gyrus, parahippocampal gyrus, and cortex of the superior temporal sulcus. Recent studies reveal that the inferior parietal lobule is composed of not one or even two but probably several cortical fields that differ in their anatomical connections and in their functional roles in spatial processing.

**Visual Pathways**

The visual inputs to the inferior parietal lobule are derived primarily from other extrastriate visual cortical fields. Until recently the exact sources of these visual inputs were unknown. It is now clear that the inferior parietal lobule receives visual information from several converging cortical sources including visual areas in the prelunate gyrus (V4 and the dorsal prelunate area; Seltzer & Pandya, 1980; Andersen, Asanuma & Cowan 1985, Andersen, Siegel, Essick & Asanuma, 1985), the parieto-occipital visual area (Colby, Gattass, Olson & Gross, 1983; Andersen, Siegel, Essick & Asanuma, 1985), cortical areas within the superior temporal sulcus and visually responsive areas within the occipital-temporal sulcus (Mesulam, Van Hoesen, Pandya & Geschwind, 1977; Desimone & Gross, 1979; Andersen, Siegel, Essick & Asanuma, 1985). None of these areas receive direct projections from primary visual cortex (V1) but some of them do receive inputs from visual areas that receive V1 projections (Andersen, Siegel, Essick & Asanuma, 1985; Colby et al., 1983; Maunsell & Van Essen, 1983). Thus, although the inferior parietal lobule receives projections from several extrastriate visual areas, all of these parallel visual pathways are at least three steps removed from the primary visual cortex. The several visual projections have different patterns of termination within the inferior parietal lobule with the different cortical fields receiving different proportions of input from these pathways (Andersen, Siegel, Essick & Asanuma, 1985).
Ascending Thalamic Inputs

The major source of thalamic projection to the inferior parietal lobule is from the medial, lateral and oral divisions of the pulvinar (Trojanowski & Jacobson, 1976; Kasdon & Jacobson, 1978; Asanuma, Andersen & Cowan, 1982; Asanuma, Andersen & Cowan, 1985). These areas in turn receive ascending projections from the deep oculomotor layers of the superior colliculus and from the pretectum (Benevento & Standage, 1983). The areas of the pulvinar that project to the inferior parietal lobule receive feedback corticothalamic projections from the same cortical areas to which they project (Asanuma et al., 1985). The areas of the pulvinar that project to the visually responsive cortical fields of the inferior parietal lobule contain light-sensitive neurons (neurons that respond to visual stimuli) with large receptive fields and no clear retinotopic organization (Benevento & Miller, 1981; Bender, 1981; Petersen, Robinson & Keys, 1982). Many neurons in these areas of the pulvinar also have activity related to saccades (ballistic eye movements) which likely obtains from the projection of the deep layers of the superior colliculus (Perryman, Lindsley & Lindsley, 1980; Petersen et al., 1982). The visual inputs of these neurons probably are largely of cortical origin and are derived from descending corticothalamic projections (Bender, 1983; Ogren & Hendrickson, 1979). The oral pulvinar, which projects to area 7b, contains neurons with predominantly somatosensory properties (Acuna, Gonzalez & Dominguez, 1983).

Area 7a receives its pulvinar input almost exclusively from three disk-like arrays of neurons in the medial pulvinar (Asanuma et al. 1982, 1985). There is a topographic relationship in the reciprocal connections between 7a and these disks indicating that area 7a is represented three times in the medial pulvinar (Asanuma et al., 1982, 1985). The medial pulvinar also projects to other higher cortical areas such as the prefrontal cortex and cortex of the superior temporal sulcus in the form of disklike aggregates of neurons (Asanuma et al., 1982, 1985; Trojanowski & Jacobson, 1974; Siqueira, 1965, 1971). In double label experiments in which the arrays of neurons projecting to two different locations in the brain can be visualized on single sections of the brain, it has been found that for the prefrontal cortex and area 7a the disks in the pulvinar are partially overlapping with the frontal projecting disks located more medially (Asanuma et al., 1982, 1985). The cells in the overlapping regions of the projections are intermingled but single neurons almost never send axons to both cortical areas.
It is likely that all of the higher cortical areas that are recipro-
cally interconnected with 7a also receive projections from disks in
the medial pulvinar. These pulvinar projections terminate most
heavily in the lower aspect of cortical layer three (Trojanowski &
Jacobson, 1976). Interestingly this cortical layer is the major source
of cortico-cortical connections (Andersen, Essick & Siegel, 1984).
Thus the medial pulvinar may act as a regulator of transmission
between these cortical regions involved in the highest aspects of
cortical function. The partial overlapping of pulvinar projection
disks and the multiple representations of cortical areas across
several disks is a revealing structure in terms of the regulation of
cortico-cortical communication. This structure enables any locus in
the medial pulvinar to connect, in an anatomically precise fashion,
segregated areas within several widely separated cortical areas.
Therefore, one might predict that the pulvinar plays a role in atten-
tion by linking together processes that are occurring in different
cortical areas. Such a general attentional role for all of pulvinar
would explain why monkeys or humans with inferior or lateral pul-
vinar lesions do poorly in visual search (Ungerleider & Christensen,
1977, 1979; Ogren, Mateer & Wyler, 1984)-they are not able to con-
join features of an object for which they are searching since these
features are processed in different parts of the brain and are atten-
tionally linked by the pulvinar. This linking process in the pulvinar
may in turn be regulated by the reticular thalamic nucleus (Crick,
1984).

Many area 7a neurons receive eye position signals. These eye
position signals are probably derived from oculomotor structures in
the brainstem that send eye position information to the cortex via
the intralaminar thalamic nuclei (Schlag-Rey & Schlag, 1984).

In summary, the major thalamic input to the inferior parietal
lobule is from the pulvinar. The pulvinar in turn receives ascending
inputs from oculomotor structures in the midbrain. A likely role of
the pulvinar is the regulation of cortical processes involved in
directed attention.

Multiple Cortical Areas in the Inferior Parietal Lobule

In the early pioneering studies of Mountcastle and colleagues,
several classes of neurons having visual, oculomotor, or somatic pro-
properties were identified in the inferior parietal lobule (Mountcastle,
Lynch, Georgopoulos, Sakata & Acuna 1975; Lynch, Mountcastle,
Talbot & Yin, 1977). The properties of these neurons were deter-
mined by recording their activity with microelectrodes while the
animals performed various motor and oculomotor tasks. These investigators made the observation that neurons with similar properties tended to be clustered together. From reconstructions of the locations of recording sights, they found no clear segregation of these properties into cortical fields (Lynch et al., 1977). They reasoned that the inferior parietal lobule was one large cortical field with a columnar organization of functional properties. The different functional columns were assumed to be more or less evenly dispersed. However, their data were pooled from several animals and referenced to sulcal patterns; this practice can smear considerably any topographic organization since the sulcal patterns and the relations of cortical fields to sulcal patterns vary extensively from animal to animal. Hyvarinen and colleagues (Hyvarinen & Shelepin, 1979; Hyvarinen, 1981) mapped the inferior parietal lobule and found a gradient of localization with somatic properties located more laterally and visual properties more medially. However, the methods used for determining the classification of cells were not well controlled in their experiments. In recent years anatomical experiments have indicated that the inferior parietal lobule can be subdivided into a number of different areas on the basis of connections (Pandya & Seltzer, 1982; Andersen, Asanuma & Cowan, 1985; Andersen, Siegel, Essick & Asanuma, 1985). Recent functional mapping experiments (in which large numbers of microelectrode penetrations are made, and the locations of the recordings are reconstructed, in individual animals) indicate functionally segregated areas in the inferior parietal lobule that coincide with the anatomically (connectionally) defined subdivisions (Essick, Andersen & Siegel, 1984; Andersen, Asanuma & Cowan, 1985; Andersen, Siegel, Essick & Asanuma, 1985). The following is a brief list of these newly recognized cortical areas, whose locations are diagrammed in Figure 3.5.

**Lateral intraparietal area.** This area is unique in being the only region of the inferior parietal lobule that projects strongly to the frontal eye fields (Barbas & Mesulam, 1981; Andersen, Asanuma & Cowan, 1985) and the superior colliculus (Lynch & Graybiel, 1983; Andersen, Asanuma & Cowan, 1985); both are structures involved with the generation of saccadic eye movements. The neurons in this area respond to both visual stimuli and to saccadic eye movements (Essick et al., 1984). Also, electrical stimulation of the area produces saccadic eye movements (Shibutani, Sakata & Hyvarinen, 1984). However, it is unlikely that this area is involved in the generation of saccades since the saccade-related component of the cells' responses generally occurs at or just after the beginning of an eye
movement (Essick et al., 1984). Rather, this area appears to be receiving a corollary discharge from motor structures such as the frontal eye fields informing this perceptual region to be prepared for a change in sensory input due to an eye movement.

**Area 7a.** Nearly every cell in area 7a can be shown to respond to visual stimuli (Andersen, Asanuma & Cowan, 1985). The receptive fields of these neurons are large and quite often bilateral (Motter & Mountcastle, 1981). Many of these cells also get eye position inputs...
and the visual response is modified by this eye position signal (Andersen & Mountcastle, 1983; Andersen et al., 1984). This area has a heavy projection to area 46 of the dorsolateral prefrontal cortex (Andersen, Asanuma & Cowan, 1985).

Area 7b. Area 7b contains neurons that respond to somatosensory stimuli and reaching behavior (Andersen, Asanuma & Cowan, 1985). This area projects to somatosensory association cortex (Andersen, Asanuma & Cowan, 1985).

Medial superior temporal area. This area is located primarily in the anterior bank of the caudal aspect of the superior temporal sulcus. This region receives a prominent visual input from the parieto-occipital area, the dorsal prelunate area, and the middle temporal area in the superior temporal sulcus (Colby et al., 1983; Andersen, Siegel, Essick & Asanuma, 1985; Maunsell & Van Essen, 1983). The middle temporal area is specialized for processing visual motion. The medial superior temporal area also appears to be specialized for aspects of motion processing since it contains a preponderance of cells with visual pursuit activity and motion sensitivity (Sakata, Shibutani & Kawano, 1983).

Dorsal prelunate area. This region is adjacent to the inferior parietal lobule and provides it with a major visual input (Andersen, Asanuma & Cowan, 1985). This cortical area is situated just dorsal to V4 on the dorsal-most tip of the prelunate gyrus and receives inputs from V4 and visual areas in the annectent gyrus (Andersen, Asanuma & Cowan, 1985).

The cortico-pontine projections and the patterns of thalamic input to the inferior parietal lobule are unique for each of the cortical areas outlined above (May & Andersen, 1984; Asanuma et al., 1985). The presence of these anatomically and functionally different subdivisions suggests that each of these sub-areas is specialized to process separate aspects of spatial perception.

PHYSIOLOGY

Most recent information on the processing role of the posterior parietal cortex for visual-spatial perception has come from single unit recordings made in behaving monkeys. In experiments of this type animals are trained to perform various motor and oculomotor tasks. Each trial of one of these tasks lasts 3 to 5 seconds. The animals do
one to two thousand trials over a six hour recording period and, with
the successful completion of each trial, they receive a drop of juice as
reward. While performing these tasks the activity of single cortical
neurons is recorded. The functional properties of a cortical area are
determined by correlating the behavior of the animals with the
activity of the neurons.

The response properties of posterior parietal neurons was first
described by Hyvarinen and Poranen (1974) and Mountcastle and
colleagues (1975). In their pioneering experiments Mountcastle and
colleagues (Mountcastle et al., 1975; Lynch et al., 1977) described
several classes of neurons in the inferior parietal lobule that were
active with reaching, with fixation, and with saccade and tracking
eye movements. These investigators proposed that the posterior
parietal cortex integrated information on the internal state of the
animal with events in the external environment, and generated gen­
eral commands for motor operation in the extrapersonal space. This
command hypothesis was challenged by Robinson and colleagues
(Robinson, Goldberg & Stanton, 1978) who found large numbers of
units in the posterior parietal cortex to be active to visual and soma­
tosensory stimuli. They proposed that cell activity related to reach­
ing was actually somatosensory in origin and that all presumed
fixation, saccade and tracking activity was an artifact of visual
stimulation by the laboratory environment as the animal moved his
eyes or fixated different locations in space. They therefore assigned
a sensory role to this area.

The issue of cell classification has recently been reexamined using
paradigms that separate the visual and eye movement or eye posi­
tion components of the neuronal responses (Sakata, Shibutani &
Kawano, 1980; Sakata et al., 1983; Essick et al., 1984). These exper­
iments show that these neurons generally receive a visual signal
and have an eye movement or eye position component to their
response. The saccade-related activity is consistent with a corollary
discharge since the activity generally occurs at or just after the
beginning of an eye movement. As elaborated in the next section,
the interaction of visual and eye position signals may result in the
mapping of visual space in head-centered coordinates (Andersen et
al., 1984).

The Representation of Space in Area 7a

The observations that motor movements are made accurately to loca­tions in space regardless of eye position and that the perception of
visual space is constant in spite of eye movements suggest that the
motor and perceptual systems have access to neural representations of visual space that are head-centered or ego-centered. A transformation from the retinal representations, which are the general feature of early cortical processing, to spatial representations requires interaction between retinal and eye position signals. This type of interaction has been shown to exist in the inferior parietal lobule (Andersen & Mountcastle, 1983; Andersen et al., 1984). Furthermore, given the profound spatial disturbances that result from parietal lobe lesions, the inferior parietal lobule is the most likely candidate for a cortical region that represents the visual scene in a head-centered or body-centered spatial frame.

The effect of the eyes' angle-of-gaze on light sensitive neurons in area 7a is to change the level of visual sensitivity dependent on eye position (Andersen et al., 1984). Thus, for the most preferred angle-of-gaze, the retinal receptive field will be extremely responsive to visual stimuli whereas at the least preferred angle-of-gaze the neuron will generally be completely unresponsive to visual stimuli. There is a smooth gradient in responsiveness with eye position between these two extremes. Thus, in its simplest form the activity of each of these cells can be described by a gain factor, which is a function of the angle-of-gaze, multiplied by the response profile of the retinal receptive field of the neuron. These cells are tuned to respond best to locations in head-centered coordinates which are defined by the best angle-of-gaze and the most sensitive part (center) of the receptive field. Cells are found tuned to every location in head-centered space in each hemisphere. Since the response is dependent on eye position, a spatially invariant response that is independent of eye position can only be found in the activity of groups of space-tuned neurons in area 7a.

Motion

Relative motion is the differential movement of elements of the visual field. This type of motion is important for spatial perception, indicating depth with motion parallax, movement in depth with expansions or contractions of the field, and the relative position of objects from occlusion and disocclusion during self-motion. Cells in the inferior parietal lobule show exquisite sensitivity for relative motion. Motter and Mountcastle (1981) described an opponent vector organization for motion sensitivity, finding cells with bilateral receptive fields which are active for stimuli moving either inward or outward from the fixation point. Such neurons would be maximally active for translation of the head forward or backward in the
environment and could provide information about position by analyzing flow fields during motion. Sakata and colleagues (Sakata, Shibutani, Kawano & Harrington, 1985) have described different sets of cells in the inferior parietal lobule that appear to be selective for rotations in the sagittal, horizontal or frontal planes. Many of the relative-motion-sensitive neurons are located in the anterior bank of the superior temporal sulcus and may receive a convergence of directional information from the middle temporal cortical field.

Attention

Considering the profound defects in visual attention after lesions to this area, it is not surprising to find that the level of activity of these neurons is dependent on visual attention. Cells respond more vigorously to a visual stimulus if it is behaviorally relevant to the animal (Bushnell, Goldberg & Robinson, 1981). The responsiveness of the cells, even for stimuli that are not behaviorally significant, is greatly facilitated when the animal performs tasks requiring attentive fixations (Mountcastle et al., 1981). This facilitation is not a sensory effect since it is still present if the fixation target is removed as long as the animal maintains fixation. The facilitation of area 7 neurons is also not one of general arousal since it is not present in tasks requiring the animal’s attention but without fixation. The spatial tuning of cells with angle-of-gaze effects is less pronounced without attentional fixations (Andersen & Mountcastle, 1983). This finding suggests a link between spatial and attentional processes in the parietal lobe.

CONCLUSION

The inferior parietal lobule plays a central role in spatial perception and visual attention. Lesions to this area in monkeys and humans profoundly disrupt spatial abilities. Many of the cells in this area appear to encode the position of visual objects in head- or ego-centered coordinates. The area also appears to play a role in extracting spatial information from relative motion. The visual activity of the cells is dependent on behavioral state and may provide clues for the role of this area in spatial aspects of attention. The inferior parietal lobule is not one cortical field but several, each of which appears to be specialized for certain aspects of spatial analysis.

In the future it will be important to know whether there is a topographic representation of space in head- or body-centered coordinates.
within this area or whether groups of cells encode locations of objects by learned connections within random networks. Do learned adjustments to spatial perceptions and motor guidance which accompany spatial distortions in visual inputs introduced, for instance, by prisms lead to long-term changes in the activity of space-tuned cells? What is the source of the eye-position signal-proprioception or efference copy? Much more needs to be known about the division of labor among the various cortical fields in the inferior parietal lobule and how they interact to provide spatial constancy.

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