THE NEUROBIOLOGICAL BASIS OF SPATIAL COGNITION: ROLE OF THE PARIETAL LOBE

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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 retinae. 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. Sideness 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

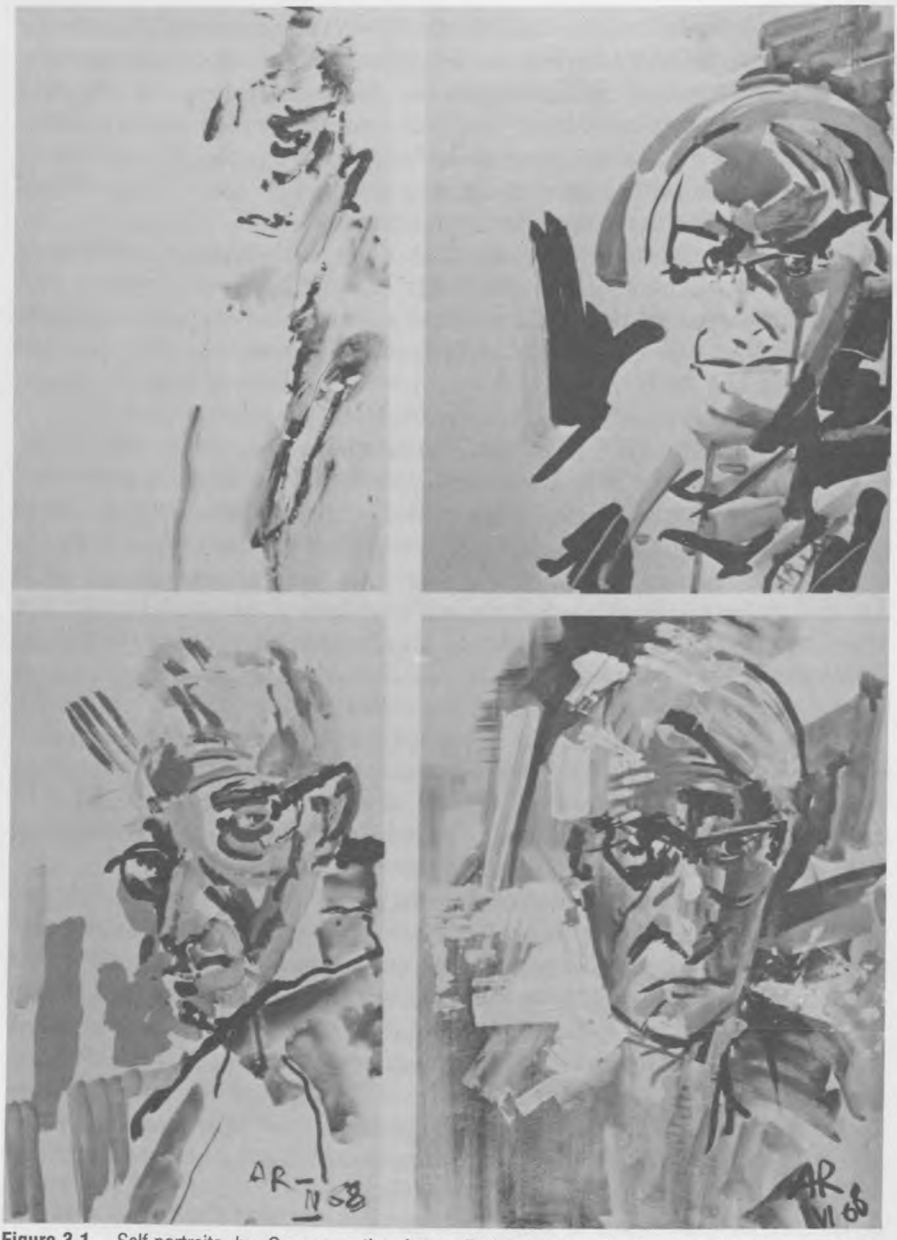

Figure 3.1. Self-portraits by German artist Anton Raderscheidt. These portraits were made at different times after a stroke that damaged the right parietal cortex. The portraits were made two months (upper left), three and a half months (upper right), six months (lower left) and nine months (lower right) 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).

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 piecemeal 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-

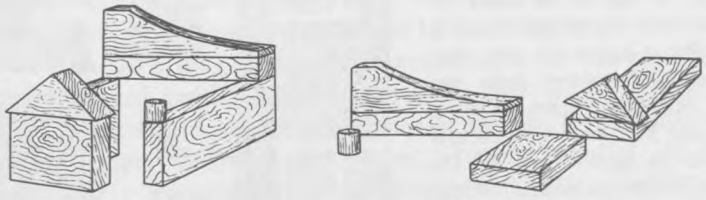

Figure 3.2. Constructional apraxia. A patient with a left fronto-parietal metastatic tumor was asked to model the block construction on the left. The patient's copy on the right indicates a poor performance on this three-dimensional task. Figure from Critchley, 1953.

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

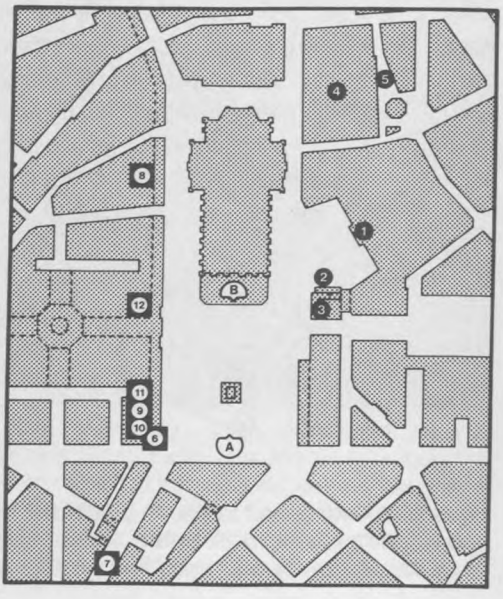

Figure 3.3. A map of the Piazza del Duomo in Milan. Contralateral neglect is demonstrated in patient I.G. who, after suffering a right hemisphere stroke, was asked to recall from memory landmarks on the square from the two perspectives indicated in the figure (A and B). The numbered dark circles indicate 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

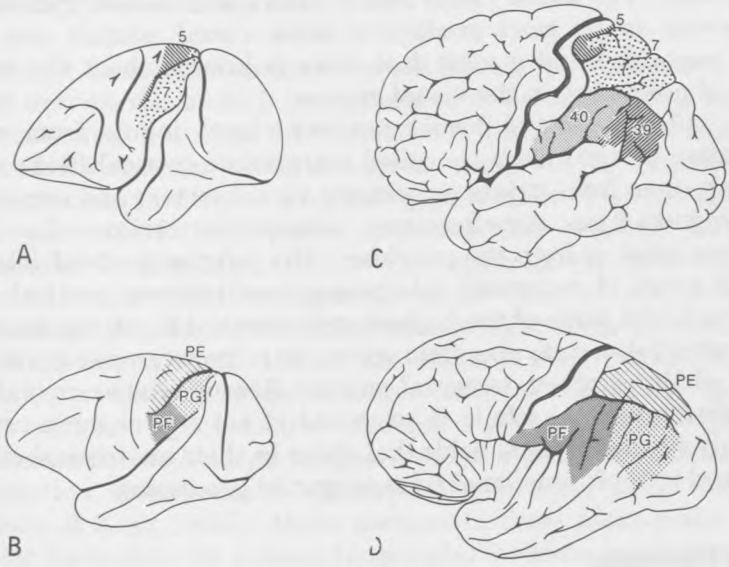

Figure 3.4. Cytoarchitectonics of the posterior parietal cortex. Lateral views of monkey and human cerebral cortices showing cytoarchitectural parcellations made by different anatomists of the posterior parietal cortex.

(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 reciprocally 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 attention 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 pulvinar lesions do poorly in visual search (Ungerleider & Christensen, 1977, 1979; Ogren, Mateer & Wyler, 1984)-they are not able to conjoin features of an object for which they are searching since these features are processed in different parts of the brain and are attentionally 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 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 determined 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 eyefields (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

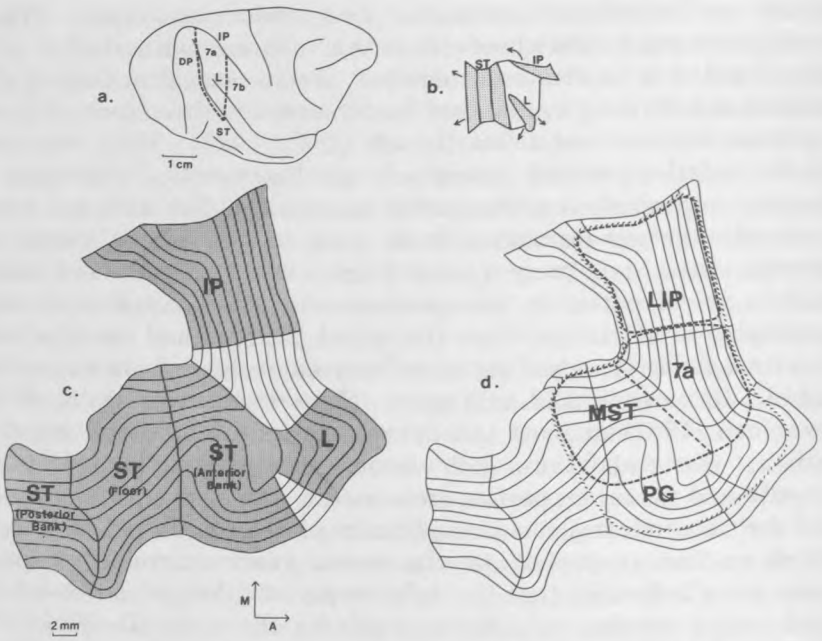

Figure 3.5. Flattened reconstructions of the inferior parietal lobule. Locations of different cortical areas in the inferior parietal and adjoining dorsal prelunate cortex.

(a) Dorsolateral view of the right hemisphere of a macaque monkey. The dotted line indicates a section of the inferior parietal lobule that has been diagramatically flattened in the remaining panels.

(b) The method of flattening the inferior parietal lobule. The cortex is treated as a folded sheet centered on layer IV. The cortex buried in the superior temporal sulcus is pulled out to the left, the cortex of the intraparietal sulcus is flipped upward and the cortical walls of the lateral sulcus are pulled apart.

(c) Shading indicates flattened areas that lie within sulci.

(d) Locations of two cortical areas, the lateral intraparietal area and the medial superior temporal area, which lie buried within the sulci. Also indicated is the cytoarchitectural subdivision PG of von Bonin and Bailey (1947) which includes the dorsal prelunate area. Figure modified from Andersen, Siegel, Essick and Asanuma, 1985.

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 anectent 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 general 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 somatosensory stimuli. They proposed that cell activity related to reaching 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 position components of the neuronal responses (Sakata, Shibutani & Kawano, 1980; Sakata et al., 1983; Essick et al., 1984). These experiments 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.

ACKNOWLEDGMENTS

Supported by NIH Grant EY 05522. The author is recipient of a McKnight Foundation Scholars Award and a Sloan Foundation Scholarship, and is a Clayton Foundation Investigator.

REFERENCES

Acuna, C., Gonzalez, F. & Dominguez, R. 1983. Sensorimotor unit activity related to intention in the pulvinar of behaving Cebus Apella monkeys. *Experimental Brain Research.* 52:411-422.

Andersen, R. A., Asanuma, C., & Cowan, W. M. 1985. Callosal and prefrontal associational projecting cell populations in area 7a of the macaque monkey: a study using retrogradely transported fluorescent dyes. *Journal of Comparative Neurology.* 232:443-455.

Andersen, R. A., Essick, G. K., & Siegel, R. M. 1984. The role of eye position on the visual response of neurons in area 7a. *Society for Neuroscience Abstract,.* 10:934.

Andersen, R. A., & Mountcastle, V. B. 1983. The influence of the angle of gaze upon the excitability of the light-sensitive neurons of the posterior parietal cortex. *Journal of Neuroscience.* 3:532-548.

Andersen, R. A., Siegel, R. M., Essick, G. K., & Asanuma, C. 1985. Subdivision of the inferior parietal lobule and dorsal prelunate gyrus of macaque by connectional and functional criteria. *Investigative Ophthalmology and Visual Science Abstract.* 26(Suppl.):266.

Asanuma, C., Andersen, R. A., & Cowan, W. M. 1982. Divergent cortical projections of the medial pulvinar nucleus: a retrograde fluorescent tracer study in the monkey. *Society for Neuroscience Abstract.* 8:210.

Asanuma, C., Andersen, R. A., & Cowan, W. M. 1985. The thalamic relations of the caudal inferior parietal lobule and the lateral prefrontal cortex in monkeys: divergent cortical projections from cell clusters in the medial pulvinar nucleus. *Journal of Comparative Neurology.* 241:357-381.

Barbas, H., & Mesulam, M-M. 1981. Organization of afferent inputs to subdivisions of area 8 in rhesus monkeys. *Journal of Comparative Neurology.* 200:407-431.

Bender, D. B. 1981. Retinotopic organization of macaque pulvinar. *Journal of Neurophysiology.* 46:672-693.

Bender, D. B. 1983. Visual activation of neurons in the primate pulvinar depends on cortex but not colliculus. *Brain Research.* 279:258-261.

Benevento, L. A., & Miller, J. 1981. Visual responses of single neurons in the caudal lateral pulvinar of the macaque monkey. *Journal of Neuroscience.* 11:1268-1278.

Benevento, L. A., & Standage, G. P. 1983. The organization of projections of the retinorecipient and nonretinorecipient nuclei of the pretectal complex and layers of the superior colliculus to the lateral pulvinar and medial pulvinar in the macaque monkey. *Journal of Comparative Neurology.* 217:307-336.

Benson, D. F., & Barton, M. I. 1970. Disturbances in constructional ability. *Cortex.* 6:19-46.

Bisiach, E., & Luzzatti, C. 1978. Unilateral neglect of representational space. *Cortex.* 14:129-133.

Brain, W. R. 1941. Visual disorientation with special reference to lesions of the right cerebral hemisphere. *Brain.* 64:244-272.

Brodmann, K. 1905. Beitrage zur histologischen Lokalisation der Grosshirnrinde. Dritte Mitteilung: Die Rindenfelder, der niederen Affen. *Journal fur Psychologie und Neurologie.* 4:177-226.

Brodmann, K. 1907. Beitrage zur histologischen Lokalisation der Grosshirnrinde. Sechste Mitteillung: Die Cortex gliederung des Menschen. *Journal fur Psychologie und Neurologie.* 10:231-246.

Brody, B. A., & Pribram, K. H. 1978. The role of frontal and parietal cortex in cognitive processing: tests of spatial and sequential functions. *Brain.* 101:607-633.

Bushnell, M. C., Goldberg, M. E., & Robinson, D. L. 1981. Behavioral enhancement of visual responses in monkey cerebral cortex. I. Modulation in posterior parietal cortex related to selective visual attention. *Journal of Neurophysiology.* 46:755-772.

Butters, N., & Barton, M. 1970. Effect of parietal lobe damage on the performance of reversible operations in space. *Neuropsychologia,* 8:205-214.

Carmon, A., & Bechtoldt, H. P. 1969. Dominance of the right cerebral hemisphere for stereopsis. *Neurospychologia.* 7:29-39.

Colby, C. L, Gattass, R. Olson, C. R., & Gross, C. G. 1983. Cortical afferents to visual areas in the macaque. *Society for Neuroscience Abstract.* 9:152.

Crick, F. 1984. Function of the thalamic reticular complex: the searchlight hypothesis. *Proceedings of the National Academy of Science, U.S.A.* 81:4586-4590.

Critchley, M. 1953. *The Parietal Lobes,* New York: Hafner Press.

Denny-Brown, D., & Banker, B. 1954. Amorphosynthesis from left parietal lesion. *A.M.A. Archives of Neurology and Psychiatry.* 71:302-313.

De Renzi, E. 1982. Memory disorders following focal neocortical damage. *Philosophical Transactions of the Royal Society of London B.* 298:73-83.

De Renzi, E., Faglioni, P., & Previdi, P. 1977. Spatial memory and hemispheric locus of lesion. *Cortex.* 13:424-433.

De Renzi, E., Faglioni, P., & Scotti, G. 1970. Hemispheric contribution to exploration of space through the visual and tactile modality. *Cortex.* 6:191-203.

De Renzi, E., Faglioni, P., & Villa, P. 1977. Topographical amnesia. *Journal of Neurology, Neurosurgery and Psychiatry.* 40:498-505.

Desimone, R. & Gross, C. G. 1979. Visual areas in the temporal cortex of the macaque. *Brain Research.* 178:363-380.

Essick, G. K., Andersen, R. A., & Siegel, R. M. 1984. Fixation and saccade-related area 7a neurons receive visual inputs. *Society for Neuroscience Abstracts.* 10:476.

Ettlinger, G., & Kalsbeck, J. E. 1962. Changes in tactile discrimination and in visual reaching after successive and simultaneous bilateral posterior parietal ablations in the monkey. *Journal of Neurology, Neurosurgery and Psychiatry.* 25:256-268.

Ettlinger, G., Warrington, E., & Zangwill, O. L. 1957. A further study of visual-spatial agnosia. *Brain.* 80:335-361.

Faugier-Grimaud, S., Frenois, C., & Stein, D. G. 1978. Effects of posterior parietal lesions on visually guided behavior in monkeys. *Neuropsychologia.* 16:151-168.

Hartje, W., & Ettlinger, G. 1974. Reaching in light and dark after unilateral posterior parietal ablations in the monkey. *Cortex.* 9:346-354.

Hecaen, H., & Ajuriaguerra, de J. 1954. Balint's syndrome (psychic paralysis of visual fixation) and its minor forms. *Brain.* 77:373-400.

Hecaen, H., Penfield, W., Bertrand, C., Malmo, R. 1956. The syndrome of apractognosia due to lesions of the minor cerebral hemisphere. *A.M.A. Archives of Neurology and Psychiatry.* 75:400-434.

Holmes, G. 1919. Disturbances of visual space perception. *British Medical Journal.* 2:230-233.

Hyvarinen, J. 1981. Regional distribution of functions in parietal association area 7 of the monkey. *Brain Research.* 206:287-303.

Hyvarinen, J., & Poranen, A. 1974. Function of the parietal associative area 7 as revealed from cellular discharges in alert monkeys. *Brain.* 97:673-692.

Hyvarinen, J., & Shelepin, Y. 1979. Distribution of visual and somatic functions in the parietal associative area 7 of the monkey. *Brain Research.* 169:561-564.

Jung, R. 1974. Neuropsychologie under Neurophysiologie des Kontar-und Formsehens in Zeichnung und Mulerei. In Wieck H. H. (ed.) *Psychopathologie Musischer Gestaltungen.* Schattauer Verlag, Stuttgart, 29-88.

Kasdon, D. L., & Jacobson, S. 1978. The thalamic afferents to the inferior parietal lobule of the Rhesus monkey. *Journal of Comparative Neurology.* 177:685-706.

Kinsbourne, M., & Warrington, E. K. 1962. A disorder of simultaneous form perception. *Brain.* 45:461-486.

LaMotte, R. H., & Acuna, C. 1978. Defects in accuracy of reaching after removal of posterior parietal cortex in monkeys. *Brain Research.* 139:309-326.

Lynch, J. C., & Graybiel, A. M. 1983. Comparison of afferents traced to the superior colliculus from the frontal eye fields and from two sub-regions of area 7 of the rhesus monkey. *Society for Neuroscience Abstract.* 9:750.

Lynch, J. C., & McLaren, J. W. 1984. A quantitative study of contralateral inattention in monkey following lesions of posterior parietal, prestriate, and prefrontal cortex. *Society for Neuroscience Abstract.* 10:59.

Lynch, J. C., Mountcastle, V. B., Talbot, W. H., & Yin, T. C. T. 1977. Parietal lobe mechanisms for directed visual attention. *Journal of Neurophysiology.* 40:362-389.

Maunsell, J. H. R., & Van Essen, D. C. 1983. The connections of the middle temporal visual area (MT) and their relationship to a cortical hierarchy in the macaque monkey. *Journal of Neuroscience.* 3:2563-2586.

May, J. G., & Andersen, R. A. 1984. Different patterns of cortico-pontine projections from different cortical regions within the inferior parietal lobule and dorsal prelunate gyrus of the monkey. *Society for Neuroscience Abstract.* 10:577.

McFie, J., & Zangwill, O. L. 1960. Visual-constructive disabilities associated with lesions of the left cerebral hemisphere. *Brain.* 83:243-260.

Mendoza, J. E., & Thomas, R. K. 1975. Effects of posterior parietal and frontal neocortical lesions in the squirrel monkey. *Journal of Comparative and Physiological Psychology.* 89:170-182.

Mesulam, M. -M., Van Hoesen, G. W., Pandya, D. N., & Geschwind, N. 1977. Limbic and sensory connections of the inferior parietal lobule (area PG) in the rhesus monkey: a study with a new method for horseradish peroxidase histochemistry. *Brain Research.* 136:393-414.

Mishkin, M. 1982. A memory system in the monkey. *Philosophical Transactions of the Royal Society of London B.* 298:85-95.

Moffett, A., Ettlinger, G., Morton, H. B., & Piercy, M. F. 1967. Tactile discrimination performance in the monkey: the effects of ablation of various subdivision of the posterior parietal cortex. *Cortex.* 3:59-96.

Motter, B. C., & Mountcastle, V. B. 1981. The functional properties of the light sensitive neurons of the posterior parietal cortex studied in waking monkeys: foveal sparing and opponent vector organization. *Journal of Neuroscience.* 1:3-26.

Mountcastle, V. B., Andersen, R. A., & Motter, B. C. 1981. The influence of attentive fixation upon the excitability of the light-sensitive neurons of the posterior parietal cortex. *Journal of Neuroscience.* 1:1218-1235.

Mountcastle, V. B., Lynch, J. C., Georgopoulos, A., Sakata, H., & Acuna, C. 1975. Posterior parietal association cortex of the monkey: command functions for operations within extrapersonal space. *Journal of Neurophysiology.* 38:871-908.

Ogren, M. P., & Hendrickson, A. E. 1979. The morphology and distribution of striate cortex terminals in the inferior and lateral subdivisions of the

Macaca monkey pulvinar. *Journal of Comparative Neurology.* 188:197-200.

Ogren, M. P., Mateer, C. A., & Wyler, A. R. 1984. Alterations in visually related eye movements following left pulvinar damage in man. *Neuropsychologia.* 22:187-196.

Pandya, D. N., & Seltzer, B. 1982. Intrinsic connections and architectonics of posterior parietal cortex in the rhesus monkey. *Journal of Comparative Neurology.* 204:196-210.

Paterson, A., & Zangwill, O. L. 1944. Disorders of visual space perception associated with lesions of the right hemisphere. *Brain.* 67:331-358.

Perryman, K. M., Lindsley, D. F., & Lindsley, D. B. 1980. Pulvinar neuron responses to spontaneous and trained eye movements and to light flashes in squirrel monkeys. *Electroencephalography and Clinical Neurophysiology.* 49:152-161.

Petersen, S. E., Robinson, D. L., & Keys, W. 1982. A physiological comparison of the lateral pulvinar and area 7 in the behaving macaque. *Society for Neuroscience Abstract.* 8:681.

Petrides, M., & Iversen, S. E. 1979. Restricted posterior parietal lesions in the rhesus monkey and peformance on visuospatial tasks. *Brain Research.* 161:63-77.

Piercy, M., Hecaen, H., & Ajuriaguerra, J. 1960. Constructional apraxia associated with unilateral cerebral lesions—left and right sided cases compared. *Brain.* 83:225-242.

Pohl, W. 1973. Dissociation of spatial discrimination deficits following frontal and parietal lesions in monkeys. *Journal of Comparative and Physiological Psychology.* 82:227-239.

Ratcliff, G., & Davies-Jones, G. A. B. 1972. Defective visual localization in focal brain wounds. *Brain.* 95:49-60.

Ratcliff, G., Ridley, R. M., & Ettlinger, G. 1977. Spatial disorientation in the monkey. *Cortex.* 13:62-65.

Riddoch, G. 1917. On the relative perceptions of movement and a stationary object in certain visual disturbances due to occipital injuries. *Proceedings of the Royal Society of Medicine.* 10:13-34.

Robinson, D. L., Goldberg, M. E., & Stanton, G. B. 1978. Parietal association cortex in the primate: sensory mechanisms and behavioral modulations. *Journal of Neurophysiology.* 41:910-932.

Sakata, H., Shibutani, H., & Kawano, K. 1980. Spatial properties of visual fixation neurons in posterior parietal association cortex of the monkey. *Journal of Neurophysiology.* 43:1654-1672.

Sakata, H. Shibutani, H., & Kawano, K. 1983. Functional properties of visual tracking neurons in posterior parietal association cortex of the monkey. *Journal of Neurophysiology.* 49:1364-1380.

Sakata, H., Shibutani, H., Kawano, K., Harrington, T. L. 1985. Neural mechanism of space vision in the parietal association cortex of the monkey. *Vision Research.* 25:453-463.

Schlag-Rey, M., & Schlag, J. 1984. Visuomotor functions of central thalamus in monkey. I. Unit activity related to spontaneous eye movements. *Journal of Neurophysiology.* 6:1149-1174.

Seltzer, B., & Pandya, D. N. 1980. Converging visual and somatic sensory cortical input to the intraparietal sulcus of the rhesus monkey. *Brain Research.* 192:339-351.

Semmes, J., Weinstein, S., Ghent, L., & Teuber, H. L. 1963. Correlates of impaired orientation in personal and extrapersonal space. *Brain.* 86:747-772.

Shibutani, H., Sakata, H., & Hyvarinen, J. 1984. Saccade and blinking evoked by microstimulation of the posterior parietal association cortex of the monkey. *Experimental Brain Research.* 55:1-8.

Siqueira, E. B. 1965. The temporo-pulvinar connections in the rhesus monkey. *Archives of Neurology.* 13:321-330.

Siqueira, E. B. 1971. The cortical connections of the nucleus pulvinaris of the dorsal thalamus in rhesus monkey. *International Journal of Neurology.* 8:139-154.

Teuber, H. L. 1963. Space perception and its disturbances after brain injury in man. *Neuropsychologia.* 1:47-57.

Trojanowski, J. Q., & Jacobson, S. 1974. Medial pulvinar afferents to frontal eye fields in rhesus monkey demonstrated by horseradish peroxidase. *Brain Research.* 80:395-411.

Trojanowski, J. Q., & Jacobson, S. 1976. Areal and laminar distribution of some pulvinar cortical efferents in rhesus monkey. *Journal of Comparative Neurology.* 169:371-392.

Ungerleider, L. G., & Brody, B. A. 1977. Extrapersonal spatial orientation: the role of posterior parietal, anterior frontal and inferotemporal cortex. *Experimental Neurology.* 56:265-280.

Ungerleider, L. G., & Christensen, C. A. 1977. Pulvinar lesions in monkeys produce abnormal eye movements during visual discrimination training. *Brain Research.* 136:189-196.

Ungerleider, L. G., & Christensen, C. A. 1979. Pulvinar lesions in monkeys produce abnormal scanning of a complex visual array. *Neuropsychologia.* 17:493-501.

Vogt, C., & Vogt, O. 1919. Allgemeine Ergebuisse unserer Hirnforschung. *Journal fur Psychologie und Neurologie.* 25:279-462.

von Bonin, G., & Bailey, P. 1947. *The Neocortex of Macaca Mulatta.* Urbana: University of Illinois Press.

von Economo, C. 1929. *The Cytoarchitectonics of the Human Cerebral Cortex.* London: Oxford University Press.

Wilbrand, H. 1887. *Die Seelenblindheit als Herderscheinung.* Wiesbaden: Bergmann.

Wurtz, R. H., Goldberg, M. E., & Robinson, D. L. 1982. Brain mechanisms of visual attention. *Scientific American.* 246:124-135.