The neurobiology of saccadic eye movements
Eds. Wurtz & Goldberg

CHAPTER 8

Posterior parietal cortex

Richard A. Andersen* and James W. Gnadt**

The Salk Institute, San Diego, CA, U.S.A.

I. Introduction

It has long been known that the posterior parietal cortex plays an important role in the processing of saccadic eye movements directed to visual targets. Rudolph Balint, a clinical neurologist (1909), was the first to describe a patient with bilateral posterior parietal lesions who evidenced disorders in saccadic eye movements. This patient demonstrated, among other deficits, an inability to will eye movements although spontaneous eye movements were unaffected. Since this early description, numerous examples of deficits in the ability to make voluntary saccades to visual stimuli after biparietal lesions have been reported in the clinical literature.

The exact role of the posterior parietal cortex in programming saccades has until recently been far from clear. With the advent of single cell recording experiments in behaving monkeys, investigators began to examine the activity of neurons in this area in relation to eye movements. Mountcastle and colleagues (1975), in their pioneering experiments in this area, reported cells with activity preceding visually evoked saccades and proposed that the area issued general commands to make saccadic eye movements. Subsequently Robinson, Goldberg and Stanton (1978) observed many cells in the posterior parietal cortex that were responsive to visual stimuli. They and Yin and Mountcastle (1977, 1978) also made the observation that the visual responsiveness of neurons was enhanced if the visual stimuli served as targets for saccades. Robinson et al. (1978) challenged Mountcastle's command hypothesis, arguing that neurons in this area were responding in a sensory fashion to the saccade targets as visual stimuli rather than as motor units to the behavior of making saccades. Based on the observation of saccadic enhancement of visual responses and the well known attentional deficits that occur after posterior parietal lesions, these investigators proposed that the area was involved in directing visual attention rather than playing any direct role in the programming of saccades.

Recent experiments have established that posterior parietal cortical neurons discharge in relation to both visual stimuli and saccadic eye movements (Andersen et al., 1987). These results suggest that the functional role of the posterior parietal cortex should be considered neither strictly sensory nor strictly motor but rather one involved with sensorimotor integration; that is, the programming of saccadic eye movements based on sensory guidance (Andersen, 1987). Another important advance has been the recent discovery of an area buried in the intra-parietal sulcus, the lateral intraparietal area

*To whom correspondence should be addressed at: Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology, 77 Massachusetts Avenue, Cambridge, MA 02139, U.S.A.

**Present address: Neuroscience Program, University of Alabama, Birmingham, AL 35294, U.S.A.

(LIP), that appears to be related to saccadic eye movements (Gnadt and Andersen, 1988).

An understanding of the role of the posterior parietal cortex in processing saccades can be gained by examining data from lesion studies and anatomical and physiological recording experiments. The first section of this chapter will review the effects of lesions of the posterior parietal cortex in humans and monkeys on saccadic eye movements. The anatomy of the posterior parietal cortex, with particular emphasis on the connectional relationship of this area with visual and eye movement centers, will be reviewed next. Physiological data and the recent history of ideas regarding the role of this area in processing saccades will then be traced and the review will conclude with evidence that area LIP is specialized for visuomotor integration.

2. Human lesions

Difficulty in making saccades occurs with bilateral lesions to the posterior parietal cortex. Generally this deficit recovers after several months. As mentioned above, while these bilateral lesions are rare, there are several reports of patients so affected in the literature, with pathologies ranging from gunshot wounds through the head to brain tumors to saggital sinus disease, CVAs and congenital injury. Eye movement defects have also been reported with unilateral lesion but they are much less dramatic. We will therefore focus on the bilateral lesion cases.

Balint first described eye movement disorders in a patient with bilateral posterior parietal lesions in 1909. There are three features of a clinical entity that has come to be known as Balint's syndrome: (1) psychic paralysis of visual fixation in which patients cannot look to a point in space defined by a visual target, but under other circumstances have normal ocular motility; (2) optic ataxia (Allison et al., 1969; Luria et al., 1963) or visual disorientation (Holmes 1918), in which patients cannot locate visual targets in space; and (3) restriction in visual attention in which patients can only perceive one object at a time, irrespective of the object's angular extent and in the absence of visual field defects. Unilateral lesions often produce the attentional and spatial defects limited to contralateral space and may also produce eye movement deficits.

Patients with Balint's syndrome have normal eye movements except when looking to visual targets. They can look left or right on verbal command (Godwin-Austen, 1965) or to a sound (Allison et al., 1964). However, they cannot look spontaneously or on command to a visual target (Balint, 1909; Cogan and Adams, 1953; Godwin-Austen, 1965; Allison et al., 1969). Often these patients have locked fixations and a paucity of eye movements (Hecaen and Ajuriaguerra, 1954; Allison et al., 1969). When eye movements are made, the patients often initially look in the wrong direction and then their eyes wander until by chance they land on target. Hecaen and Ajuriaguerra (1954) and Allison et al. (1969) have argued that the locked fixation is a defence against the extreme disorientation that would result from constantly wandering eye movements. Other accompanying oculomotor deficits include (1) defective or absent smooth pursuit eye movements (Hecaen and Ajuriaguerra, 1954; Godwin-Austen, 1965), (2) difficulties in accommodation and vergence (Holmes, 1918; Holmes and Horrax, 1919; Cogan and Adams, 1953; Godwin-Austen, 1965), (3) absence of the blink reflex for approaching objects (Holmes, 1918; Holmes and Horrax, 1919), and (4) occasional problems maintaining fixation (Godwin-Austen, 1965). Luria et al. (1963) recorded intact smooth pursuit eye movements in a patient who could not make saccades. Holmes (1918) has speculated that the lack of blink reflex may be secondary to a loss of distance perception, since his affected patient did not realize when objects were brought close to his face.

Cogan (1965) described two types of oculomotor deficit with cerebral cortex bilateral lesions. What he termed ocular motor apraxia (which was equivalent to the disorders of Balint's syndrome) was characterized by an inability of patients to make willed eye movements to visual targets. The second type was a paralysis of gaze. The difference between the two was that in the former motor apraxia the

patient only exhibited difficulty in initiating movement with no limitation in the excursion of the eyes, while in the latter paralysis the excursions of eye movements were severely restricted. He further reported that oculomotor apraxia generally resulted from biparietal lesions, whereas paralysis of gaze resulted from bilateral frontal lobe lesions.

Patients with visual disorientation have normal visual acuity as well as normal limb movements, being proficient at any task not requiring visual guidance. However, all movements requiring visual guidance are severely disrupted. Such patients cannot reach accurately to objects, being inaccurate in all three dimensions but being most inaccurate in depth. Patients have difficulty walking, colliding with clearly visible objects and exhibiting difficulty walking around them (Holmes, 1918; Holmes and Horrax, 1919; Cogan and Adams, 1953; Hacaen and Ajuriaguerra, 1954; Luria et al., 1963; Godwin-Austen, 1965; Allison et al., 1969). The specific nature of this deficit is vividly demonstrated by the example of Holmes' and Horrax's (1919) patient who had no difficulty feeding himself soup from a bowl with a spoon as long as he could hold the bowl in one hand. However, if he could not hold the bowl, he would then have to grope for it with the spoon until he touched it. Holmes (1918) interpreted the visual disorientation as a loss of association between retinotopic visual inputs and proprioceptive information about eye and head position. Of course another possibility would be a disruption of the integration of efference copy motor signals and memory sensory signals.

The third sign of Balint's syndrome is a restriction of attention, which has also been referred to as a disorder of simultaneous perception or simultagnosia. Patients with this disorder exhibit extinction; when confronted with two simultaneously presented visual targets, usually one in each visual hemifield, they perceive only one. These patients are widely reported to complain that when they look at one object all other visual stimuli disappear from perception. Generally foveal vision overrides peripheral vision so that these patients only see what they are fixating. The deficit appears to be object-based, since perception is limited to a single object regardless of its retinal size; if attention is drawn to a detail or small part of the object then the perception of the object as a whole is lost (Holmes, 1918; Holmes and Horrax, 1919; Hacaen and Ajuriaguerra, 1954; Allison et al., 1964; Godwin-Austen, 1965).

There are several lines of evidence suggesting that the three signs of Balint's syndrome are functionally independent. It could be argued that visual disorientation (spatial deficit) is a result of disordered eye movements. However, patients with brainstem lesions that produce severe eye movement disorders have no difficulty locating visual targets with reaching (Holmes, 1918; Allison et al., 1969). On the other hand, patients with biparietal lesions also mislocate targets even if they are fixating them. The attentional and visual disorientation deficits do not appear to be interrelated, since visual inattention often occurs as a unilateral phenomenon when spatial orientation is unaffected. Another argument against the independence of the signs of Balint's syndrome would be that the impairment in eye movements is due to visual disorientation and inattention. For instance, the locking of fixation is due to the foveally restricted attention that does not allow peripheral visual targets to enter consciousness, and the wandering of the eyes during eye movements is due to the fact that the eyes do not know where to look. This is a more difficult possibility to rule out. One of Holmes' (1918) patients could look accurately to his own hands, suggesting that he had knowledge of the spatial location of his hands and could therefore direct gaze to them. This result would argue for a disruption in visual-spatial perception being at the root of the eye movement disorders. However, two of his other patients could not look at their own fingers or bring their gaze to positions touched on the body. These results suggest a fundamental impairment of eye movements.

3. Monkey lesions

Most work on monkeys has emphasized deficits in reaching after unilateral or bilateral parietal lesions

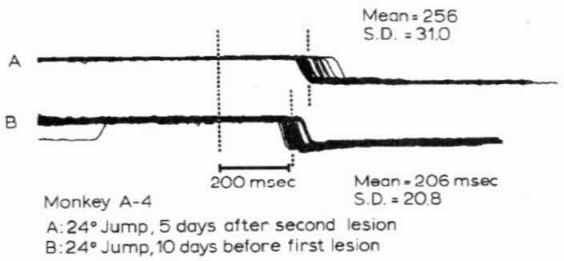


Fig. 1. Latency of visually evoked saccades before (B) and after (A) bilateral posterior parietal lesions. The horizontal eye-position records indicate an increased latency of 25% as a result of the lesions. The long dashed vertical line indicates the time at which the command to make a saccade was made and the two short dashed lines indicate the mean saccade latencies. From Lynch, 1980.

(Ettlinger and Kalsbeck, 1962; Moffet et al., 1967; Hartje and Ettlinger, 1974; Ratcliff et al., 1977; Faugier-Frimaud et al., 1978; Stein, 1978; LaMotte and Acuna, 1978; Glickstein and Mays, 1982). Oculomotor deficits have not been studied in much detail. Lynch (1980) found an increase in saccade latencies after bilateral parietal-occipital lesions (Fig. 1). Tusa et al. (1986) found that unilateral decortication produced deficits in intentional saccades for movements made away from the side of the lesion and for all saccades in the contralateral craniotopic space; however, since these lesions were large it is not clear to what extent these deficits reflect the loss of posterior parietal cortex or cortex in other brain regions. In conclusion, there are presently few data on the effects of lesions of the posterior parietal cortex in monkeys on saccadic eye movements.

4. Anatomical and functional organization

The posterior parietal cortex is located in the caudal aspect of the parietal lobe. This cortical area contains the superior and inferior parietal lobules. Brodmann (1905) designated the superior parietal lobule area 5 and the inferior parietal lobule area 7 (see Fig. 2A). Area 5 contains exclusively somatosensory association cortex. Area 7 was later further subdivided into two areas on cytoarchitectural grounds; a caudal medial area designated 7a by Vogt and Vogt (1919) or PG by von Bonin and Bailey (1947) and a more lateral-rostral area 7b or PF (see Fig. 2B). The inferior parietal lobule is comprised of not only the cortex on the gyral surface but also the cortex in the lateral bank of the intraparietal sulcus, the cortex in the anterior bank of the caudal third of the superior temporal sulcus, and a small section of cortex on the medial wall of the cerebral hemisphere.

The homologies between areas in the posterior parietal cortex in monkeys and humans are unclear. Brodmann claimed that the superior parietal lobule of man was cytoarchitecturally the same as the inferior parietal lobule of the monkey (Fig. 2A,C). Thus there would be no homologous area in the monkey for Brodmann's areas 39 and 40 which comprise the inferior parietal lobule of humans (Fig. 2C). Von Bonin and Bailey criticized Brodmann's work in the monkey and claimed that their PG and PF of the inferior parietal lobule were homologous to von Economo's PG and PF which

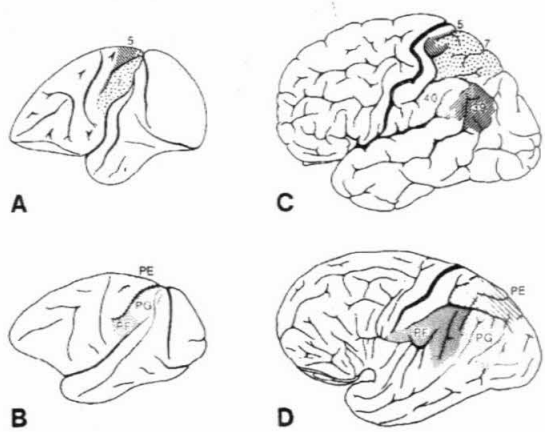


Fig. 2. Lateral views of monkey and human cerebral hemispheres showing different cytoarchitectural parcellation schemes of the posterior parietal cortex. Panel A: Brodmann's (1905) subdivisions of the monkey cortex (*Cercopithecus*). Panel B: von Bonin and Bailey's (1947) classification of the monkey cortex (*Macaca mulatta*). Panel C: Brodmann's (1907) parcellation of the human cortex. Panel D: von Economo's (1929) parcellation of the human posterior parietal cortex. From Andersen, 1987.

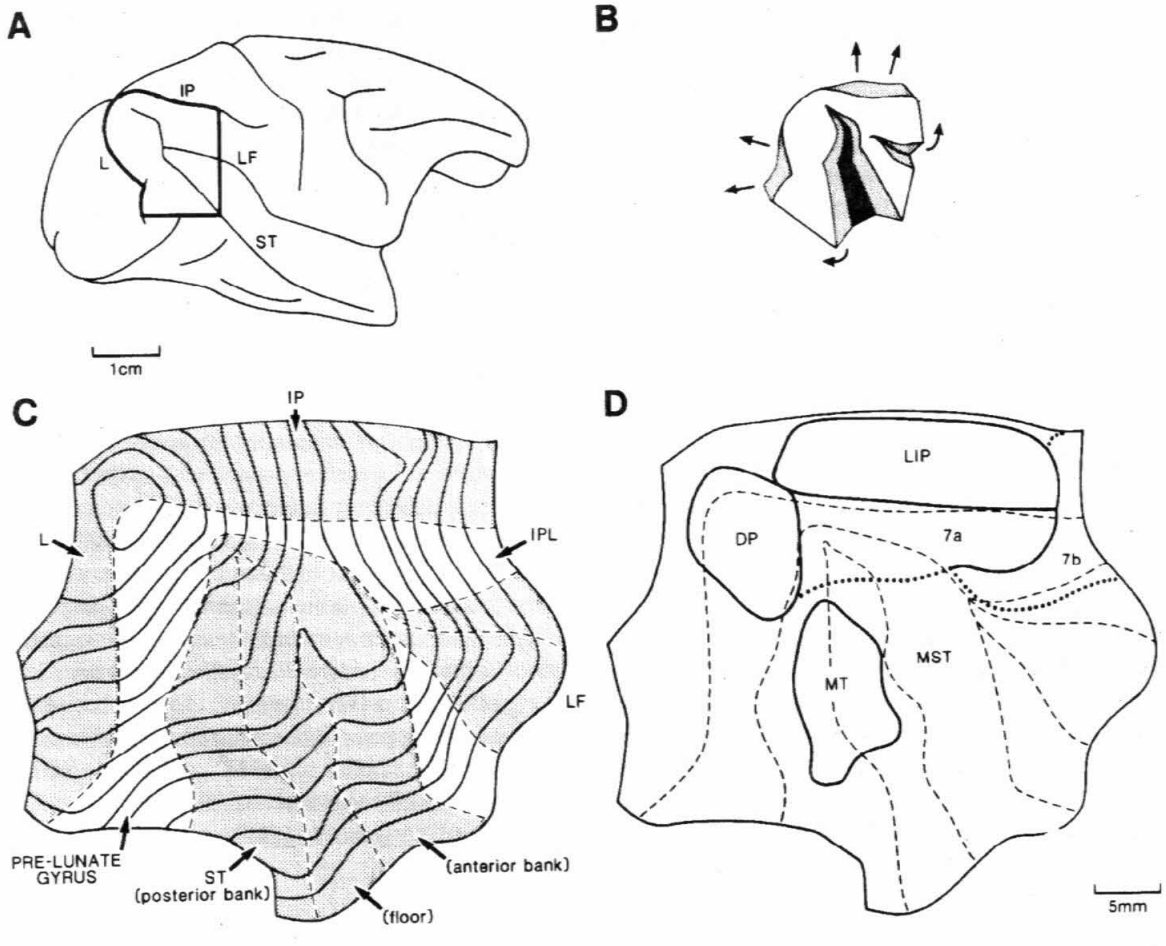


Fig. 3. Parcellation of the inferior parietal lobule and adjoining aspect of the prelunate gyrus based on physiological, connectional, myeloarchitectural and cytoarchitectural criteria. Panel A is a lateral view of the monkey hemisphere with the darker lines outlining the area of cortex that is flattened. Panel B shows this area of cortex removed from the rest of the brain. The stippled areas represent cortex buried in sulci and the blackened area the floor of the superior temporal sulcus. The arrows schematize the movement of local cortical regions that would result from mechanically flattening this cortical block. Panel C shows a completely flattened representation of the area. The stippled areas show cortical regions lying buried within sulci. The series of contour lines are tracings of layer IV taken from frontal sections through this area that are used in the reconstruction. Panel D shows the locations of different cortical areas on this reconstruction. The dotted lines indicate borders of cortical fields that are not precisely determinable. Abbreviations: L, lunate sulcus; IP, intraparietal sulcus; ST, superior temporal sulcus; LF, lateral fissure; DP, the dorsal prelunate area; LIP, the lateral intraparietal area; MST, the medial superior temporal area; MT, the middle temporal area. From Andersen, 1987.

comprise the inferior parietal lobule in man (Fig. 2B,D). The fact that lesions of the inferior parietal lobule in monkey and man produce similar visual disorders whereas superior parietal lobule lesions in monkey and man generally result in somatosensory disorders argues for von Bonin and Bailey's view.

The inferior parietal lobule has been found to contain at least six distinct subdivisions based on differences in cortico-cortical connections, subcortical connections, and functional differences. Fig. 3 shows a flattened reconstruction of the cortex of the inferior parietal lobule and adjacent prelunate gy-

rus. The various cortical areas of the inferior parietal lobule are indicated on this map.

4.1. Area 7b

A majority of the cells in area 7b respond to somatosensory stimuli (Hyvarinen and Shelepin, 1979; Robinson and Burton, 1980a,b; Hyvarinen, 1981; Andersen, 1985b). Robinson and Burton (1980a,b) found that although the receptive fields of the cells were very large, there was a crude topographic arrangement of the body in area 7b. Cells responsive to reaching and hand manipulation have also been recorded from this area (Mountcastle et al., 1975; Andersen et al., 1985b). A small minority (10%) of the cells in this region have also been reported to respond to visual or visual and somatosensory stimuli (Robinson and Burton, 1980a,b; Hyvarinen, 1981). Not surprisingly, area 7b has cortico-cortical connections primarily with other areas involved in somatosensory processing. These areas include the insular cortex, area SII and area 5 (Andersen, 1987). Area 7b also receives its primary thalamic input from the oral subdivision of the pulvinar (Asanuma et al., 1985). This thalamic nucleus also connects to other somatosensory areas such as area 5.

4.2. Area LIP

The lateral intraparietal area is located in the lateral bank of the intraparietal sulcus and appears to play a role in saccadic eye movements. Shibutani and colleagues (1984) have reported lower thresholds for evoking saccades with electrical stimulation to this area than to other regions in the posterior parietal cortex, although these currents were still rather high when compared to those required to evoke eye movements from the frontal eye field or superior colliculus. Andersen et al. (1985b) have reported finding many more saccade-related neurons in this area than in area 7a. Area LIP provides a much stronger projection than area 7a to the frontal eye fields and superior colliculus (Barbas and Mesulam, 1981; Asanuma et al., 1985; Lynch et al., 1985); both are structures involved in the generation of saccades. This area receives inputs from several extrastriate cortical areas, including the middle temporal area, a cortical field implicated in visual motion processing.

4.3. Area MST

The medial superior temporal area (MST) appears to be specialized for motion analysis and smooth pursuit eye movements. Sakata and colleagues (1983) and Wurtz and Newsome (1985) have found cells responding during smooth pursuit to be located primarily in this area. Many cells in this area are sensitive to relative motion, responding to such parameters as size change and rotation (Sakata et al., 1985; Saito et al., 1985). Lesions to MST produce deficits in smooth pursuit eye movements. The speed of initiating smooth pursuit to match target speed is underestimated and the maintenance of pursuit is defective for tracking toward the side of the lesion (Wurtz et al., 1986). Area MST receives direct projections from several extrastriate visual areas, including area MT, and it projects to area 7a and LIP (Maunsell and Van Essen, 1983; Seltzer and Pandya, 1984; Colby et al., 1985; Siegel et al., 1985).

4.4. Area 7a

Area 7a appears to play a role in the integration of eye position and retinotopic visual information for spatial analysis. A majority of the cells studied in this area have visual receptive fields (Hyvarinen, 1981; Motter and Mountcastle, 1981; Andersen et al., 1987). Many of these cells also carry eye position signals and some cells have saccade-related activity (Andersen et al., 1987). For many neurons visual excitability changes as a function of the angle of gaze. This gating of visual signals by eye position produces a tuning for locations in head-centered space (Andersen et al., 1985c).

Area 7a has more extensive connections with high-order areas in the frontal and temporal lobes and the cingulate gyrus than do the other areas in

the posterior parietal cortex. It projects strongly to the prefrontal cortex in and around the principal sulcus (area 46 of Walker) but, unlike area LIP, is only weakly connected to the frontal eye fields (Barbas et al., 1981; Andersen, 1987). It has very strong interconnections with the entire cingulate gyrus, with the most dense connections being to area LC in the posterior half of the gyrus (Pandya et al., 1981; Andersen, 1987). Area 7b is connected primarily, if not exclusively, to area LA in the anterior aspect of the cingulate gyrus (Andersen, 1987). Area 7a also demonstrates the most extensive connections of the posterior parietal areas with the cortex buried in the superior temporal sulcus, including area MST (Andersen, 1987).

Additional visual areas defined by differences in connections include the ventral intraparietal area (VIP) located in the fundus of the intraparietal sulcus (Maunsell and Van Essen, 1983) and the dorsal prelunate area on the prelunate gyrus above area V4 (Andersen et al., 1985b). The functional properties of neurons in these areas have not been explored.

The above observations indicate that the inferior parietal lobule can be subdivided into a largely somatosensory area more or less coextensive with PF, and several visual areas within PG.

5. Physiology

5.1. The command hypothesis

The earliest single unit recording experiments in the posterior parietal cortex in behaving monkeys in conjunction with eye movement recordings were made by Vernon Mountcastle and his colleagues (Mountcastle et al., 1975; Lynch et al., 1977). These pioneering studies found area 7 neurons to be activated by visual and somatosensory stimuli, by oculomotor behaviors including fixation and eye movements, and by the animal's reaching for objects and manipulating them. They reported that these oculomotor cells did not respond to visual stimuli and that the saccade and tracking neurons were activated before the behavior.

Mountcastle and colleagues reported that the saccade cells were active only when the animal made purposeful movements (such as following the jump of the fixation target in the behavioral task) but were not active when the animal made 'spontaneous' saccades (Mountcastle et al., 1975; Lynch et al., 1977). The activity was reported to precede the actual eye movement by an average of 55 ms. Saccade cells were found to be selective for the direction of the saccade but not the amplitude. Some saccade cells also showed positional dependence, with saccadic activity varying for saccades of the same amplitude and direction but initiated from different orbital positions (Yin and Mountcastle, 1978).

Mountcastle and colleagues proposed that the posterior parietal cortex contained a command apparatus for issuing commands for exploratory motor activity within the extrapersonal space (Mountcastle et al., 1975). This command hypothesis was consistent with their observations that the oculomotor, reach and hand manipulation neurons had behaviorally related responses, that their activity often preceded the behavior, that the cells were not activated by sensory stimuli, and that the activity of the neurons depended upon the internal drives that initiated the behaviors (i.e. were not active for spontaneous saccades). The commands were believed to be general in nature; for instance, the saccade cells did not specify with much precision the direction and amplitude of the saccades; these details of the movement were presumably elaborated by structures efferent to the posterior parietal cortex. It was proposed that many of the deficits following posterior parietal lesions in monkeys and humans, such as the reluctance to use the contralateral limb, difficulty in disengaging fixations, instabilities in fixations, and deficits in saccadic and smooth pursuit eye movements, could be accounted for by the loss of the oculomotor command neurons.

5.2. Saccade enhancement

In later experiments Robinson, Goldberg and Stanton (1978) found that many of the cells in area 7 did,

in fact, respond to visual or somatic stimuli. They argued that the behaviorally related responses reported by Mountcastle and his colleagues could be accounted for either by visual stimulation from the target for movement or from visual/somatosensory stimulation resulting from the movement. It was proposed that area 7a was involved in sensory processes including selective attention and did not play a role in motor behavior as proposed by Mountcastle and colleagues.

Yin and Mountcastle (1977, 1978) and Robinson et al. (1978) found that light-sensitive cells of the inferior parietal lobule increased their visual response if the stimulus served as a target for a saccade. This enhancement in light sensitivity occurred only when the saccade target was within the visual receptive field of the neuron, indicating that it was not a result of generalized arousal. The enhancement effect has been thoroughly studied in many areas of the primate brain by Wurtz, Goldberg, Robinson and colleagues, and is reviewed by Robinson and McClurkin in this volume (chapter 9). The major distinguishing feature of enhancement for parietal lobe neurons is that whereas saccades must be made for enhancement to occur in several oculomotor areas, in IPL it is sufficient for the animal simply to attend to the visual target and, for instance, detect its dimming (Bushnell et al., 1981). These observations are consistent with the posterior parietal cortex playing an important role in selective attention.

5.3. Visual-motor integration

Studies since those of Mountcastle and colleagues and Robinson, Goldberg and Stanton have established that the inferior parietal lobe neurons have both motor-related and sensory-related responses. This has proven to be the case for smooth pursuit (Sakata et al., 1983; Kawano et al., 1984; Wurtz and Newsome, 1985), fixation (Sakata, et al., 1980; Andersen et al., 1987) and reach movements (Bioulac and Lamane, 1979; Kalaska et al., 1983; Chapman et al., 1984; Seale et al., 1985), as well as saccadic movements (Andersen et al., 1987). In general,

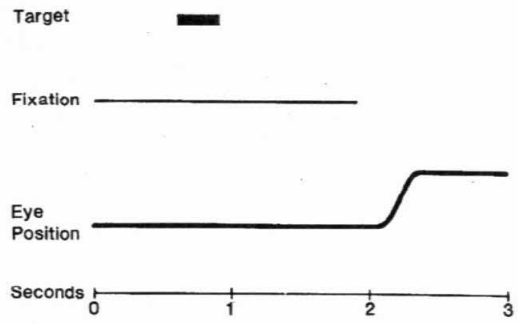
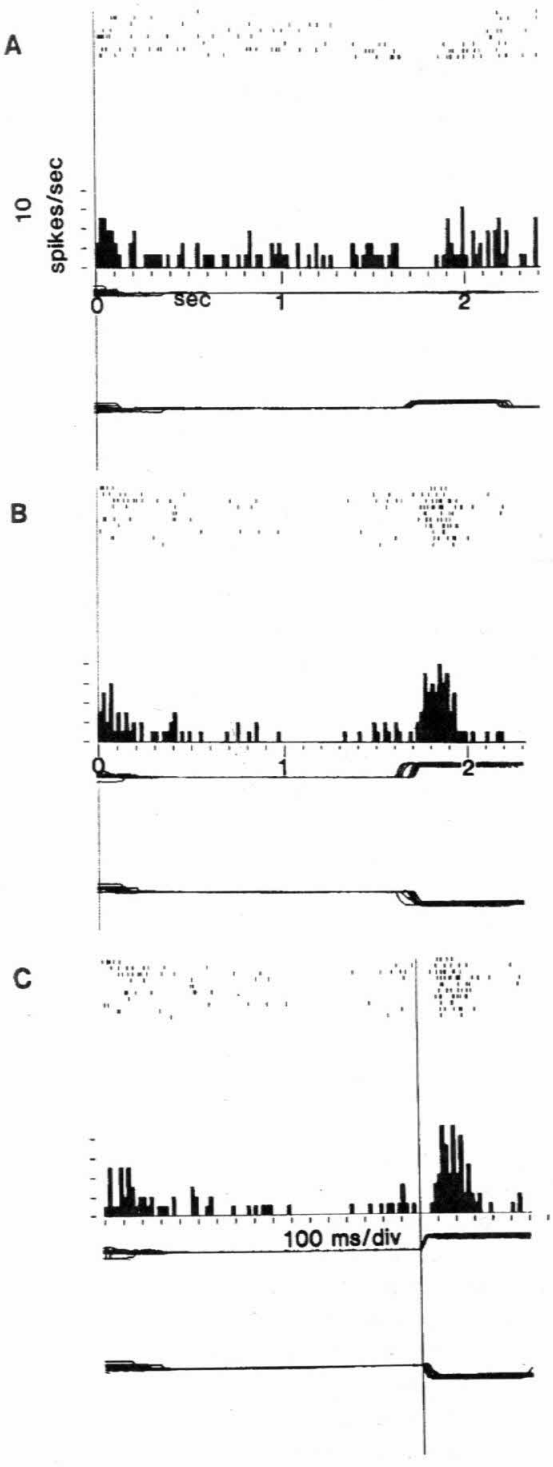


Fig. 4. Diagram of the sequence of stimulus events in the memory saccade task.

these experiments utilize protocols designed to separate the sensory and motor components of a cell's activity. An example of such a protocol for studying saccades is described below (from Gnadt and Andersen, 1988).

Visual stimulation can result from stimulation by the target for the saccade or by the movement of the contours of the visual background across the retina as the eyes move. To prevent these visual contaminants from possible contribution to cell responses, animals were trained to make saccadic eye movements to remembered locations in total darkness. Fig. 4 shows the sequence of stimulus events used in such tasks. Eight hundred milliseconds after the animal looked at a fixation target a saccade target was flashed peripherally for 200 or 300 ms. The fixation target was extinguished a variable time 500 to 1500 ms after the offset of the saccade target, and the animal then made the eye movement in complete darkness to the location where the second target had been. Fig. 5 shows a representative example of recordings in this task in which the animal was required to remember the stimulus location for 700 ms. As indicated in panels B and C, the cell showed a response related to the saccade. In Fig. 5C the spike rasters are aligned to the beginning of the eye movement rather than to the stimulus events as in Fig. 5B, indicating that the bursts of activity are better correlated with the eye movements. In the final control (Fig. 5A) the animal performed a task in which it maintained fixation at the same spatial locus when the fixation light was turned off for one



second (there is a dark drift upward which is common in monkeys with fixed heads). This control demonstrates that the saccade-related response was not due to the offset of the fixation target.

The convergence of sensory and motor-related responses suggests that the posterior parietal cortex plays an important role in sensorimotor integration. Andersen (1987) has proposed that the posterior parietal cortex should not be considered exclusively motor or sensory in nature, but rather an interface between sensory and motor systems for accomplishing motor movements under sensory guidance. Clinical evidence supports this view, since lesions to the posterior parietal cortex produce both sensory-perceptual and motor disturbances. One aspect of such a role in sensorimotor integration is likely to be the transformation of incoming sensory coordinate frames to spatial and motor coordinate frames that are necessary for accurate motor behavior under sensory guidance. Consistent with this proposal is the observation that visual cells in area 7a are tuned to locations in head-centered space by integrating both eye position signals and retinotopically coded visual signals. Also consistent with this idea are the spatial and visual disorientation deficits seen with posterior parietal lesions and reviewed above.

The fact that most of the cells in the inferior parietal lobule can be vigorously driven by sensory stimuli without generating movements would tend

Fig. 5. Task for making saccades to remembered locations in total darkness. (A) Control in which the fixation point is turned off 1500 ms into the trial and the animal must maintain fixation at that location in space for 500 ms. The lack of activity when the fixation point is turned off and the animal withholds the saccade indicates that the cell is not responding to the offset of the fixation point. The horizontal and vertical eye position traces are located below the histogram. Note that there is a small vertical drift when the fixation light is extinguished, which is common for monkeys under these conditions. (B) The target was flashed on for 200 ms at 800 ms into the trial and the command to make the saccade (offset of the fixation point) occurred at 1500 ms. The spike rasters and histogram are aligned to the stimulus events. (C) The same spike rasters as in B are now aligned to the beginning of the eye movements.

TABLE 1

Area 7a (241 cells)		**Area LIP (141 cells)**	
Visual	80% (194)	Visual	66% (93)
Visual only	12% (30)	Eye position only	11% (15)
Eye position only	16% (38)	Saccade-related	48% (67)
Visual and eye position	53% (127)		
Saccade-related	19% (46)		
Area 7a saccade-related (46 cells)		**Area LIP saccade-related (67 cells)**	
Saccade and visual	80% (37)	Saccade and visual	15% (10)
Saccade only	20% (9)	Saccade only	49% (33)
Pre-saccadic	11% (5)	Pre-saccadic	43% (29)
Post-saccadic	89% (41)	Post-saccadic	57% (38)
Intended movement	(not tested)	Intended movement	36% (24)

to argue against their issuing the commands for movement as originally formulated by Mountcastle and colleagues. However, as Lynch (1980) has pointed out, primary motor cortex neurons which are generally believed to issue commands for motor movements also respond to somatosensory stimuli; thus the presence of sensory activity does not in itself rule out a motor command function. However, unlike motor cortex in which movement-related responses usually precede movements, the responses of the oculomotor neurons in the inferior parietal lobule often follow the initiation of movement, at least for the fixation neurons and many of the saccade neurons in area 7a (latencies have not yet been published for the tracking cells) (Andersen et al., 1987). Although movement-related activity in area 5 often precedes limb movements, it still lags behind the movement-related activity of primary motor cortex, suggesting that it is an efference copy rather than a motor command (Kalaska et al., 1983; Lamarre et al., 1985). However, since many saccade cells in area LIP do respond before the initiation of eye movements, and some of these cells have no discernible visual receptive fields, the possibility that there is a subset of command cells in the posterior parietal cortex for saccades cannot be ruled out. However, since these cells have rather enormous motor fields and are rather poorly temporally linked to the movement, it would seem more probable that these cells are involved in earlier stages of processing that will later become commands to move the eyes in other parts of the nervous system.

It is also not appropriate to consider this area as largely sensory in function, especially considering recent findings that the saccade neurons in area LIP code saccades in motor coordinates (Gnadt and Andersen, 1987). Also, the finding that cells in this area have memory-related activity and code the amplitude and direction of intended eye movements indicates that the area plays a role in motor planning. These findings are discussed in the next section.

5.4. Area LIP

As outlined above, based on electrical stimulation and anatomical experiments area LIP appears to be a likely candidate for a cortical field within the inferior parietal lobule that plays a role in the processing of saccades. Recent recording experiments in this area show that only areas LIP and 7a have saccade-related responses. About half of the neurons in LIP sampled had saccade-related responses whereas only 19% of area 7a neurons had saccade-related activity (Table 1) (Gnadt and Andersen, 1988; Andersen et al., 1987). More importantly, 43% of area LIP neurons with saccade activity be-

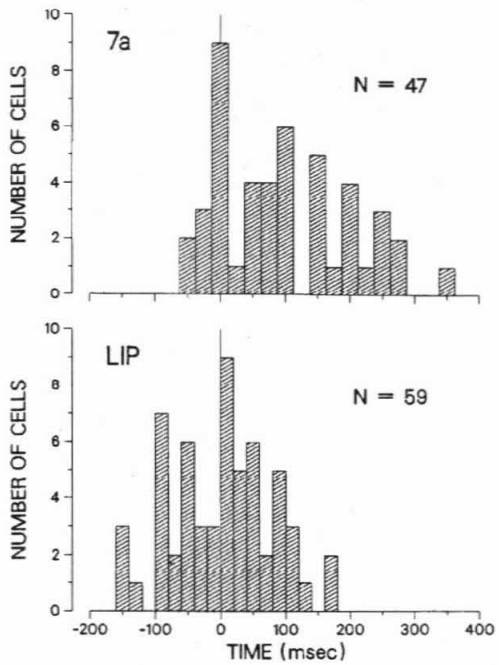


Fig. 6. Histograms of latency of saccade responses. About one-half of area LIP saccade-related neurons fired before the eye movement, whereas most area 7a cells fired after the eye movement. All cells were tested in the memory saccade task that ruled out the responses being related to the sensory stimulus; ie. all activity is saccade-related.

gan firing before the eye movement whereas only 11% of the area 7a sample showed presaccadic activity (Table 1, Fig. 6).

Table 1 shows the breakdown of cell types in area 7a and LIP recorded by Andersen and colleagues (Andersen et al., 1987; Gnadt and Andersen, 1988) using the same protocols so the two areas could be directly compared. A crucial feature to consider is that most of the cell properties listed occur as gradients within the cell populations of areas 7a and LIP. For instance, cells are found with eye-position-only or visual-only responses, but a majority of the cells in area 7a receive both visual and eye position inputs of varying amounts. Also many of the properties listed represent intersecting subsets and a cell may exist in more than one category. For instance, an LIP neuron may respond to visual stimuli and be listed as *visual*, but also have a presaccadic eye-movement-related response.

An interesting issue that has recently been investigated is the possible role of area LIP in the spatial transformations required for the encoding of saccades based on visual signals. There is now considerable evidence that saccades are programmed with a knowledge of eye position (Robinson, 1975; Hallet and Lightstone, 1976; Hansa and Skavenski, 1977; Mays and Sparks, 1980). It has been proposed by these investigators that the retinal location of the visual target can be programmed with respect to eye position, resulting in a head-centered coordinate frame. Later in the processing chain the current eye position is believed to be subtracted from the head-centered position of the desired goal to produce a motor error signal, the amplitude and direction of the intended eye movement. This scheme presents two problems–the transormation of the location of targets from retinotopic coordinates to spatial coordinates and the transformation of spatial coordinate-encoded signals to motor coordinates of the eye movement. The posterior parietal cortex is a likely area for at least the first and possibly both operations. The angle-of-gaze effects on light sensitivity recorded from area 7a neurons produce a coding of visual stimuli in head-centered coordinate space (Andersen et al., 1985c). Yin and Mountcastle (1978) have noted an orbital position dependence for saccade-related activity of posterior parietal neurons, and more recently Gnadt and Andersen (1986) have studied in detail the effect of eye position on the encoding of saccades by area LIP neurons.

Fig. 7 shows an example of the orbital position effect on saccade responses of area LIP neurons. The histograms show the saccade-related responses for eye movements made to remembered target locations in the dark. The saccades are all of 15 degrees amplitude and directed downward; however, they are initiated from 12 different orbital postions. Note that the cell is most responsive for saccades beginning from 15 degrees down from straight ahead, whereas for saccades made from upper orbital positions the cell does not respond at all. The

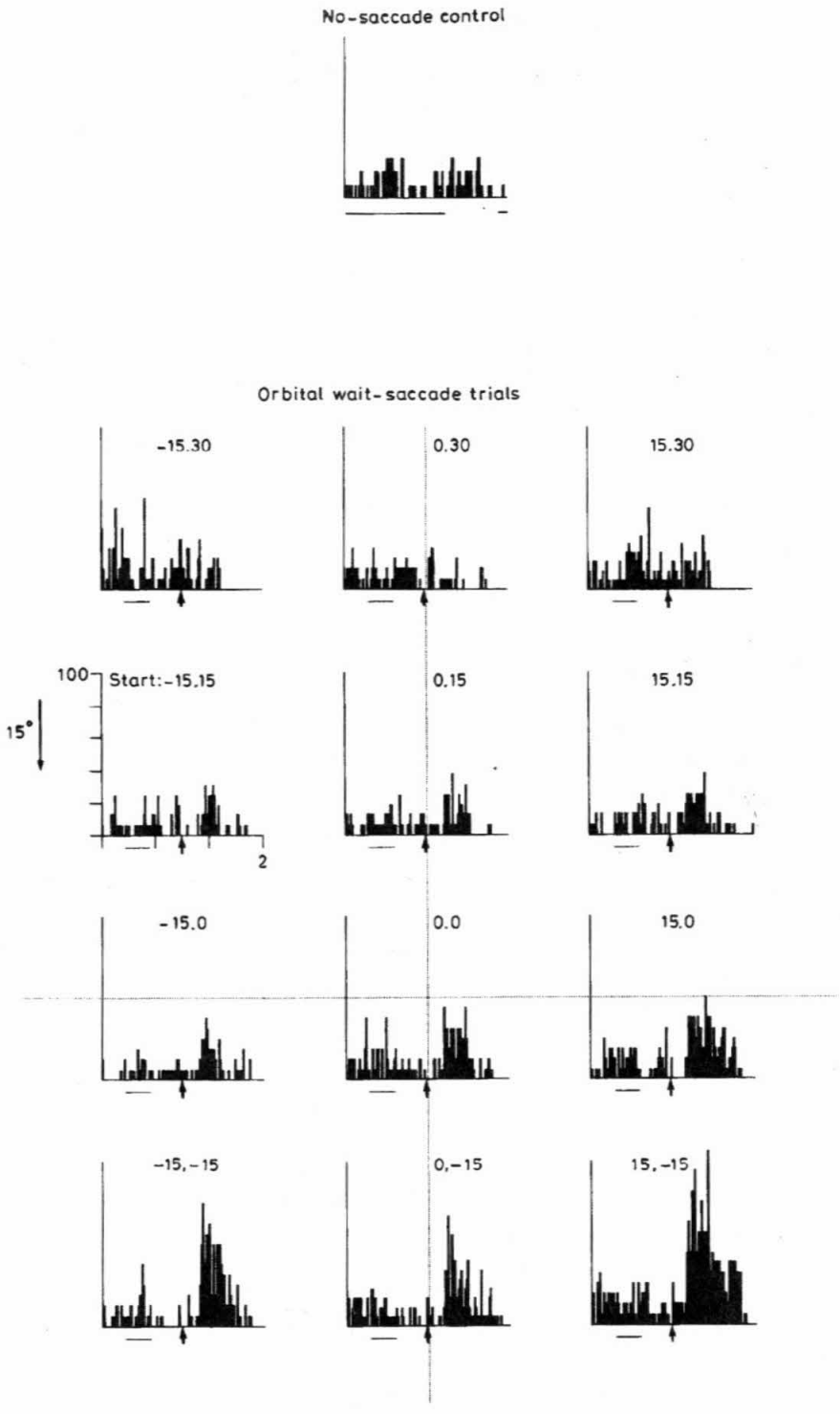


Fig. 7. Neural responses for saccades of equal direction and amplitude but initiated from different orbital positions. This cell responds best for eye movements of 15 degrees amplitude in the down direction when the animal initiates the saccade from downward angles of gaze. The arrow represents the time of offset of the fixation target. Increased activity precedes the saccade.

orbital position dependence is a common feature of area LIP neurons, occurring in a majority of the cells sampled that had saccade-related activity.

The orbital position dependence of the saccade responses of LIP neurons appears to be the result of one of two possible spatial encoding mechanisms. In the first the cells are encoding the goal of the saccade in head-centered coordinate space. In this circumstance the neuron will fire regardless of the direction or amplitude of the eye movement, as long as the outcome of the eye movement is a specific orbital position. Thus such an encoding of spatial position is eye-position-independent. In the second possibility, the cells would always fire for a saccade of a specified direction or amplitude; however, the magnitude of the response would vary as a function of eye position. This gating by eye position would still tune a cell for a best response at a particular location in head-centered space which would be the combination of the best eye position and the center of the motor field of the neuron; however, this tuning is eye-position-dependent.

A double eye movement task modified from Hallet and Lightstone (1976) and Mays and Sparks (1980) was used by Gnadt and Andersen (1986) to decide between these two possibilities. This task is diagrammed in Fig. 8. The animal first fixates a fixation stimulus in otherwise total darkness. Two targets are flashed sequentially on the screen for 60 ms beginning at the offset of this fixation target. Since reaction times for saccades are approximately 180 ms under these conditions, the first eye movement begins after the offset of the target. Fig. 8B shows that the first eye movement is made using only information derived from the retinal location of the stimulus (vector A). However, the second eye movement (vector C) must be calculated from both the retinal location of the flashed stimulus (vector B) and the change in eye position (vector A). If only retinal information is used then a spatially incorrect saccade of vector B′ is made. Since the animals make eye movements of vector C instead of vector B′ the saccade system must calculate the second eye movement by transforming to a head-centered coordinate space. An encoding using eye position and

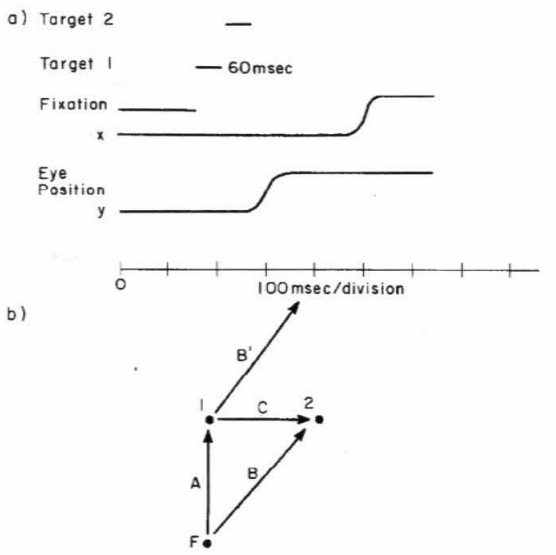


Fig. 8. (A) Diagram of the sequence of events in the double saccade task. (B) Eye movements in the double saccade task will be between A and C if the second saccade is programmed spatially and A and B′ if it is programmed retinotopically using only retinal vector B.

retinal position is equivalent to the encoding of the goal in head-centered coordinates.

In the experiments of Gnadt and Andersen (1986) the second eye movement was always made to the same location in space but from different directions. If LIP neurons are encoding spatial location in a goal-directed, eye-position-independent fashion then the cell will fire regardless of the direction of the second eye movement. However, if the encoding is eye-position-dependent then the cell will fire only when the saccade is made in its preferred direction, regardless of whether it is the first or second saccade. Fig. 9 shows an example of the activity of LIP neurons in this task. The cell discharged for leftward eye movements only, regardless of whether they were the first or second saccade. Area LIP saccade cells have motor fields that are oculocentric. Thus LIP neurons encode spatial location by an eye-position-dependent mechanism that is formally identical to that seen for the encoding of spatial location of sensory targets by area 7a neurons. The response fields in both cases are

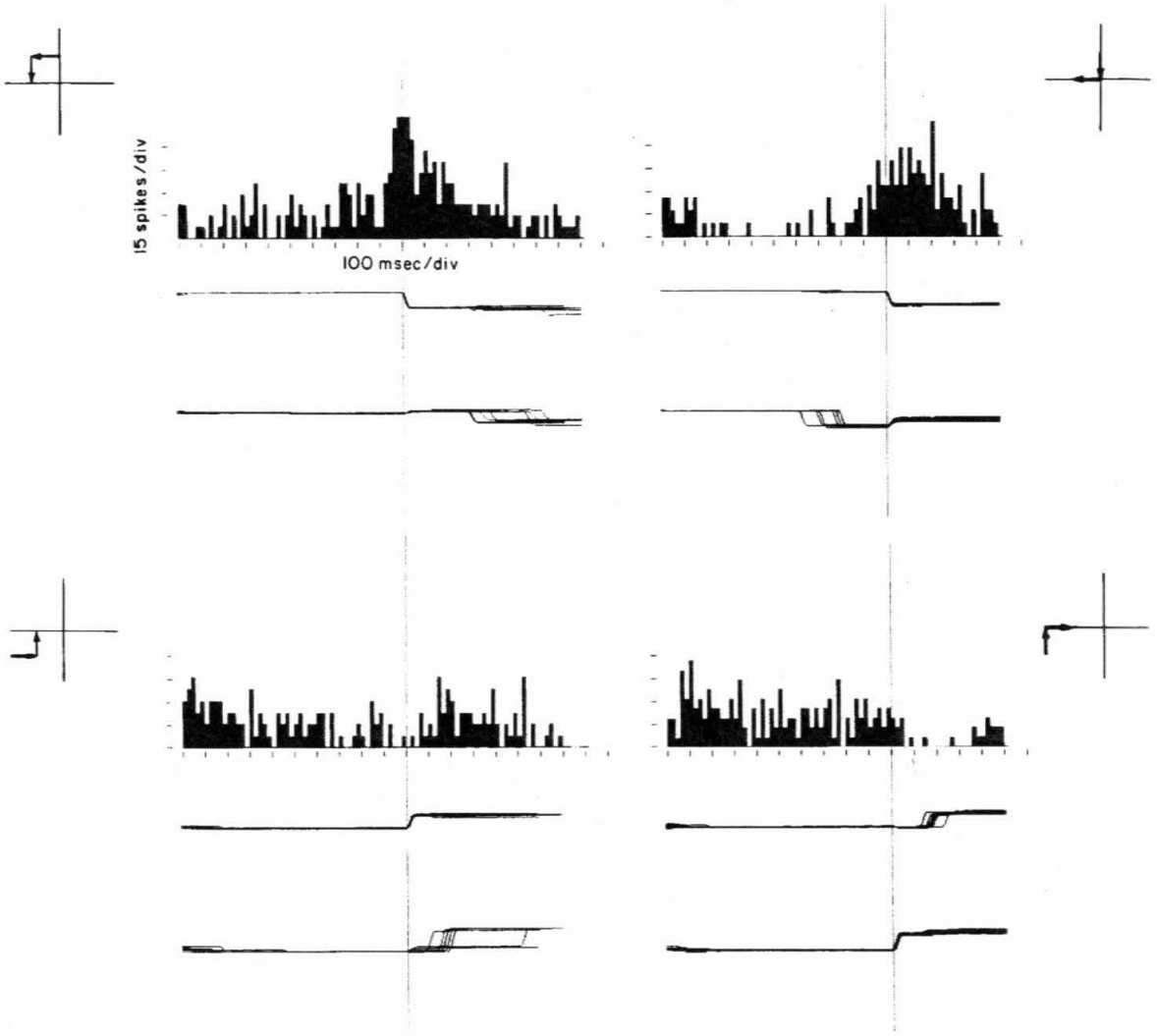


Fig. 9. Double saccades were made to the same location 15 degrees to the left of straight ahead. The cell is active for either the first (retinotopic) saccade or the second (spatial) saccade if the movement is made to the left.

oculocentric and the magnitude of the response varies as a function of eye position.

The results of the double saccade task of Fig. 9 have two additional and important implications. The first is that for the second saccade to be executed the brain must make spatial transformations. Thus it cannot be argued that a goal-directed, eye-position-independent spatial coding was not seen at a single cell level because the animal was not required to do a task requiring such a transformation. Secondly, this experiment demonstrates that the LIP neurons are encoding saccades in motor coordinates. The cell in Fig. 9 has a restricted response field centered on 15 degrees to the left. For the trials in the upper right panel, neither the first nor the second target light ever appeared in the response field of the cell yet the neuron still became active prior to the 15-degree leftward saccade. Thus the cell is encoding the direction and amplitude of the eye movement and not the retinotopic location

of the visual target; that is, the cell is coding in motor coordinates and not sensory coordinates.

5.5. *Motor planning activity in area LIP*

Recently a class of neurons has been found in area LIP whose responses appear to represent a memory-related motor-planning signal encoding motor error for saccadic eye movements (Gnadt and Andersen, 1988). The temporal relation of the cell's activity to the behavior of the animal suggests that the response represents the intent to make eye movements of specific direction and amplitude. These cells have been labeled intended movement neurons (Gnadt and Andersen, 1988). These cells appear to be visual cells by many routine criteria. They did respond phasically and less vigorously to visual stimuli in trials that did not require eye movements. However, if the animal is required to make an eye movement, but withholds the response for a delay period, these cells show a maintained high rate of activity throughout the delay period. Fig. 10 shows the response of one of these cells during eight different delay periods using the memory saccade task described earlier and diagrammed in Fig. 4. The cell began to respond within 100 ms of the onset of a visual stimulus within its response field. It continued to discharge after the 300-ms duration stimulus was extinguished until the eye movement was made, regardless of whether the movement occurred within a few hundred milliseconds or was withheld for more than 1.5 seconds. Fig. 11 shows the tuning of the activity of an intended movement cell for saccades in several radial directions and for several lengths along the preferred direction. This cell is tuned for leftward saccades of about 15 degrees. The broad tuning for both direction and amplitude is typical for these neurons. Fig. 12 shows the activity of an intended movement neuron in a modified version of the double saccade task. The cell has a response field centered 20 degrees above fixation. In Fig. 12A the animal is required to withhold the upward saccade for 700 ms to demonstrate the memory-linked activity. This cell also gives a phasic saccade-related

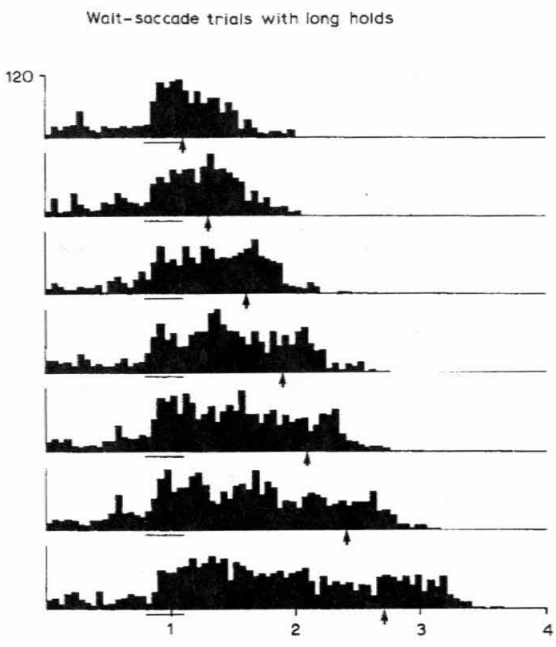


Fig. 10. Activity histograms of an intended movement cell during saccades to a remembered target location in the cell's motor field. Trials are grouped according to increasing response delay times from top to bottom. The horizontal bar below each histogram indicates the stimulus presentation time. The arrow indicates when the fixation light was extinguished commanding the saccade. From Gnadt and Andersen, 1988.

burst of activity which is not uncommon for this class of neurons. In Fig. 12B the animal performs the double-movement task with the first eye movement and flashed target occurring in the response field of the cell. As would be predicted the cell responds for the upward movement. In the task in Fig. 12C the animal first makes an eye movement down to the location of the flashed light and then back up to the remembered location of the extinguished fixation point. The rasters and histograms are aligned on the first eye movement. Note that the activity of the neuron increased as soon as the monkey achieved the first target and was in a position to make the next saccade into the cell's receptive field. Thus the cell could not be coding the signal in sensory-related retinotopic coordinates. Also, since the upward saccades in panels B and C

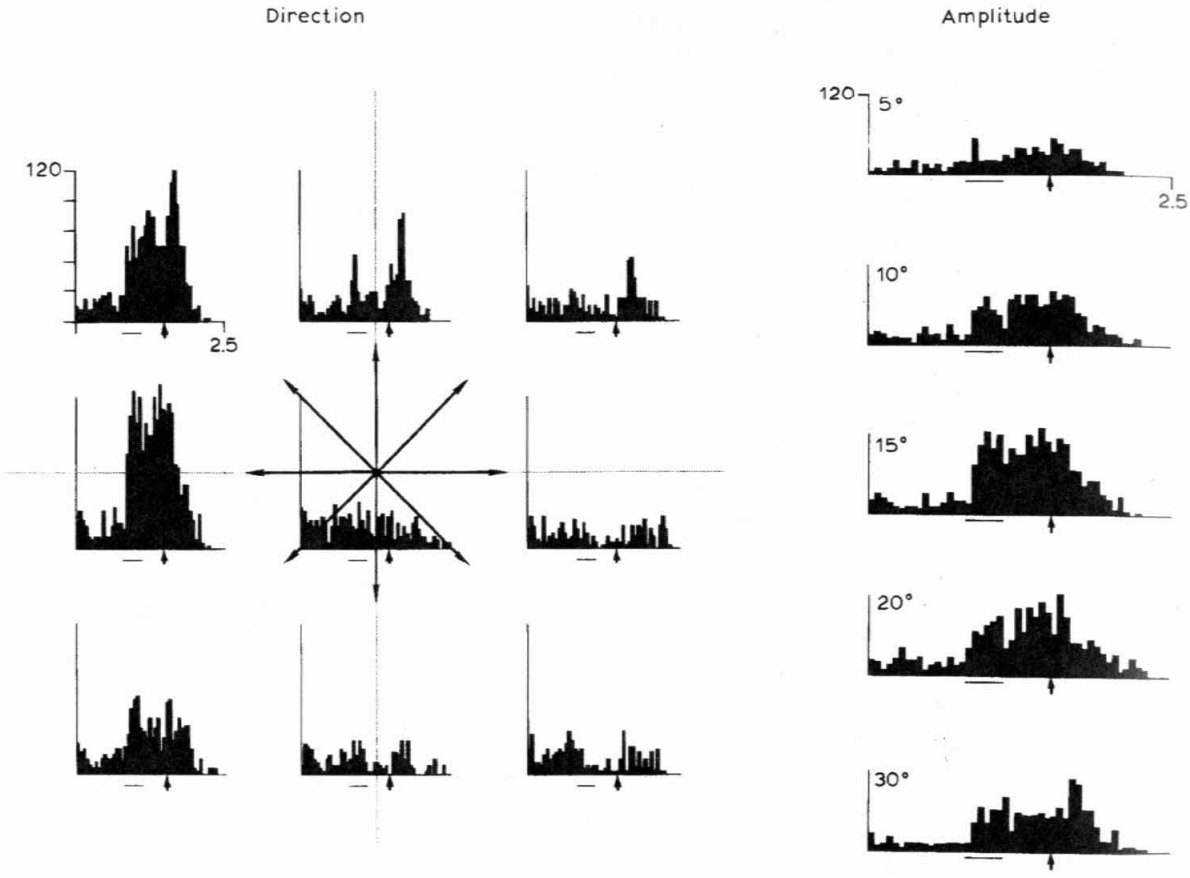


Fig. 11. Direction and amplitude tuning of an intended movement cell. Horizontal bars and arrows below each histogram represent stimulus presentation and fixation spot offset, respectively. The radial directions were tested to targets located on a 15 x 15 degree grid in the directions indicated. The middle panel of the direction figure shows trials in which no target was presented and the monkey was trained to maintain fixation while the fixation spot blinked out and then back on again (arrow-heads). The indicated target amplitudes were tested in the direction of straight left.

are made to different spatial locations, the cell could not be encoding the location of the sensory stimulus in head-centered coordinates. Therefore, the cell is encoding in motor coordinates and the memory activity represents the intended trajectory of the impending saccade.

There has been some indication from the lesion literature that the posterior parietal cortex plays a role in motor intention. Valenstein et al. (1982) have presented behavioral evidence that the characteristic hemi-neglect syndrome produced by parietal lesions can be explained as a deficit of movement intention rather than sensory neglect. These investigators trained monkeys to respond manually using the arm contralateral to the stimulus cue. They found that posterior parietal lesions produced deficits related to reaching with the contralateral arm regardless of which side of the body the cue was delivered to and no deficit for responding to cues on either side of the body with the ipsilateral limb. They interpreted their results as a deficit in the intent to make a movement with the contralateral limb. Also, the ocular motor apraxia and defective visual search of Balint's syndrome could be in part due to the loss of neural elements responsible for the planning and retention of intended movements.

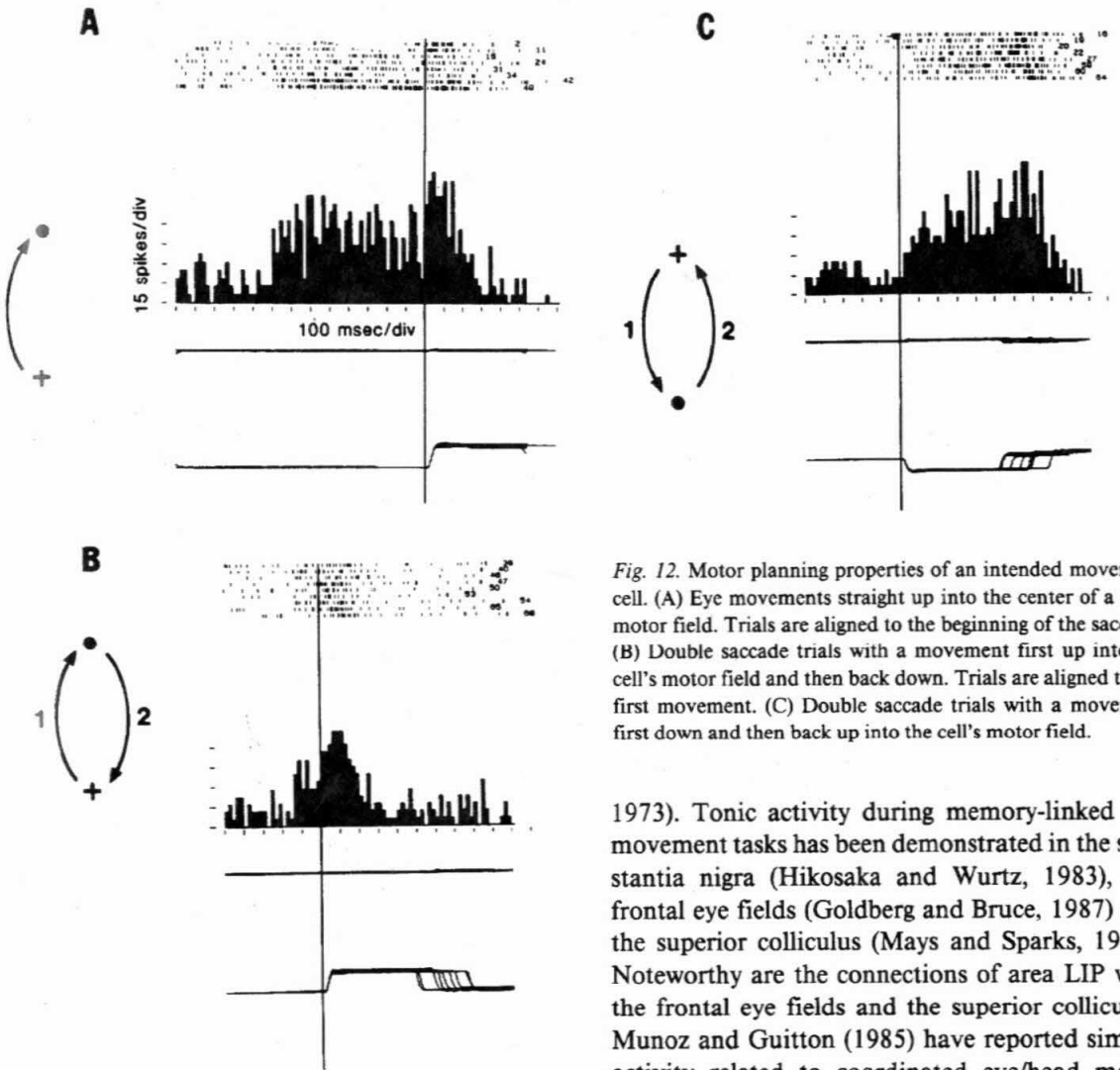


Fig. 12. Motor planning properties of an intended movement cell. (A) Eye movements straight up into the center of a cell's motor field. Trials are aligned to the beginning of the saccade. (B) Double saccade trials with a movement first up into the cell's motor field and then back down. Trials are aligned to the first movement. (C) Double saccade trials with a movement first down and then back up into the cell's motor field.

Other laboratories have described elevated tonic neural activity during the delay period of memory-linked delayed response tasks. Among the areas where such responses have been described for arm movements are prefrontal cortex (Niki, 1974; Sakai, 1974; Niki and Watanabe, 1976, 1979; Watanabe, 1986a,b), cingulate cortex (Niki and Watanabe, 1979), inferotemporal cortex (Fuster and Jervey, 1982), hippocampus (Watanabe and Niki, 1985) and thalamus (Fuster and Alexander, 1973). Tonic activity during memory-linked eye movement tasks has been demonstrated in the substantia nigra (Hikosaka and Wurtz, 1983), the frontal eye fields (Goldberg and Bruce, 1987) and the superior colliculus (Mays and Sparks, 1980). Noteworthy are the connections of area LIP with the frontal eye fields and the superior colliculus. Munoz and Guitton (1985) have reported similar activity related to coordinated eye/head movements in the tectum of cats.

It has been suggested that this memory-linked activity may be the substrate for short-term memory (Fuster and Alexander, 1971; Watanabe and Niki, 1985) and/or may be involved in time estimation and temporal planning (Sakai, 1974; Niki and Watanabe, 1979; Fuster et al., 1982). A critical issue in the analysis of memory-related activity is the need to determine the specific parameters to which the activity is related. The data presented here indicate that the memory-linked activity we have seen

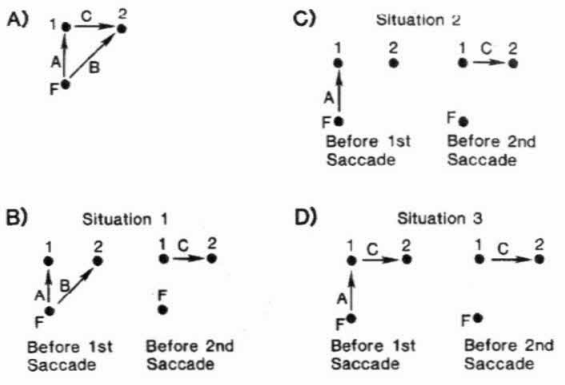


Fig. 13. Schematics of three possible ways in which area LIP can program double eye movements. (A) F, fixation point; 1, first saccade target; 2, second saccade target; A, B, and C are vectors that can be encoded by the motor fields of area LIP neurons. See text for explanation of panels (B), (C) and (D).

in area LIP is a motor or motor-planning signal.

Further investigation of the intended movement cells will help to determine the nature of memory representations and how motor programs are planned and sequenced. For instance, there are three possible dynamic mechanisms for the planning of intended movements by area LIP. These will be outlined for the double saccade task and with reference to Fig. 13. Possibility one is that both saccades of the double saccade task are mapped in motor coordinates based on retinal inputs and the change in eye position after the second saccade causes a recalculation for a new representation of the second movement vector. Fig. 13B shows the sequence of activity for this proposed mechanism. LIP cells with motor fields A and B become active prior to the first eye movement and cell B turns off and the cell with movement vector C turns on prior to the second eye movement. Possibility two is that only the next intended movement is represented in area LIP. Referring to Fig. 13C, the cell with movement vector A turns on before the first saccade and cell C turns on prior to the second eye movement. Possibility three is that both correct vectors are mapped immediately into area LIP. Fig. 13D illustrates that cells with vectors A and C turn on before the first eye movement and cell A turns off after the first eye movement.

The first proposal is a motor theory of memory in which the retinal location of the target is coded in motor space. The trajectory of the second saccade is updated based on the memory of the first eye movement. In this case all the memories are contained in area LIP. The current eye position is also found in fixation cells in area LIP; these cells are of course quite abundant in area 7a. Additional machinery for this mechanism includes a controller to point to the next eye movement and a neural mechanism for recalculating the vector of the second movement.

If the sequence of events occurs as outlined in situation 2 then there are two possible mechanisms. In the first mechanism, the spatial location coded in head-centered coordinates is held in memory. The calculation for the next saccade is made using the spatial location of the target and the current eye position. For this hypothesis the sensory memory must be coded in spatial coordinates. Such a spatial memory has not been found in area LIP and would have been apparent in the double movement tasks. Such a spatial memory may be located in area 7a. It would be interesting to determine whether the cells in area 7a showing spatial tuning hold spatial locations in memory register in double saccade tasks. A second possible mechanism is similar to a proposal by Goldberg and Bruce (1987) for the frontal eye fields; neurons in the frontal eye fields show a similar sequence of activity in double saccade experiments to that proposed in situation 2. Their model uses a vector subtraction of a feedback signal of the vector of the first eye movement from the retinotopic vector of the memory of the second target to calculate the second eye movement. This model requires a retinotopic memory, which does not appear in area LIP, as indicated by the special double saccade experiments of the previous section. The efference copy signal is present in area LIP. Finally both of these proposals under situation 2 require, as in situation 1, a controller for sequencing the eye movements and neural machinery for making the calculations.

Whereas the preliminary data are compatible

with situations 1 and 2, they do not appear to be compatible with situation 3. An example is shown in Fig. 12c, where the cell coding for vector C becomes active only after the first eye movement. In situation 3 cell C should become active before the first eye movement. Note that in situation 1 the memory is contained within area LIP and in situation 2 outside area LIP.

6. Conclusion

The literature on the posterior parietal cortex reviewed in this article indicates that it plays an intermediate role between sensory input and motor output in the processing of visually guided saccadic eye movements. Biparietal lesions in humans produce deficits in willed but not in spontaneous eye movements and the deficit is selective for visually evoked movements. Although not studied in great detail, lesions to monkeys appear to disrupt the performance of voluntary saccades. Recording experiments have established that neurons in the posterior parietal cortex have both sensory-related and motor-related responses. The behavior of the cells suggests that the area plays a role in visuo-motor integration. One important aspect of this integration appears to be the transformation of retinotopic visual signals to spatial and motor coordinate frames. These coordinate transformation functions are indicated both by the eye-position-dependent behavior of the visual and saccade-related responses and by the deficits in spatial orientation in monkeys and humans following lesion to this area. An important recent finding is the discovery of area LIP, the saccade area lying within the intraparietal sulcus. Furthermore, the finding that many of the neurons in this area have memory-linked activity which codes the amplitude and direction of intended eye movements indicates that information for motor planning should be considered an additional role of the posterior parietal cortex in saccades.

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