CEREBRAL POTENTIALS PRECEDING VISUALLY-TRIGGERED SACCADES

by

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Abstract

Brain activity associated with the generation of human saccadic eye movements was investigated in relation to the eliciting visual stimulus and the intended target of the saccade. The subject's task was to fixate a stimulus light presented randomly at left or right positions on a screen (normal task) or else to fixate the position at the opposite side (anti task). In two control conditions for the same tasks the stimulus light jumped to the opposite side when the subject initiated a saccade. Two additional control conditions consisted of no saccades for the same visual stimulation, and no visual stimulation but left and right saccades following verbal command from the experimenter.

Results showed no significant task or stimulus condition effect for saccade reaction time. The stimulus-evoked potential recorded from three midline scalp locations showed a prominent negative peak of 150-200 msec latency with maximum amplitude at vertex and minimum at the frontal location. The anti task produced significantly greater amplitudes at the central and parietal locations. Response-related data showed a positive-going potential (Pre-Motion Positivity, PMP) prior to saccade onset. The topography of the PMP was similar for all five movement conditions with a frontal minimum and a parietal maximum which was significantly greater for the normal condition. No effect of target movement was found for any dependent variable.

Interpretation of the reaction time results in relation to previous research is based on the nature of the particular task used in the present experiment and also evidence for independent programming of direction and magnitude of saccades. The evoked potential effects found are suggested to occur because the anti and normal conditions require different degrees of selective attention which has been shown to augment a similar negative evoked potential component. The PMP scalp distribution obtained is related to the hypothesized location of cortical generators in the parietal lobe where cells have been found which are activated prior to visually-elicited saccades in monkeys. The amplitude difference found for normal and anti saccades suggests that the PMP is sensitive to the different demands of the two tasks.

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Introduction

Saccades are ballistic movements of the eyes which direct the fovea to a selected part of the retinal image and maximize acuity. As with other ballistic movements cerebral potentials have been found to precede saccades which are made both in a self-initiated, voluntary manner (Becker et al., 1972) and also in response to a target light (Kurtzberg & Vaughan, 1979). An additional complexity for any investigation of the potentials preceding stimulus-elicited as compared to self-initiated saccades is the contribution of visual evoked potentials to the movement-related potentials. The present research was designed in part to compare the potentials preceding saccades made in response to a stimulus light which was or was not the intended target of the eye movement. Additional conditions attempt to assess the possible effects of the visual stimulation following the saccade on the earlier movement-related potentials. The results of such research will hopefully relate to the question of how the brain generates the motor command for a saccade.

The following sections will more fully introduce the topics covered in this thesis and review the earlier research on which the present experiment is based.

The published research of relevance falls into four general categories: 1) neurophysiological research on the activity of

various parts of the brain in relation to saccadic eye movements; 2) experimental studies of human cerebral potentials preceding saccades, the category which is most relevant to the present research and which also happens to be the least voluminous; 3) human evoked potential research; and 4) detailed investigations of the timing and characteristics of human eye movements (and no other responses) related to a great variety of stimulus conditions. Each of these categories will be discussed in turn. The first will be included with the following general introduction to oculomotor neuroanatomy.

Structure and function of the oculomotor system

This section will briefly consider the parts of the nervous system involved in movements of the eyes, with emphasis on those structures mediating horizontal saccades. Due to an absence of anatomical knowledge for many areas, relevant physiological evidence will also be presented. The general organization of this section will be to discuss the various parts in the order of peripheral to central. In evaluating this undertaking a recent remark by Bender (1980) should be kept in mind: "Based on clinical, pathological, experimental, physiological and recent anatomical studies it appears, as Graybiel (1977a) commented, that the oculomotor system is one of the most diffusely represented subsystems in the cerebrum and brain-stem and that its intrinsic organization is one of the most complex

of any in the central nervous system." (p. 57). Most of the structures to be discussed are schematically presented in Figure 1.

Brainstem structures

The six extraocular muscles of each eye are innervated by three cranial nerves, oculomotor, nIII; trochlear, nIV; and abducens, nVI. Unfortunately, "oculomotor" is thus doubly used to refer either to nerve and nucleus III alone or else all three nerves and nuclei together as well as associated structures. In this discussion the numerical designation will be used where there is a possibility of ambiguity, otherwise "oculomotor" will be used in both ways.

Control of the six extraocular muscles is unequally divided among the three brainstem nuclei. Innervation of four muscles, the superior, inferior, and medial recti and also the inferior oblique, is derived from the ipsilateral oculomotor nucleus. The lateral rectus is supplied by the ipsilateral abducens nucleus which, to point out a simple fact, does function to abduct the ipsilateral eye. Lastly, the trochlear nucleus controls the superior oblique, but in this case, of the contralateral eye.

Although the oculomotor nuclei and nerves clearly constitute the

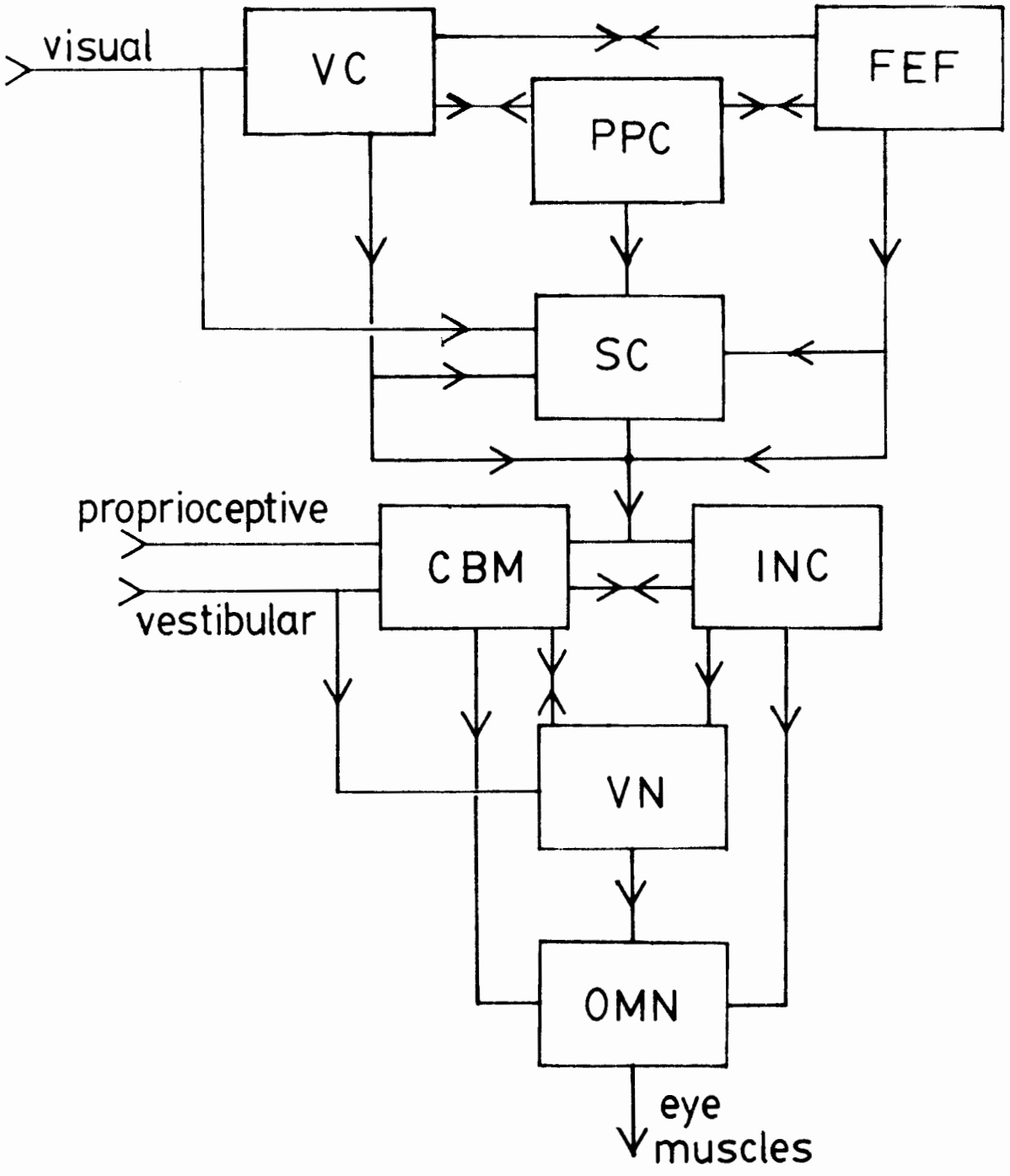


Figure 1. Schematic diagram of the major structures and pathways with demonstrated importance for oculomotor function. Not included is the paramedian pontine reticular formation, which has connections with all the other structures shown. Based on Carpenter (1977), the major change being the addition of the posterior parietal cortex. Abbreviations: VC, visual cortex; PPC, posterior parietal cortex; FEF, frontal eye fields; SC, superior colliculus; CBM, cerebellum; INC, interstitial nucleus of Cajal; VN, vestibular nucleus; OMN, oculomotor nuclei.

peripheral final common path for eye movements, the more central efferent pathways are less well defined and understood. The three oculomotor nuclei are located in the reticular formation of the brainstem, near the midline and ventral to the fourth ventrical. Important direct connections exist with: 1) the medial longitudinal fasciculus (MLF), a large fiber tract which passes close to all three nuclei; 2) two small nuclei situated just dorsal to NIII, the nucleus of Darkschewitsch and the interstitial nucleus of Cajal; 3) the dentate nucleus of the cerebellum, which sends direct fibers to the contralateral NIII; 4) the vestibular nucleus, adjacent to the abducens nucleus; and 5) the paramedian pontine reticular formation (PPRF), an area with diffuse boundries lying ventral to the MLF and extending between the levels of the trochlear and abducens nuclei (summarized in Carpenter, 1977).

Absent from the above list, a major subcortical structure which is clearly involved in the generation of eye movements, the superior colliculus, has not been found to have direct connections to the oculomotor nuclei (Szentagothai, 1950). A plausible reason for the necessity of intermediate connections is that the motor outflow of the superior colliculus consists of commands for eye movements which are coded in terms of retinal coordinates, rather than coordinates fixed in relation to the direction of the head (Robinson, 1972), which is required for appropriate control of tension in the extraocular muscles.

Additional processing of collicular output is therefore required at some intermediate location. The hypothetical site for this mechanism is the PPRF, or the cerebellum, or both (Carpenter, 1977). Evidence for the cerebellar control of eye movements will be discussed in a later section on the higher centers. The following will consider some of the relevant information concerning the brain stem.

Human clinical studies in the last century found that unilateral pontine lesions resulted in contralateral hemiplegia and paralysis of ipsilateral conjugate gaze (i.e. left pontine lesions eliminated saccades to the left), while lesions above the level of NIII resulted in paralysis of contralateral conjugate gaze (reviewed by Bender, 1980). Although hypothesized much earlier, the anatomical structures responsible for such effects have only recently been discovered. The abducens nucleus, which innervates the ipsilateral external rectus muscle, also sends out interneurons which cross the midline and ascend the medial longitudinal fasciculus to NIII, which controls the opposite medial rectus (Baker, 1977). This pathway presumably is important for the execution of conjugate horizontal saccades. Electrical stimulation studies have further revealed that while stimulation of the abducens nucleus may result in disconjugate ipsilateral abduction, stimulation of a more ventral area in the PPRF resulted in ipsilateral conjugate deviation of both eyes (Bender & Shanzer, 1964).

As further support for the importance of the PPRF, neurophysiological recordings have revealed "medium lead" saccade-burst neurons in this area which begin firing a few milliseconds before the onset of the saccade and continue throughout the duration of the saccade. These units showed greater response for saccades in a preferred direction, and the time course of the decay in firing rate during the saccade was highly correlated with the velocity component of the saccade in the preferred direction (Eckmiller et al., 1980). This research and other evidence, has supported the idea that the PPRF is an important structure controlling the coordinated functioning of the oculomotor nuclei for conjugate eye movements. Further anatomical grounds for the major role of the PPRF include the fact that this area receives direct inputs from the other higher centers involved with eye movements, including the superior colliculus, the cerebellum, and the cerebral cortex (summarized in Carpenter, 1977).

Concerning this input to the brainstem, results of the early human clinical work mentioned above are explainable by the recent stimulation and stereotaxic lesioning research which showed that the oculomotor pathways from higher centers cross the midline in the brainstem at a level between that of NIII and NIV (Bender & Shanzer, 1964).

Following the intended organization of this section, some of the more important higher-level structures mentioned above will now be discussed.

Cerebellum

The cerebellum has been included as one structure with direct connections to NIII, although the wealth of indirect connections are probably more important, particularly for horizontal saccades. The cerebellum is highly complex, both anatomically and functionally, and is involved in motor control for all types of movements for the whole body (Eccles et al., 1967). The four major areas of the cerebellum most involved with eye movements are: 1) the vestibulocerebellum (flocculus, nodulus, and uvula); 2) vermal lobuli VI and VII ("oculomotor vermis"); 3) the lateral cerebellar hemispheres, lobuli simplex, crus I and II; and 4) the four cerebellar nuclei, globose, emboliform, dentate, and fastigial. The first three areas correspond to parts of the archi-, paleo-, and neo cerebellum, respectively, subdivisions based on the phylogenetic development of the cerebellum.

The vestibulocerebellum receives direct input from primary vestibular fibers and is reciprocally connected with the vestibular nucleus (summarized in Carpenter, 1977). Lesions of the floculo-nodular lobe result in positional nystagmus, while

electrical stimulation does not evoke any eye movements, but does inhibit ongoing vestibular nystagmus (Kornhuber, 1971). Another interesting finding, which although not likely to lead to a preferred treatment for a common human malady, does indicate how closely this area is associated with vestibular function, is that lesions of the archicerebellum relieve susceptibility to motion sickness (Bard et al., 1947, cited in Kornhuber, 1971).

Physiological evidence for the importance of the oculomotor vermis includes findings that saccades are elicited by electrical stimulation and impaired by stereotaxic lesions, and also that vermal Purkinje cells fire prior to saccades (Precht, 1977). The processing role of this area is suggested by the fact that vestibular, neck, and extraocular proprioceptive inputs converge here with visual inputs, all of which provide necessary information concerning positions of the head, eyes, and visual target, required for appropriate execution of eye movements.

The more lateral areas of the adjacent neocerebellum appear to have similar functions in respect to eye movements. Kornhuber (1971) has presented a theory of the function of the cerebellar cortex for ballistic movements in general, which will be briefly described. Kornhuber (1971) considers ballistic movements to be ones that are so rapid that any peripheral feedback would be too

late to be of use in their continuous regulation, therefore they must be largely preprogrammed. For the pure case of the fastest possible movement, all motoneurons will be recruited together and the single controlling signal is simply the time duration of the efferent impulses. Kornhuber (1971) cites evidence (Shiller, 1970) that during saccadic eye movements, the agonist extraocular muscle always fires at the highest possible rate. The distance the eye moves will therefore be a function only of how long the contraction lasts. Since the time delay for transmission of visual information from the retina to the visual cortex in man is relatively long, at least 35 msec, and because small saccades have total durations less than this, a saccade, once initiated, must be completed without the assistance of any visual information indicating where the eye is to stop.

According to Kornhuber, the mechanism required to control the magnitude of saccadic eye movements is an accurate clock (actually a variable-interval timer) and its location is in the cerebellar cortex. The evidence for this comes primarily from clinical studies of ocular motility in patients with cerebellar lesions (Kornhuber, 1973). These patients usually exhibit an inability to produce saccades of the correct amplitude (dysmetria) and usually undershoot the target (hypometria). For unilateral lesions, dysmetria might occur, for example, for left-to-right saccades but the return saccades would be normal. The dysmetric eye movements are aimed in the correct direction,

but the correct magnitude is only achieved by trial and error.

Kornhuber (1973) states that in pure cases of cerebellar cortical atrophy there are not other symptoms of gaze paresis, gaze nystagmus, abnormal smooth pursuit movements, or slowing of saccadic velocity; such symptoms occur in cases with damage also to the cerebellar nuclei and brain-stem oculomotor nuclei.

The cerebellum anatomically seems highly suited for timing short intervals (Llinas, 1974) and recordings of electrical activity show very high frequencies of discharge from Purkinje cells, up to 500 impulses/sec (Thach, 1970, cited in Kornhuber, 1971).

As well as saccadic eye movements, Kornhuber considers the same cerebellar functions to exist for ballistic limb movements. The clinical evidence is that patients with cerebellar cortex lesions similarly show dysmetria for fast voluntary movements, and also adiadcchokinesis, the inability to start the next rapid movement after the end of the first, or chain together a series of movements (e.g. playing the piano).

Kornhuber (1971) further theorizes that the cerebellar nuclei have a function complementary to that of the cerebellar cortex: following a movement these structures act to maintain a constant position. Clinical symptoms resulting from lesions of the cerebellar nuclei include the so-called "intention tremor" of

the arm which occurs after a rapid movement (Kornhuber, 1971). Pendular nystagmus, tremor of the eyes during attempted steady fixation, is the corresponding disturbance of eye movements.

Superior colliculus

The superior colliculus (SC) is a mesencephalic structure important for the control of eye movements. The primate SC is homologous with the optic tectum of submammalian vertebrates, but over phylogenetic development the mammalian visual cortex has assumed many of the functions of the SC in simpler vertebrates.

Related to this separation of functions, some authors have distinguished between a "first" and "second" visual system; the first being the major geniculostriate pathway and the second a less clearly demarcated colliculo-pulvinar-parietal cortex pathway (Schneider, 1969; Zihl & Von Cramon, 1979). The former mechanism is supposedly responsible for form perception (discrimination and identification) while the latter subserves the detection of events, their localization in space, and the control of orienting responses. A well established finding of neuropsychology is that bilateral striate cortex ablations result in blindness as measured by most tests. However, monkeys with such damage to the first visual system can still detect and localize briefly presented visual stimuli as judged by their

reaching movements (Weiskrantz, Cowey, & Passingham, 1977). Human patients with unilateral damage to the geniculostriate pathways are similarly able to make accurately directed saccades to visual targets presented in their "blind" visual hemifields (Poppel, Held, & Frost, 1973; Sharpe et al., 1979). This evidence suggests that the geniculostriate visual system is therefore not necessary for the detection and localization of visual stimuli.

Anatomically, the SC has all the appropriate connections for the control of eye movements based on visual information. As summarized in Carpenter (1977), visual information arrives at the SC via inputs from the retina, the lateral geniculate of the thalamus, and the visual cortex. Other afferents include fibers from the eye fields of the frontal lobe, as well as from the reticular formation. Output of the SC includes bilateral projections to parts of the pontine and medullary reticular formation (including the PPRF), and to the interstitial nucleus of Cajal and the nucleus of Darkschewitsch. These areas, as has been mentioned, have direct connections to the oculomotor nuclei. Other efferent pathways travel to the tectospinal tract, the medial and lateral geniculate nuclei and the pulvinar of the thalamus. SC output also reaches the cerebellum by way of the dorsolateral pontine nuclei.

Visual input to the SC does preserve a retinotopic arrangement,

i.e., the afferent projections maintain information concerning the relative displacement of a stimulus from the point of fixation (Robinson, 1972; Schiller, 1978). Units in the SC respond to stationary and moving stimuli, and show an enhanced response if the stimulus elicits a saccade (Wurtz & Mohler, 1976).

One important experiment related to the involvement of the SC in visual-oculomotor functioning has been reported by Sparks, Pollack, and Mays (1978). Briefly, neurons in the (more anterior) SC exhibit a burst of activation following the presentation of a visual stimulus in a particular section of the visual field if the visual stimulus elicits a fixation saccade. Neurons in the more superficial layers of the SC show a response which is most highly correlated with the presentation of the visual stimulus, while the activation of neurons in deeper layers is more tightly coupled to the initiation of the saccade (see also Schiller, 1978). A function of the SC, therefore, appears to be the translation of visual sensory information regarding the retinal location of a stimulus into the corresponding motor commands for fixating that stimulus. The question Sparks et al. (1978) raised was whether or not the generation of motor signals from the deeper layers was completely dependent upon sensory activation of the superficial layers. For monkeys trained to follow a stimulus which first shifted one direction and then returned, even if the second

stimulus movement was completed during the reaction time for the monkey's first saccade, the second saccade followed activation in the deeper layers of the corresponding part of the SC, even though there was no previous visual stimulation of the corresponding part of the visual field.

Over a century ago Adamuk reported reported that electrical stimulation of the SC evoked contralateral conjugate eye movements (Adamuk, 1870, cited in Roucoux & Crommelinck, 1976). Recent research methods have shown that brief, localized stimulation produces saccades which direct gaze to that part of the visual field predictable from the retinotopic arrangement of the SC, while prolonged stimulation results in a sequence of saccades with similar direction and amplitude, the so-called "staircase effect" (Robinson, 1972). This evidence supports the idea that the SC codes information for eye movements in terms of retinal coordinates. There is also, however, a dissenting opinion on this view (Roucoux & Crommelinck, 1976; Crommelinck et al., 1977; Guitton et al., 1980) which holds that electrical stimulation has also been found to result in "goal directed" saccades, the direction and amplitude of which are dependent upon the initial position of the eye. Corroborative evidence that the SC does utilize information concerning eye position has recently been reported by Donaldson and Long (1980). This experiment studied the responses of single units in superficial layers of the SC to both visual stimuli and also mechanical

stretch of the extraocular muscles. Muscle stretch was found to increase activity in about half of the units studied and also to moderate responses to the visual stimuli.

Given that there is evidence for the processing of eye position information in the SC as well as a retinotopic organization, it appears that the current concepts of SC function are, at best, incomplete. Possibly signals for eye movement are coded in terms of both retinal coordinates and head coordinates. Alternately, even though the basic organization of the SC is retinotopic, eye position information might be necessary for the successful foveation of targets presented in the far periphery outside of the maximum range of eye movement, which would also require coordinated head movement.

Contrary to what might be expected from the electrical stimulation studies, small lesions within the SC do not have the effect of eliminating visually elicited saccades for targets in the affected area. The form of the saccades produced following such lesions is normal, and the only effect found is an increase in the latency of the response (Wurtz & Goldberg, 1972). This same finding has been reported for human patients following hemicolliculectomy, with the added result that the effect shows recovery over time (Heywood & Ratcliff, 1975). Since an intact SC is not necessary for the performance of visually elicited saccades, other parts of the brain must also participate in this

function. The following discussion of the role of the cerebral cortex will suggest some possible candidate structures.

Cerebral cortex

During the last ten years there appears to have been a major shift in the relative emphasis placed on the various areas of the cortex implicated in the control of eye movements. Older sources discuss primarily the eye fields of the frontal lobe and the visual cortex of the occipital lobe, while recent research has concentrated on parts of the parietal cortex. For example, Carpenter, in his 1971 review of central oculomotor pathways, only briefly mentions the parietal cortex as one source of input to the superior colliculus. The following discussion will summarize some of the major experimental findings for each of these three cortical areas.

The occipital cortex has been mentioned earlier as part of the "first visual system." Research in the last century found that electrical stimulation of the occipital lobe resulted in conjugate eye movements to the contralateral side (Schafer, 1888, cited in Carpenter, 1977). Lowest stimulation thresholds are found near the calcarine fissure in Brodmann's area 17 (Figure 2), the primary projection area for geniculostriate visual afferents, but responses are also obtained from the surrounding areas 18 and 19 (Carpenter, 1971; Bender, 1980). In

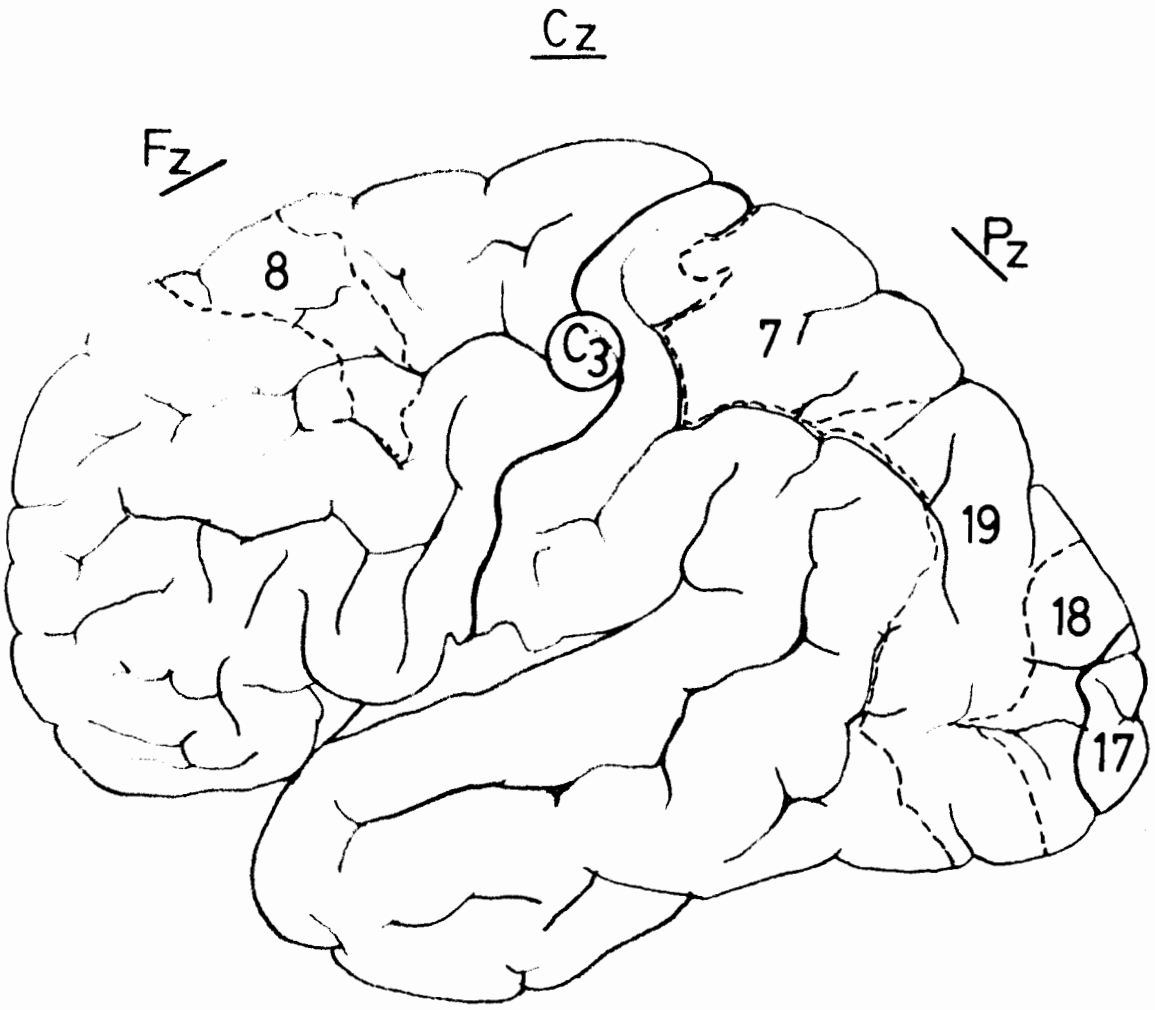


Figure 2. Human cerebral cortex. Outlined are those cytoarchitectonic areas of Brodmann which are mentioned in the text (based on Crosby et al., 1962). The less well-defined boundaries (Elliot, 1947) are indicated by dashed lines. Also shown are the midline and left scalp electrode locations (Jasper, 1958) used in this study.

evaluating the relevance of these stimulation effects for the role of the occipital cortex in oculomotor control, an important concept that shculd be kept in the back of one's mind is that such stimuli are perceived by human subjects as visual phosphenes located in the visual field at a place determined by the retinotopic organization of the visual cortex (Brindley & Lewin, 1968), and so an electrical stimulus could act purely as a visual target which the eyes attempt to foveate. The electrical stimulation evidence for the visual cortex, as well as the results of the lesion studies mentioned earlier, are consistent with the hypothesis that the visual cortex is primarily ccncerned with sensory processing and not the control of eye movements. This is not to deny, however, that the major output of the visual cortex to the superior colliculus is undoubtably imprtant for the functioning of the latter structure.

Electrical recording methods have revealed the intricate visual properties of striate neurons as shown in the well-know work of Hubel and Wiesal. Wurtz and Mohler (1976) investigated the neuronal responses to visual stimuli in relation to the occurrence of an elicited saccade. Few striate neurons were found to show any effect of eye movement. Those that were influenced showed an enhancement effect that was nonselective in relatica to the particular response, i.e. the same response enhancement was found for both saccades made to the stimulus or

to some other location, and also for a bar press response. This indicates that the responsiveness of the visual cortex is influenced by general activation, but single units do not reflect a selective attention process.

The next cortical area to be discussed, the frontal eye fields (FEFs), were discovered in the early electrical stimulation research of Fritsch and Hitzig in 1870. The location of the FEFs roughly corresponds to parts of area 8 in Brodmann's cytoarchitectonic map (Figure 2), the caudal part of the middle frontal gyrus and the adjacent inferior frontal gyrus (Carpenter, 1971). There is a separate representation of specific eye movements in both the upper and lower areas. The FEFs receive visual information from the visual cortex by way of the pulvinar nucleus of the thalamus, as well as from intercortical connections. Important efferent pathways are directed to the medial pontine nuclei, a structure providing a link to the cerebellum; the pontine reticular formation, which as described earlier, is connected to all the other oculomotor structures; and the superior colliculus (Kuypers & Lawrence, 1967).

Electrical stimulation of the FEFs yield conjugate contralateral saccades at low threshold and a relatively short latency of about 15 msec (Robinson, 1972). Generally, stimulation of the FEFs yield results similar to that for the SC, saccades of

realistic appearance, the direction and amplitude of which depend upon the location stimulated, not the initial eye position.

This last evidence might lead to the conclusion that the FEFs effect eye movements by means of the SC. This can not be true, however, because of the short latency of saccades following FEF stimulation, and also because colliculectomy does not eliminate this effect (Schiller, 1977). Furthermore, if both the FEFs and the SC are stimulated simultaneously, the resulting saccade is the weighted vector sum of the two saccades which would be obtained by stimulation of each location alone, the weighting factor being the relative stimulus intensity (Schiller et al., 1979). These findings indicate that both the FEFs and the SC control eye movements by means of independent pathways which converge on brain stem centers.

Given this independence in FEF and SC function, an attractive assumption is that the SC is the structure responsible for the initiation of saccades made in response to a visual stimulus, while the FEFs organize saccades made spontaneously. However, a major problem with the concept that the FEFs act to initiate eye movement comes from single unit recording studies which have found no cells in the FEFS which are activated prior to the onset of spontaneous saccades (Bizzi, 1968; Bizzi & Schiller, 1970; Guitton & Mandl, 1977; Mohler et al., 1973). However,

recently some brief reports of such units have appeared (unpublished observations by Arrezzo et al., cited in Goff et al., 1978; Bushnell & Goldberg, 1979), as well as one report of gross recordings from the surface of frontal cortex which showed potentials preceding saccades (Rosen et al., 1978).

Excluding these controversial findings, recording studies have generally reported that about one-half of the cells in the FEFs respond to visual stimuli and show large, nonspecific receptive fields. Almost one-half of these visual cells exhibit an enhancement effect which is selective for saccades to a visual stimulus in the receptive field of the cell (Wurtz & Mohler, 1976). Although the visual response of the FEFs is similar to that found in the superior colliculus, the latency is much longer in the FEFs (50-120 msec versus 35-60 msec, cited in Wurtz & Mohler, 1976), which is consistent with the hypothesis that the FEFs are most active not before, but rather during and after saccades.

The last cortical area to be discussed is the posterior parietal cortex, corresponding approximately to Brodmann's area 7 (Figure 2). This area has a wide variety of anatomical connections with other parts of the brain. Those important for oculomotor control include connections with frontal eye fields, visual cortex, superior colliculus, pulvinar nucleus of thalamus, and pontine nuclei (summarized in Robinson et al., 1978; Lynch,

1980). The classical conception of area 7 is that of an association area where visual, somatosensory, and behavioural information is analyzed and integrated (Robinson et al., 1978).

Some of the complex functions of area 7 are indicated by human clinical findings. A major perceptual symptom of parietal lesions in humans is referred to as "contralateral neglect." This is a lack of attention to the side of the body and the visual field contralateral to the lesion (Lynch, 1980). When human patients with such lesions are asked to draw pictures they usually leave out details in the affected hemifield. Weinstein (1980) describes one case of a man with neglect of the left visual field, who when asked to draw a daisy began by placing petals only on the right side and then filled in the others by rotating the page! Patients tend to avoid making eye movements to the affected side, although they maintain the ability to do so (Weinstein, 1980).

Primary evidence for the importance of the parietal cortex in oculomotor control has come from recently accumulating electrophysiological research. Several groups of researchers have emphasized different functional aspects of this area including: spatially organized convergence of information from different sensory modalities (Hyvarinen & Poranen, 1974; Hyvarinen, 1981), visual sensory functions (Goldberg & Robinson, 1977; Robinson et al., 1978), and the control of eye movements

(Mountcastle et al., 1975; Lynch et al., 1977).

The last-mentioned group has described different populations of cells which were active in association with saccades, smooth pursuit eye movements, or visual fixations. The so-called "saccade neurons" discharged on the average 70-80 msec prior to saccade onset for visually-elicited saccades (Lynch et al., 1977). These authors proposed the hypothesis that area 7 in the parietal cortex contains the neural mechanisms for the direction of visual attention to objects of interest including the apparatus necessary for the initiation of saccades. The visual stimulus was thought to be unimportant for this function because of the reported finding that saccade neurons which responded when a saccade was elicited by a small spot of light did not respond when the same visual stimulus did not elicit a saccade (Lynch et al., 1977).

In disagreement with this last point, Goldberg and Robinson (1977) performed a similar experiment and found that saccade neurons which showed no visual response to a small stimulus did in fact respond to a larger, brighter stimulus. These authors maintain that all active cells in area 7 show a sensory response and none discharge reliably before movement when a stimulus was not presented. However, as Lynch (1980) points out, a large enhancement in the visual response which occurs only when the stimulus is the target of a saccade can also be considered as a

sign of oculomotor function.

For saccades made in the absence of a visual stimulus, a plausible explanation for the lack of cortical single units found to show a reliable burst of activation preceding spontaneous saccades is that some cortical neurons are activated and do initiate each saccade, but it is not the same neurons which are activated prior to each saccade. If this is the case, gross recordings might give a better picture of what large areas of cortex are involved in the production of saccades. A major interest of the present research is the relative participation of the frontal and parietal areas in visually elicited versus non-visually elicited eye movements as revealed by scalp-recorded event related potentials. The next two sections will review other relevant research which features this technique.

Human saccade-related potentials

A great deal of research on electrocortical potentials preceding voluntary movement has been done since Kornhuber and Deecke's (1965) original report, however very little of it deals specifically with eye movements. Most of the studies that have investigated activity in the electroencephalogram (EEG) related to saccades were concerned with the potentials following the

initiation of eye movement, generally denoted as "lambda waves" (Barlow & Ciganek, 1969; Fourment et al., 1976; Scott & Bickford, 1967; Yagi, 1979). Since lambda waves occur after the eye has started to move the observed waveforms may include contributions from a) eye muscle potentials (Becker et al., 1973); b) the corneo-retinal potential, a well known source of EEG artifact; c) the visual evoked potential to the shifting retinal image (Kurtzberg & Vaughan, 1977; Scott & Bickford, 1967); and also d) a possible electrophysiological correlate of the "corollary discharge," i.e. a burst of activation from motor to sensory centers which signals the latter that altered sensory input is due to some voluntary movement of the organism itself, rather than any change in the distal stimulus (Jeannerod, Kennedy, & Magnin, 1979). Given the problems of interpretation for potentials occurring after the initiation of the saccade, the present research has been limited to investigation of pre-saccade activity only. This is a reasonable limitation also because saccades are strictly ballistic movements of such short duration. Carpenter (1977) reviews a number of studies and states that saccade duration is a linear function of amplitude approximated by 21 msec plus 2.2 msec for every degree of amplitude, for saccades of five degrees or more. This means that, for example, the execution of a relatively large saccade of 25 degrees requires only about 50 msec, which is not enough time for visual feedback to be of use in controlling any aspect of the eye movement, and therefore the saccade must be

preprogrammed.

Of the relatively few studies of pre-saccade potentials, the one by Barlow and Ciganek (1969) was an extension of their investigation of lambda waves. The single EEG channel recorded in the experiment was a bipolar parietal-occipital derivation. For the condition in which the subject's task was tc look from a dim spct to a bright spot, at a self-paced rate, the averaged EEG showed a potential which was positive at the parietal electrode and appeared about 150 msec prior to the start of the saccade (Barlow & Ciganek, 1969, figure 5).

Of the more recent relevant studies, the most extensive experimentation has been reported by Becker, Hoehne, Iwase, and Kornhuber (1972, 1973), and comparable research has been published by Kurtzberg and Vaughan (1973, 1977), which will be discussed jointly.

In both series of experiments the subject's usual task was to look back and forth between fixation points, not faster than once every 2-3 seconds (Kurtzberg & Vaughan) or 10 seconds (Becker et al.). Both groups found, starting up to 1 sec prior to the saccade, a slow negative-going potential, referred to as either the Bereitshaftspotential (BP), or readiness potential (RP), depending on chcice of language. This BP is very similar to the negative slow potential found to precede self-paced

voluntary limb movements (Kornhuber & Deecke, 1965). According to both groups of researchers, the BP does not occur if the eye movement is elicited by the presentation of a visual stimulus. Actually, of course, there is no time for a slow wave to occur during the approximately 200 msec reaction time for saccades. Becker et al. (1972, 1973) also report a variation on the basic experiment in which the subject's task was to look back and forth from a fixation point to a table of letters, each time fixating a different letter, so as to spell out a word on consecutive saccades. This more involving task increases the BP amplitude for glances to the tablet, and also, interestingly, markedly decreases BPs for the return saccade (Becker et al., 1972).

Just before the beginning of the saccade both Becker et al. and Kurtzberg and Vaughan report the occurrence of a positive-going potential, starting approximately 150 msec before the saccade. This waveform is also found just prior to voluntary limb movements and has been named the "Pramotorische Positivierung" or PMP by the group in Germany.

One seemingly unresolved difference between the findings of the two groups is that Becker et al. report maximal BP and PMP amplitudes at the vertex (the top of the head, overlying the frontal gyrus), less at frontal and parietal locations, and least or no activity in the occipital region. Kurtzberg and

Vaughan (1973, 1977), on the contrary, report maximal amplitudes of both waveforms at the parietal location, slightly less at the frontal, and minimal activity at occipital and vertex sites. Both groups of researchers do appear to agree on their basic explanation of both the BP and the PMP. Becker et al. (1973) say that the BP is "a sign of the general preparation of premeditated voluntary acts and that it is only indirectly related to the initiation and the motor control of the movement." (p. 104). The PMP is thought to correspond to the actual motor command to move the eyes (Becker et al., 1973). On the assumption that the PMP is the electrocortical potential most directly related to the generation of the saccade, it is therefore of major interest for this research.

Some other relevant experiments have been reported by Armington (1978a, 1978b). In these two strikingly similar papers Armington presents data showing BP and PMP waveforms quite like those presented by the other groups. Armington's records obtained from vertex seem to confirm the reports of Becker et al., rather than those of Kurtzberg and Vaughan concerning the presence of a large BP and PMP at that location. However, one difficulty for interpretation of these data is the fact that Armington used "an auditory or visual pacing signal" (1978b, p. 365) which is not further described.

An additional experiment reported by Armington (1978b) is at

least suggestive of an interesting finding concerning the PMP. When the size of (rather small) saccades was varied in four steps from 7 to 81 minutes of arc, the initial positivity became evident earlier with the larger saccades, possibly implying that the duration of the PMP is a function of saccade amplitude.

Very recently Kurtzberg and Vaughan (1979) investigated the scalp topography of potentials preceding visually triggered and self-initiated saccades. As described in their preliminary report, the subject's task was to quickly move the eyes from a fixation mark to a dim target light which appeared to the opposite side of the fixation mark and to maintain fixation for the one second the light remained on, then to return the eyes to the original fixation mark. In another condition the subject simply performed voluntary saccades between two stationary fixation points. Kurtzberg and Vaughan recorded EEG from seven midline locations and analyzed the "slow positive antecedent potential" which seems to correspond to Becker et al.'s (1972, 1973) PMP. For the triggered saccades the relative amplitudes were largest at parietal and smallest at frontal locations, while the return saccades showed the opposite, greater amplitudes at vertex and frontal locations. The self-initiated saccades, which were produced in the condition similar to that used in the previous research, showed a more even distribution of antecedent potentials with the greatest amplitude at vertex, confirming Becker et al. (1972, 1973) but not Kurtzberg and

Vaughan's (1977) own earlier work.

In their latest paper, Kurtzberg and Vaughan suggest that the triggered condition results in greater potential amplitudes over the parietal lobe because of the activation of cells in that area which have been found to respond to visual stimuli which elicit saccades (Lynch et al., 1977). Non-triggered saccades, on the other hand, are supposedly more dependent upon activation of the frontal eye fields (Mohler, Goldberg, & Wurtz, 1973). Unexplained, however, is why the return eye movements, which were actually made in response to the offset of the target light, should show more frontal and less parietal activity than the saccades which were entirely self paced.

Another question I have about Kurtzberg and Vaughan's (1979) results concerns the latencies of the saccades to the light stimulus. The authors do not mention any actual values, but state that the initial positive potential started up to 350 msec before the saccade onset. Assuming a reasonable additional time for retinal delay, the total reaction time (RT) to the light must be no shorter than 400 msec, which is quite long compared to other studies and my own research. Also reported was that for the five out of twelve subjects whose records did not show the early positive potential, there occurred an initial negative wave approximately 150 msec before movement. Whether or not this variability was related to average RTs or other variables

was not discussed.

The major contribution of the Kurtzberg and Vaughan paper is the evidence that a parietal-frontal distribution difference was found, in the predictable direction, for positive potentials preceding saccades in the experimental conditions employed. However, because there was a different visual stimulus used in the triggered condition (light onset) from that used in the return condition (light offset), which was in turn different from that in the self-initiated condition (no light) the evoked potential contribution cannot be equivalent. As a means to avoid this confounding, in the present study the same visual stimulus is to be presented before the saccade in all experimental conditions, even though the instructed target of the saccade will be different.

Evoked potentials and attention

Kurtzberg and Vaughan (1979) also reported some data on the visual evoked response to the target onset. They found for the stimulus-synchronized averages a large enhancement of both early and late components in the triggered condition compared to a no-movement control condition. An enhancement was also found for a condition in which a hand movement was the required response. In this study the one quantitative measure of early EP activity occurring before the movement onset consisted of the

peak-to-peak amplitude for a positive/negative complex which showed a more parietal-occipital distribution for eye movements compared to the precentral maximum for hand movements.

A number of other studies have investigated the influence on the visual evoked potential of a required hand movement. An experiment by Eason, Harter, and White (1969) used stimuli similar to those of the experiment to be reported here, 1 degree in diameter lights presented 20 degrees left or right of a central fixation mark. Evoked potentials were obtained from a single derivation with the active electrode at the occiput. The most prominent component was a negative peak with a latency in the interval of 130-200 msec, labeled N1. When the subject was instructed to attend just to one of the two positions and press a button following the occurrence of stimuli at that side only, this component acquired a much greater amplitude for stimuli on the attended side. A similar effect was found in a condition in which the subject was required to accurately count the number of stimuli on one side, but not make any immediate motor response.

Confirmation of Eason et al.'s findings have been provided by Van Voorhis and Hillyard (1977). These investigators replicated the earlier study and also recorded from a vertex electrode from which a similar N1 component was observed. The results clearly showed that stimuli which were responded to produced larger N1 amplitudes.

One other study that will be mentioned is less directly related to the present experiment, but does provide important information about the nature of the N1 evoked potential component. Hink, Hillyard, and Benson (1978) report that for dichotically presented auditory stimuli N1 is enhanced to all stimuli presented to the attended ear. This and other research led these authors to suggest that the amplitude of N1 is an index of the degree of selective attention the subject has directed to the stimulus.

Human eye movement studies

The experiments discussed above have all concentrated on analyzing the EEG activity associated with very simple stimulus conditions. Other research that directly relates to the question of how saccades are programmed has investigated the details of eye movements occurring under more complex experimental situations. The one study most relevant to the present proposal was reported by Hallett (1978). In this experiment the visual stimulus was an oscilloscope spot which would suddenly shift to one of eight positions equally spaced to the left and right. The subject's task was as follows for four different conditions: 1) Normal saccade. The subject was to simply fixate the spot as soon as it has moved. 2) "Hyper" saccade. When the target moved to one of the three nearest

positions on either side of the fixation point, the subject was to fixate one position farther in the same direction. 3) "Hypo" saccade. The subject was to fixate on a point one-half the distance to the target. 4) "Anti" saccade. The subject was to fixate a point in the opposite direction to the stimulus jump at the same absolute distance. Hallett found that, as might be expected, subjects initially tended to make a large number of errors in the last three conditions and counter to instructions, fixate the target. After some practice, however, subjects were able to reduce the error rate to 5-7% and perform the required saccades with some degree of accuracy. The major result was that for the anti condition, although the amplitude vs. duration relationship of the saccades was very near normal (see Carpenter's findings discussed above), the latency of the anti saccades was clearly longer than that of the normal saccades by some 40-100 msec. The effect was similar, although not as large, for the hyper and hypo saccade conditions.

In discussing these results Hallett points out that in the anti task the laterally displaced stimulus is visually processed by the contralateral hemisphere, while the ipsilateral hemisphere must send out the motor command to activate the appropriate medial and lateral recti muscles to turn the eyes away from the stimulus (see Bender, 1980 for a review of the neuroanatomy). Hallett suggests that the increased time required for trans-hemispheric conduction accounts for some of the added

latency. Hallett also hypothesizes that in the anti task, the subject responds by internally creating some kind of "neural image" somewhere within the brain at a location corresponding to the retinotopic projection of that part of the retinal hemifield contralateral to the retinal image of the displaced target. The observed anti saccade results when the subject attempts tc fixate this peripheral neural image. The extra delay according to Hallett can then be attributed to the time required for construction of this neural image.

I personally favor a revised version of this latter explanation, one which makes more specific hypotheses about what parts of the brain are involved in producing saccades. Specifically, in the normal condition the saccade might be generated for the most part by the parietal-collicular mechanisms discussed earlier, which are highly involved in directing the eyes to visual stimuli presented in the peripheral visual field. The engagement of these mechanisms fcr a stimulus-directed eye movement quite possibly utilizes the fastest pathways and the most direct conections, resulting in faster reaction times. Saccades produced in the anti condition can be considered less visually elicited in that the stimulus does not direct the eyes to a particular location, but only supplies the temporal signal for the eyes to move. Some other unknown parts of the brain must be involved in prcgramming the appropriate direction and magnitude of the saccade. The anti condition then might be

expected to activate the frontal eye field area to a greater degree, as was hypothesized by Kurtzberg and Vaughan (1979) to be the case for their return condition. The research reported here is intended to investigate this question of frontal/parietal distribution, at least to the degree of localization possible with scalp-recorded EEG.

One general area of research related to Hallett's (1978) paradigm is the so-called "S-R compatibility effect." This term refers to the finding that some response made to the left or right shows the shortest latency when the imperative stimulus occurs at the same side. When the stimulus is presented to the opposite side, an additional 40-80 msec is required to respond (Cotton et al., 1980). Hallett's anti task can be seen in this perspective as an example of an "incompatible" response in a complex choice reaction time situation in which there are a total of eight possible stimuli and eight corresponding responses.

Although most of the S-R compatibility research has employed manual responses, two studies have used saccadic eye movements. Bertera et al., (1975) reported both simple and complex RT experiments for which the imperative stimulus was a tone presented monaurally to the left or right ear. In the simple RT situation the required movement in a block of trials was always left or right, regardless of the ear stimulated. No difference

in RT was found for this condition. For choice reaction time, a monaurally presented low pitched tone signalled left movements and a high tone signalled right movements. In this case compatible responses (i.e. looking right in response to a high tone in the right ear) produced faster responses by an average of 54 msec.

Posner, Nissen, and Ogden (1978) refer to a simple RT experiment in which both the eyes and the hand had to move in one direction in response to a light appearing to the left or right. The eyes generally responded faster than the hand and also showed a larger compatibility effect of about 50 msec. Posner (1978) reviews this and other research and indicates that the more straightforward experiment in which just an eye movement is required has also been performed, with similar results, although the data which are illustrated (Figure 7.5) appear identical to those previously described from Posner et al. (1978). Posner (1978) also mentioned that at that time he was unaware of a choice RT experiment investigating eye movement responses to light stimuli.

Another important area of human eye movement research includes investigations of the effects of warning stimuli on the programming of a saccade to a peripheral target. Ross and Ross (1980) had subjects look at targets presented 15 degrees left or right following a variable forperiod. The warning signal was a

change in foveal stimulation resulting from stimulus onset, offset, or change in form. Results showed that all three types of warning signals were effective in reducing RTs with forperiods of 300 or 600 msec, but stimulus offset was always more effective than the other stimulus types and was the only effective warning signal with a 50 msec forperiod. When the central visual stimulus was presented after the peripheral target, stimulus onset significantly increased saccade latencies, while stimulus offset had no such effect. The authors also mention a preliminary experiment in which a manual response was required to the same peripheral stimulus. In this case no advantage was found for stimulus offset rather than onset in decreasing manual RT. A number of suggestions were put forward to explain these results. Perhaps following the presentation of new information to the fovea saccades are delayed in order to allow for information processing before interference occurs from subsequent visual input. In support of this is evidence cited from Potter and Levy (1969) which showed an almost complete suppression of spontaneous saccades when subjects were presented with a rapid succession of pictures (4-8/sec). Ross and Ross suggest that the interference effects of stimulus onset in their experiment might result from some automatic process which delays all eye movements following a new stimulus, or else possibly a microsaccade occurs to the central stimulus which delays the saccade to the target because of the imposition of a refractory period which is normally found to

separate saccades of all magnitudes (e.g. Carpenter, 1977).

A comparable experiment investigating the effects of the interval between fixation stimulus offset and target onset has been presented by Saslow (1967a). This study used four target lights spaced at 4 degree intervals left and right of the central fixation light. A buzzer signaled a fixed forperiod of 1500 msec before target onset. At some time ranging in 50 msec intervals from 400 msec before to 350 msec after target onset the central fixation light extinguished. When the fixation light offset occurred 100 msec or more prior to target onset RTs were fastest, about 150 msec. Simultaneous offset and onset produced RTs averaging about 200 msec. When the central fixation light offset was delayed until 100 msec or more after target onset RTs were further increased to about 250 msec. The same effects were found with either blocks of trials with the same asynchrony or else scrambled trials. I think this last result is important for interpreting the previous study for the following reason. Since the buzzer was the only constant forperiod warning signal for the scrambled trials the fixation light offset provided no temporal information as to when the target would appear. This implies that it is not the case that stimulus offset, as opposed to onset, is simply a more accurate temporal cue and is therefore a more effective warning stimulus.

The explanation that Saslow offers for the reduction in RT when

central fixation light offset precedes target onset is similar to one suggestion made by Ross and Ross (1980) and mentioned above, that microsaccades occur more frequently when a subject is provided a fixation point and the refractory interval following a microsaccade delays the initiation of a saccade to the target. After rereading Saslow's article I have an alternate explanation for some of these results, which, while I do not necessarily believe it to be true, does suggest some further experimentation. For the identical neon bulbs used by Saslow, the offset of one light followed after a delay by the onset of another light at a displaced retinal position is the type of stroboscopic stimulation which can produce apparent motion (also referred to colloquially as the movie-marquee effect). If the same visual mechanisms are employed in both the detection of motion and the direction of the eyes towards objects in the visual environment, it seems a plausible hypothesis that stimuli resulting in strong apparent movement will also be effective in evoking a saccade with a minimum latency. Physiologically, there is evidence that stroboscopic stimulation activates the same retinal ganglion cells which respond to actual movement of an object in the same preferred direction (Barlow & Levick, 1965). Those ganglion cells which show the greatest response to movement also have large receptive fields and are in the class of the largest and fastest-conducting cells in the optic nerve, the Y-cells (Cleland, Dubin, & Levick, 1971; see also the review by Stone et

al., 1979). According to my explanation these velocity selective Y-cells which project to both the visual cortex and the superior colliculus will show the earliest response to peripheral light onset because they are facilitated by the previous central light offset.

Some aspects of Saslow's data do seem consistent with the idea that the optimal conditions for stroboscopic motion produce the fastest saccade latencies. In Saslow's experiment the targets were located at 4 or 8 degrees from center. The delay between center offset and target onset which results in the maximal apparent motion (and shortest RTs) should be larger for the 8 degree target than the 4 degree target. Unfortunately, Saslow does not present any RT data for individual targets. However, each analysis of variance of the four reported (two replications for each of two subjects) shows a significant interaction of target location and asynchrony which would be predicted from the above hypothesis.

Another interesting aspect of Saslow's results is that the interval between center offset and target onset which showed the smallest RTs was 200-300 msec, which corresponds to an image velocity of 20-30 degrees per sec for the average target excentricity. This compares closely to the optional velocity for apparent motion, 15-25 degrees per sec (Kolers, 1964).

A simple test of the above explanation would be to replicate Saslow's experiment using targets at different distances from the center and thereby determine if the optimal asynchrony is in fact directly related to the retinal excentricity of the target. Another suggested experiment might be to present the center and target lights to opposite eyes. This condition should result in greater RTs at the optimal asynchrony for binocular stimulation because apparent motion is reduced with dicoptic stimuli (Frisby, 1972). A few additional comments will be made about this question after the following discussion of some of the other relevant research.

One stimulus paradigm which has been used in a number of studies is the presentation of two successive targets in different parts of the visual field. In an early study by Wheeless et al. (1966) a light on a CRT screen moved from center position to 6 degrees left or right. On less than half of these trials the light was extinguished after 50, 100, or 200 msec and reappeared 6 degrees to the opposite side of center. The two-light stimulus was referred to as a "pulse-step," while single targets were called "steps." Results showed that regardless of the duration of the pulse, when the eye responded only to the step the reaction time was the same for all pulse-step trials and equal to the reaction time to the step alone plus 40 msec. Wheeless et al. interpret this 40 msec period as the time required by the nervous system to cancel the programmed response

to the initial pulse.

A more recent study by Becker and Jurgens (1979) reported a 20 msec increase in RT for similar experimental conditions. A possible expanation for the reduced effect might be related to the greater complexity of the stimulus conditions used in this experiment which consisted of single steps and pulse-steps randomly presented in any of five different stimulus positions. The step could be in the same direction as the pulse, or in the opposite direction but of one half or equal amplitude. The major finding of Becker et al. is that when the eyes perform two saccades, the time interval between the first and second is inversely related to the latency of the first. This is concluded to indicate that the visuomotor system is programming the second saccade before the first one begins; if there is a greater delay before the first more of this programming can be completed and therefore the intersaccadic interval is shortened.

Further research on this question has been reported by Hou and Fender (1979). An important feature of this study is that the eight stimulus positions used were equally spaced around a circle and both the vertical and horizontal components of eye movements were recorded. For pulse-step trials in which the step is in an opposite direction to the initial pulse, when the eye does not follow the pulse but moves directly to the location of the step there was for all orientations an increase in

reaction time similar to that found by Wheeless. However, when the final stimulus location was adjacent to that of the initial pulse (i.e. the stimuli were separated by just 45 degrees on the circle) RTs were much faster and not different from RTs to single steps. If the pulse and step were in orthogonal directions this condition produced RTs as long as those for steps to the opposite side. These results are consistent with those reported by Komoda et al. (1973) which showed smaller RTs when the pulse and target were in the same direction rather than in opposite directions. Hou and Fender (1979) discuss these findings in relation to Wheeless et al.'s cancellation theory and the programming of the agonist and antagonist muscles effecting the saccades.

Another possible explanation is the suggestion offered earlier, that some motion-detection mechanism is responsible for signalling the fastest responses. This theory would also predict that the saccade made in response to a target step would be faster if a target pulse occurs in the same direction. If the pulse is in the opposite or orthogonal direction possibly an inhibitory connection between the different orientation-specific detectors activated by the two stimuli is responsible for the observed increase in RT.

In support of the above the last research to be discussed in this section concerns an interesting phenomenon of the

relaticnship between saccades and smooth pursuit eye movements elicited by a step-ramp stimulus. Normally, if the eyes attempt to track a target light which suddenly starts moving at a constant moderate velocity (a ramp movement) the eyes first make a saccade in the direction of movement and then accurately track the stimulus with smooth pursuit movement. This initial saccade functions to overcome the reaction time of the smooth pursuit movement and allcw foveation of the target without requiring any modification in the velocity of the smooth pursuit movement. When the target only ramps in one direction this initial saccade is always observed. If, however, there is a step movement of the target in the opposite direction just prior to onset of the ramp, the saccade is reduced or absent (in man, Rashbass, 1961; Wheeless, 1966; in monkey, Fuchs, 1967). As might be expected, the optimal stimulus parameters for eliminating the saccade have been found to be a step magnitude in degrees .15 to .2 times the ramp velocity in deg/sec, so that the target recrosses its initial position after about 150-200 msec (Robinson, 1965), approximately one reaction time. An initial saccade cannot be prevented by using a target stimulus which increases in velocity gradually rather than suddenly (Fleming et al., 1969). The suggestion offered here is that the saccade made in the direction of the ramp is triggered by some motion detector mechanism which responds to the ramp, but that activation of this mechanism is inhibited by the target step in the oppcsite direction.

The foregoing discussion has digressed somewhat from the present topic, but was included to illustrate some of the important aspects of oculomotor function which have been revealed by eye movement recordings in studies utilizing more complex experimental situations. A basic idea behind the present research is to incorporate some of this experimental sophistication in a study which also records other measures of nervous system function, namely event-related potentials. Further details are presented in the following sections.

Rationale for the present research

This research relates to the general problem of how the brain organizes saccades. Of the possible means by which this question may be approached, the one selected is the analysis of cerebral potentials recorded from the scalp immediately preceding the start of eye movement. Scalp potentials, even though they do correspond to some aspects of brain activity, provide little information as to the physiological process responsible and its anatomical locus (Goff et al., 1978). Even though such measures do not indicate what the brain is doing or even what part is doing it, if scalp potentials do show a significant difference related to experimental conditions then it may be logically inferred that there is some corresponding

difference in brain function.

A major requirement of such a research approach is that in order for results to be interpretable the experimental design must allow for the rejection of alternate explanations for the differences obtained. The experimenter's burden is to devise conditions which isolate the causative factors from other plausible influences. An objective of the present research is to identify the particular situational variables which account for observed differences in the potentials preceding saccades.

One basic question to be considered is how the brain organizes eye movements which are visually elicited in comparison to those which are self-initiated (i.e. not triggered by a visual stimulus). A possible hypothesis is that all saccades are generated by the same brain mechanism regardless of other factors. There is no reason to expect, therefore, that the scalp potentials signaling the activation of this mechanism should be influenced by precursory or subsequent conditions. If a visual stimulus occurs prior to the saccade other sensory processing mechanisms will be activated as well, but these will be independent of the saccade-generation process. This hypothesis is consistent with the view that sensory and motor functions may be considered as separate systems. Stimulus-evoked potentials and movement-related potentials, while they might overlap in time, should not otherwise interact.

An alternate hypothesis, the one which this writer finds more tenable, is that motor functions are inherently interconnected and integrated with sensory functions. This hypothesis is consistent with the idea that the movement-related potentials preceding saccades will be influenced by the situation in which they occur. The present research is designed to investigate some aspects of the visual stimulus and saccade response relationship in terms of the associated event-related potentials. More specific statements of the problems to be addressed in this study will be presented following a discussion of some additional considerations from other research.

As reviewed earlier in the introduction, much of the previous research on movement-related potentials has concentrated on the slow negative wave (the BP) which arises up to one second prior to the start of a saccade. It is obvious that the BP cannot be a necessary precursor of movement because stimulus-elicited movements occur after a much shorter reaction time during which the BP could not theoretically appear. Another possibly important methodological aspect of the published research is that the subject is required to maintain fixation for a relatively long time (e.g. 10 seconds, Becker et al., 1973) between each saccade, while humans not instructed to fixate normally move their eyes more than once a second (Carpenter, 1977). The BP therefore can be considered to occur during an

interval when the subject must inhibit the performance of a movement.

Risking some controversy, I might also mention that a similar negative slow wave (which in this situation is referred to as the "contingent negative variation" or CNV) is observed in a fixed-foreperiod warned reaction time paradigm during a one- to several-second interval separating the warning and imperative stimuli. The subject is instructed to make no response prior to the imperative stimulus. Put another way, the subject's task is to inhibit a response during this interval. Viewed within the conceptual framework of classical conditioning, since the warning stimulus is always paired with the imperative stimulus, a conditioned response should arise shortly after the warning stimulus. Consistent with this idea, Rebert and Knott (1970) reported that the CNV started about one-half second after the onset of the warning stimulus, a time interval equal to that which yields the fastest learning of a conditioned response in classical conditioning. A final point to be mentioned as supporting evidence is that, as is well known to CNV researchers, an inexperienced subject in a CNV experiment is frequently found to (incorrectly) make a response to the warning stimulus, particularly on the first few trials.

In conclusion, the suggestion put forward, based on the above considerations, is that BPs (and CNVs) are observed in

experimental situations which require subjects to withhold or delay the execution of a response, and therefore the slow negative wave might correspond more closely to activation of a mechanism for response inhibition rather than response generation. Regardless of the validity of this last statement, it is still true that the BP cannot be an electrical sign of the process by which the brain initiates a voluntary movement simply because it appears at too great a time interval before movement onset.

As Deecke et al. (1976) also conclude in their review of the German group's research on movement-related potentials, the pre-motion positivity (PMP) is the cerebral potential most likely to reflect the motor command for movement. Furthermore, the distribution of this potential, with a maximum amplitude over the parietal cortex, is consistent with the parietal localization of the movement command mechanism which is also suggested by human clinical findings, as well as much animal research (recently reviewed by Lynch, 1980). Anatomically, the rich interconnections both within parietal cortex somatosensory association areas and with other sensory cortices, as well as major descending pathways to subcortical motor areas, particularly the cerebellum and basal ganglia, make the parietal cortex a likely site for a movement command mechanism which requires direct access to sensory information for appropriate functioning.

Accordingly, a major objective of the present research is to investigate the PMP as an electrical sign of the motor command for eye movements and in particular whether it also reflects not just motor outflow, but also relevant sensory input. As indicated in the discussion of the rationale for event-related potential research, simply observing a PMP provides little information about the underlying mechanism. On the other hand, finding that this potential is influenced by other experimental variables, independent of the movement itself, would contribute some additional knowledge about how eye movements are organized.

Objectives and design considerations

Given this rationale, more can now be said about the experimental conditions for which event-related potentials are to be investigated. In spite of the comments made about previous methods of research with self-initiated movements as being not comparable with stimulus-elicited movement, it is thought necessary to include a self-initiated condition in the present study. This will make possible comparisons with other research findings as well as constitute a necessary control for the stimulus-triggered reaction-time conditions.

Before describing the other experimental conditions, some distinctions will be made for the terminology to be used. The

term "stimulus-elicited" will be used when the onset of the stimulus is the cue for initiating a saccade and the stimulus itself is to be the target of the saccade. "Visually-triggered" is used to describe the situation where the saccade is initiated following stimulus onset, but does not necessarily redirect gaze to the location of the stimulus. Visually-elicited saccades are therefore also visually triggered, although the converse is not true. The importance of this distinction is that a stimulus which elicits a saccade might activate a brain mechanism such as that found in the superior colliculus for directing the eyes towards a visual target, while a saccade made in some other direction would have to be organized by a process which includes some extra-visual input in order to produce a saccade in the correct direction.

The method chosen for this study is a simplified version of Hallett's (1978) paradigm in which for each trial a stimulus light appears either at the left or right of a central fixation point. Following Hallett's terminology, in the "normal" condition the saccade is visually elicited, i.e., direction and amplitude, as well as the time for initiation, is specified by the stimulus. In the "anti" condition the stimulus provides the temporal cue for initiation of the saccade, but the saccade must be made in the opposite direction, as required by instructions.

The advantage seen in this method is that (for left and right

trials combined) the same visual stimulus is presented in both conditions and the required motor response is also identical. The only difference is whether or not the stimulus direction is the same as the required direction of movement. Both conditions are two-choice reaction time situations, and unlike Hallett's more complex method, each required response is of the same amplitude. Conceptually the experimental variable tested with these normal and anti conditions is the relationship between stimulus and response direction, which could also be considered as S-R compatibility.

A major objective of this study is to determine if this variable has an influence on the event related potentials preceding the movement. Such an effect on the PMP would suggest that this potential relates to more than just motor outflow, but also the integration of sensory information related to the intended movement. The evoked potential to stimulus onset, in particular the early components occurring prior to the start of the PMP, will be investigated for a possible effect of the nature of the subsequent response.

A major concern for the design of this study has been to make the normal and anti conditions the same except for the single factor of the relationship between the direction of the stimulus and the direction of the saccade. A possible confounding variable which remains, however, is the difference in visual

stimulation following the saccade, which is also dependent upon the task factor To explain this, in both of the experimental conditions proposed so far there occurs first a peripheral visual stimulus, then a saccade is produced, and then there is effectively another visual stimulus, i.e. the stimulus light at the fovea (normal condition) or the stimulus light in the far periphery (anti condition). The saccade can be considered to separate the two stimuli because of the well-known phenomenon of saccadic suppression, the reduction of visual perception at the time of a saccade (reviewed by Carpenter, 1977). In the normal condition the eyes saccade toward the target and then a foveal stimulus is received. In the anti condition the eyes move away from the target and a peripheral stimulus is received. A concern, therefore, is whether any difference for the normal and anti conditions might not entirely be due to the eye movement response per se, but also to the subsequent visual stimulation.

One possible reason for such an effect would be that when the subject saccades to the target the offset of the stimulus provides information concerning the subject's reaction time. In this study the visual stimulus has a duration of 500 msec and therefore the subject will perceive the target briefly before it terminates. Assuming that the duration of the stimulus may be more accurately estimated when seen foveally instead of peripherally, the subject might be more encouraged to minimize reaction time in the normal condition in order to receive a

longer glimpse of the target. A factor such as this would correspondingly have an influence on the event related potentials observed for this condition.

Another reason for investigating the possible role of post-saccade stimulation relates to plasticity in visual-oculomotor processes. If for visually-elicited saccades there is activation of some mechanism specific for the direction of gaze towards a visual target, perhaps successful foveation is necessary to maintain the normal function of this mechanism over repeated trials. Although this is purely speculative, a good example of the type of plasticity suggested has been found for vestibular nystagmus, the compensatory movement of the eyes that occurs with rotation of the head. Gonshor and Melvill Jones (1976) had subjects wear reversing prisms which made the visual field move in the opposite direction during head rotation. After a few days vestibular nystagmus, tested in the dark, was found to be reversed in direction. This indicates that the presumably simple vestibulo-ocular reflex mechanism was actually highly modifiable by alteration of the movement-related visual input.

Besides the reasons stated above there might of course be some other unidentified factor associated with post-saccade visual stimulation which would influence the results for the normal and anti tasks. In order to investigate these possibilities two

additional experimental conditions have been included which are described below.

The method used in this study to investigate the role of post-saccade visual stimulation is to experimentally make independent the retinal location of stimulation following the saccade and the direction of the saccade. This can be done easily for the limited stimuli used in this study by having the saccade-detecting circuitry turn off the target stimulus at the onset of the saccade and turn on the light at the opposite side. The present study will therefore include two additional conditions in which the stimulus light moves to the opposite side of the screen during the saccade.

For reasons of clarity in discussing the design of this experiment the labels "same" and "opposite" will be used to describe the post-saccade stimulus location for these conditions, rather than "foveal" and "peripheral" because the latter of course depend upon the subject's eye movement and not just the experimental manipulation. In the "normal saccade, opposite (post-saccade) stimulus" condition, the eyes saccade toward the target, but during the saccade the stimulus changes to what becomes the far periphery in the opposite direction as the saccade (see Figure 3). In this condition the initial stimulus and the final stimulus are exactly the same as in the original anti condition, only the direction of eye movement is

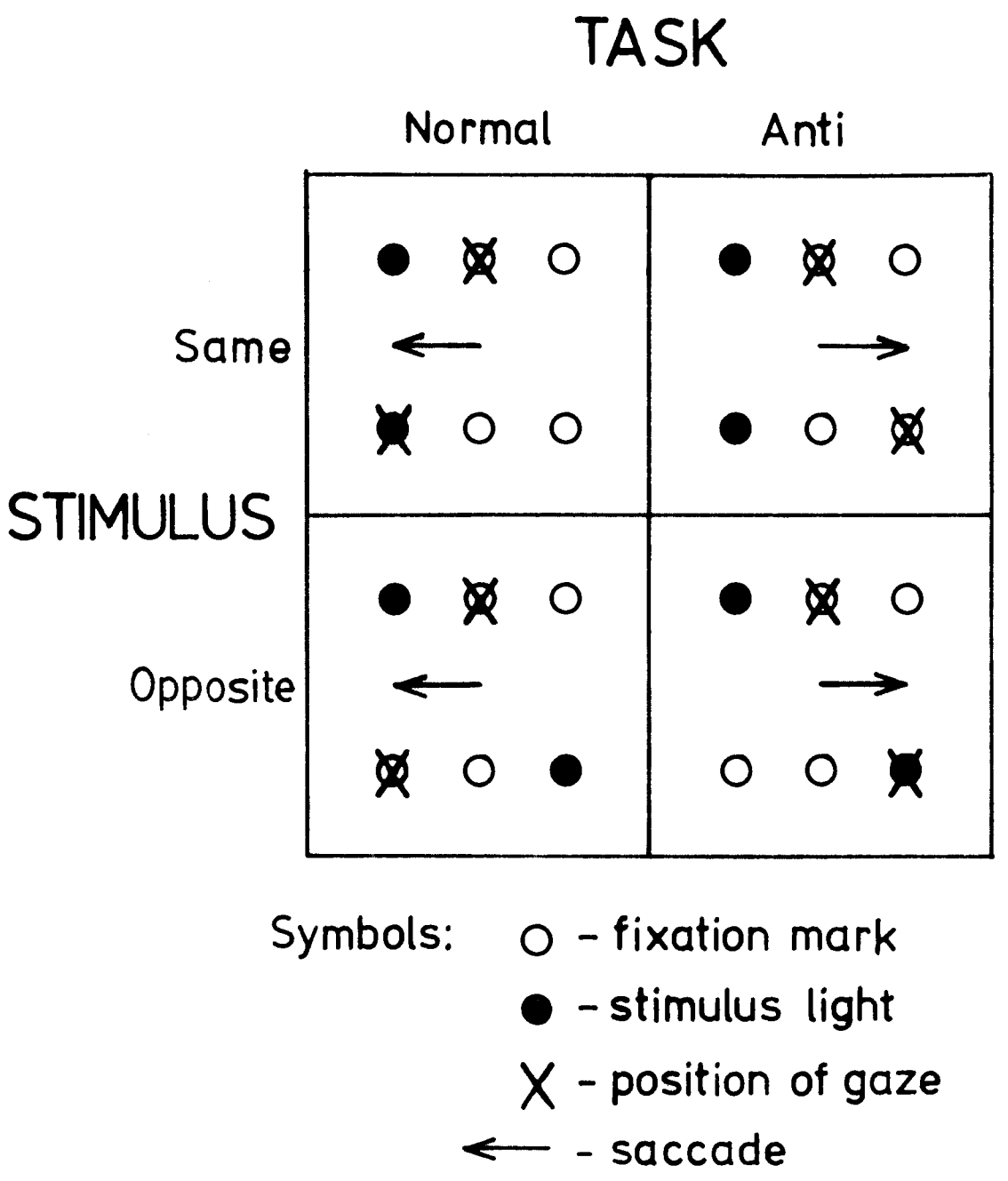


Figure 3. Schematic representation of the four reaction time conditions. The examples shown are for the left initial stimulus position only. Each box depicts the initial stimulus position prior to the saccade (top) and the final stimulus and fixation positions (bottom).

different. Similarly, the other additional condition consists of "anti saccade, opposite (post-saccade) stimulus" in which the subject is instructed to look away from the target, but during the saccade the light moves to the new position of gaze, resulting in the same foveal stimulus as in the original normal condition.

In addition to the four RT conditions described above and the self-initiated condition mentioned earlier, one more condition is necessary in order to investigate the evoked potential to the stimulus. In the "no saccade" condition the same visual stimuli will be presented, but the subject is instructed to maintain fixation. Results from this condition will be of use in identifying the stimulus-evoked potentials observed in the four RT conditions.

Methods

Apparatus and data recording techniques

EEG was recorded with Beckman Ag-AgCl electrodes attached at standardized locations according to the international ten-twenty system (Jasper, 1958; see Figure 2). Three channels were derived from frontal (Fz), precentral (Cz or vertex), and parietal (Pz) sites along the midline, referenced to a linked pair of electrodes attached to left and right mastoids. In

addition, one bipolar derivation was obtained from electrodes located over the central sulcus at homologous left and right positions (C3 and C4, respectively). Horizontal eye movements were recorded as the electrooculogram (EOG) from another pair of electrodes placed at the outer canthi of the two eyes. An electrode on the forehead served as a ground.

At each electrode site the skin was first prepared by cleansing with rubbing alcohol and light abrading with Redux electrode paste, which contains ground quartz. The electrodes placed on the scalp were attached with Grass EC2 self-adhesive electrode cream; for the other electrodes adhesive collars were used.

Three channels of EEG derived from the midline were amplified by Grass model 10 amplifiers set for a one-half amplitude bandpass of .3 to 300 Hz. As only three functioning Grass amplifiers were available, the C3-C4 derivation and the EOG were amplified with other physiological amplifiers designed at Penn State University which were set for 3 db frequency cutoffs at .07 and 500 Hz.

All four channels of EEG were amplified with a gain of 5000 and, along with the amplified EOG, were conducted to a 12-bit analog-to-digital converter controlled by a Data General Nova 3/D minicomputer. The EOG was also used to detect the beginning of each saccade by means of an electronic trigger. The amplified

EOG signal was full-wave rectified and input to a voltage comparator circuit which produced a pulse cutput when there was a change in EOG corresponding to approximately 2 degrees of eye movement.

For each single trial the computer digitized 1024 data values at one msec intervals for each channel. At the beginning of each trial a 900 msec epoch of the most recent data was continuously stored in memory which enabled data collection for each trial tc start 900 msec before the trigger pulse and end 124 msec after (see Figure 4.).

A digital marker channel was also recorded alorg with the five analcg data charrels. For the four RT conditions a marker signal turned on at the time of stimulus onset and turned off when the EOG circuitry detected a saccade. This marker signal therefore indicated the subject's reaction time.

The marker channel also recorded a second marker pulse of lower amplitude which was activated by the EOG trigger. Beca use the trigger circuitry was a.c. coupled to the EOG signal, the trigger output "bounced" following the saccade and the marker pulse is therefore not interpretable with respect to kinematics of the eye mcvement. Not indicated on the marker channel is the visual stimulus presented immediately following the onset of the saccade, which was not affected by any bounce in the trigger

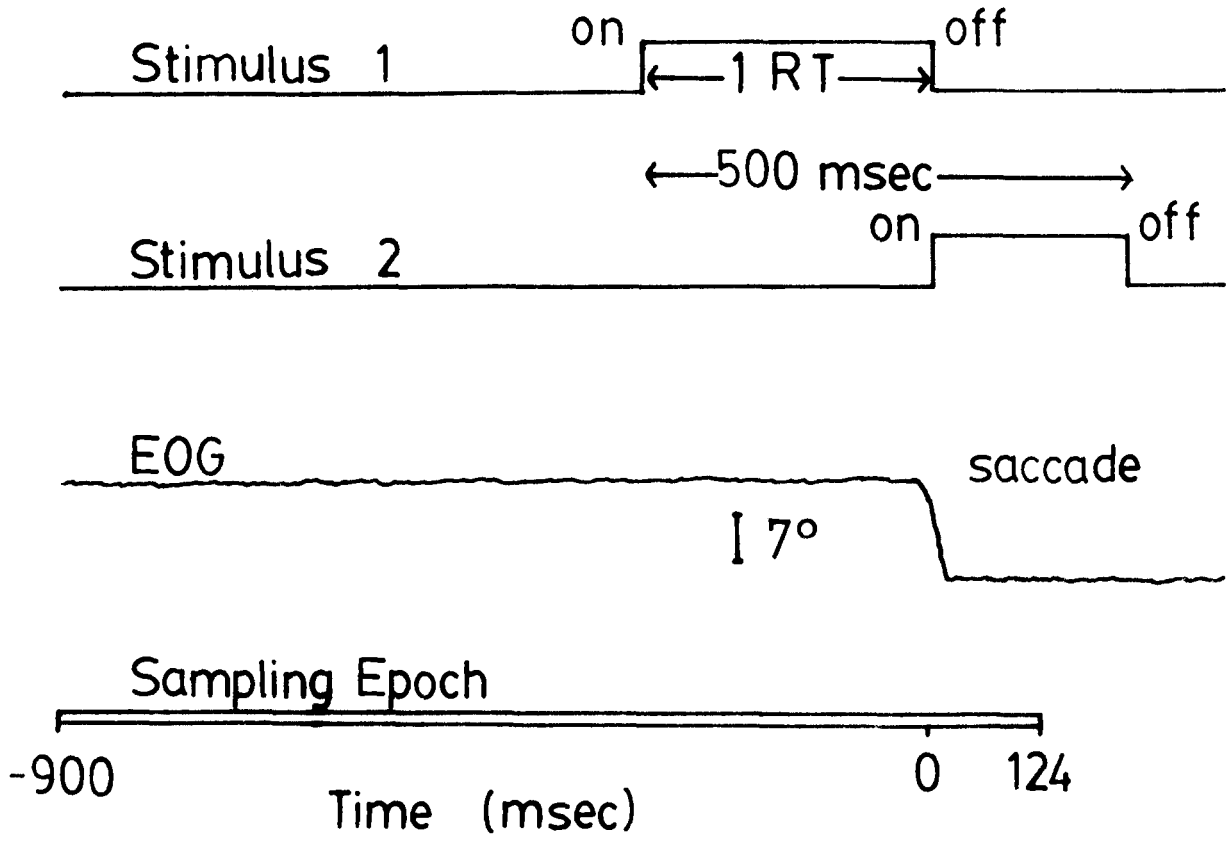


Figure 4. Timing diagram for the RT conditions. Shown is a simulated trial with a RT of 300 msec.

output.

The subject sat in a comfortable chair with a high cushioned back which provided support for the head. A bite bar or chin rest was not used as pilot work had found that these restraints produced an increased amount of myogenic artifact in the recordings.

The subject faced a translucent screen 1 m away upon which the visual stimuli were back-projected. The screen was placed in a window connecting the electronically-shielded subject room and the adjoining laboratory control room. Lights in the control room produced a constant screen luminance of .7 cd/sq.m and provided the only illumination in the subject room. Attached to the screen were three circular fixation marks located at the center and 14 degrees left and right. One of these fixation circles is illustrated on the title page where it surrounds the "C" in the copyright symbol. The visual stimuli were circular dots of light, slightly less than 1 degree in diameter, which were presented at the location of the left or right fixation marks. The lights were projected by two slide projectors with electronic shutters. The luminance of the stimuli on the screen was 5.2 cd/sq.m. The computer and logic circuitry in the lab were programmed so that for each trial initiated by the experimenter, one shutter would open and remain open until either 500 msec elapsed or the EOG trigger detected the

beginning of a saccade. When a saccade was detected the first shutter would close and the other one open for the remainder of the 500 msec period. As measured by a photocell, the delay from the EOG trigger to the second shutter opening was not more than 7 msec. The second slide projector contained slides that were either the same (left or right stimulus) as those in the first projector, or else different (opposite side). If both slides were the same the stimulus would appear stationary to the subject, but if the slides were different the stimulus would appear to move to the opposite side of the screen when the subject made a saccade. In the "same" condition the projectors were always switched when the eyes moved, even though the identical stimulus was presented, in order to ensure that any possible instrumentation artifacts were equal for both "same" and "opposite" conditions.

Subjects

Seven male and five female university students served as subjects. An honorarium of $7 was offered for participation. An additional three subjects were run but the resulting data were not used, for the following reasons. In one case the subject's EOG showed abnormal eye movements (dysconjugate saccades). For the other two subjects the combination of subject error (looking in the wrong direction) and malfunction of the recording equipment or experimenter error resulted in too

few acceptable trials for at least one experimental condition.

Procedure

When the subject arrived the various experimental conditions and the electrode application procedure were briefly explained and the subject was asked to sign a consent form.

After this was done and the electrodes were applied the subject was given more detailed instructions and a series of practice trials. The four conditions in which the subject's task was to move their eyes in response to the visual stimulus (referred to as the RT conditions) are depicted in Figure 3. The "normal" task was to maintain fixation on the center fixation mark until the spot of light appeared to the left or right, and then to make a single eye movement and fixate the light. The eye movement was to be made as fast as possible. In the "same" stimulus condition the light would remain at the same location at which it first appeared, while in the "opposite" stimulus condition the light would shift to the opposite side of the screen when the subject's eyes started to move. In the latter case, the subject was instructed not to try and follow the light, but to maintain fixation at the location the light first appeared.

For the other two RT conditions the subject's task was, when the

light appears at the left or right, to move the eyes as fast as possible and fixate the mark on the opposite side ("anti" task). In the "anti-same" condition the light remained where it first appeared, while in the "anti-opposite" condition the light would move to the opposite side of the screen when the subject's eyes started to move. The result of this is that even though the subject looked away from the original stimulus, the light would be at the center of gaze after the eye movement. The subject was instructed to only make a single eye movement and not to attempt to look away a second time. Before each trial the subject was given a verbal "ready" signal at a variable interval of a few seconds before the experimenter initiated the trial. The subject was instructed to fixate the center fixation mark and minimize eye blinks and head movements following the "ready" signal. The subject was not allowed to chew gum during the experiment. At least five practice trials were conducted for each of the four RT conditions until the experimenter felt that the subject understood the instructions and was performing reliably.

Two other control conditions attempted to separate the evoked potential from the movement-related potential. In the "self-initiated" condition no visual stimulus was presented. The experimenter would simply say "left" or "right" after the "ready" signal. The subject was instructed to first fixate at center following the "ready" signal and to wait a couple of

seconds and then move the eyes to the appropriate left or right mark on the screen. The experimenter made sure that the subject did wait a short time before making a saccade. Left and right movements were alternated, except if some artifact occurred or the trigger failed the same movement was repeated.

The other control condition was intended to investigate the evoked response to the visual stimulus. In the "no saccade" condition the subject was instructed not to move the eyes at all, but to maintain center fixation until the light turned off after 500 msec. The subject was instructed to simply "detect" the stimulus. An equal number or left and right stimuli were presented in random order.

For each subject a single run of 20 trials was executed for each of the six conditions. One of the two control conditions was presented first and the other control condition was presented at the end of the experimental session. The order of the control conditions alternated over subjects. The order of the four RT conditions was randomized over subjects.

Results

Preliminary data treatment

For each subject the data collected during the experimental

session consisted of 20 trials for each of the six conditions. As the first stage of analysis the EOG channel of each trial was visually inspected in order to identify those trials not to be included in the averaging process. Trials were rejected if there was evidence of an eyeblink, if the subject made an eye movement in the wrong direction or of longer latency than 500 msec, or if the trigger circuit malfunctioned and "detected" a saccade early or late. Because of these rejected trials the minimum number remaining for each average of left and right saccades was six (out of a possible ten). For each condition the left and right trials were first averaged separately and then these averages were averaged together, in order to ensure that the final average was weighted equally for left and right saccades. Consequently, the averages show an EOG record that is flat except for the effects due to a difference in reaction time for left and right movements.

The averages to be described in the following sections were calculated in two different ways. Since the original data were collected with respect to the onset of the saccade the usual type of averaging yields event-related potentials which are time-locked to the response and which will be described under the heading "movement potentials." The onset of the visual stimulus occurred at a varying time before the response (corresponding to one reaction time). The averaged marker signal showing stimulus onset will therefore indicate the range

of reaction times, reversed in time. Since in the method of this study the marker signal was recorded via the computer's digital sense lines rather than as a digitized analog channel which would unavoidably contain some noise, median reaction time can be calculated with 1 msec precision and without any loss of accuracy simply by locating the half-amplitude value in the marker channel average.

The other method of data averaging used in this study was required in order to investigate the evoked potentials to stimulus onset. This was done by first shifting in time all six channels of recorded data for every individual trial so that the stimulus onset, as recorded on the marker channel, was located at the same time point for each trial. When these re-oriented trials are averaged, the resulting waveforms are equal to what would have been obtained if the data sampling was originally synchronized to the stimulus, which is the normal method for evoked potential research. The marker channel, so averaged, will show the abrupt onset of the initial stimulus. However, it should be pointed out that because of the other marker pulse from the EOG trigger recorded at the time of the saccade, the gradual offset of this marker does not accurately indicate reaction times.

Reaction times

From the averaged marker channel the median reaction time was

found for the four applicable conditions for each subject. Means for all 12 subjects are graphed in Figure 5. Raw data are included in Appendix A. Contrary to expectations, there was no significant effect of the normal vs. anti task. Averaged over the same/opposite stimulus condition the normal task resulted in saccade latencies only 2.2 msec different from the anti task. Of the four conditions, normal-same showed the fastest RT, normal-opposite the slowest, while both anti conditions were intermediate. A 2x2 analysis of variance performed on these data found no significant main effect or interaction (see Appendix B).

Evoked Potentials

For the four RT conditions the individual trials which were recorded with respect to the occurrence of the response were also re-oriented and averaged about the onset of the stimulus light. Stimulus onset for these averages was set to occur 500 msec after the beginning of the record. Since for these averages the data are synchronized with respect to the visual stimulus, the averaged waveforms can be interpreted as containing the evoked potential (EP) to the light onset, although pre-movement potentials will also contribute at longer latencies. As the first step in analyzing across-subject condition effects grand averages were computed from all twelve subjects' data. Figure 6 presents these stimulus-oriented

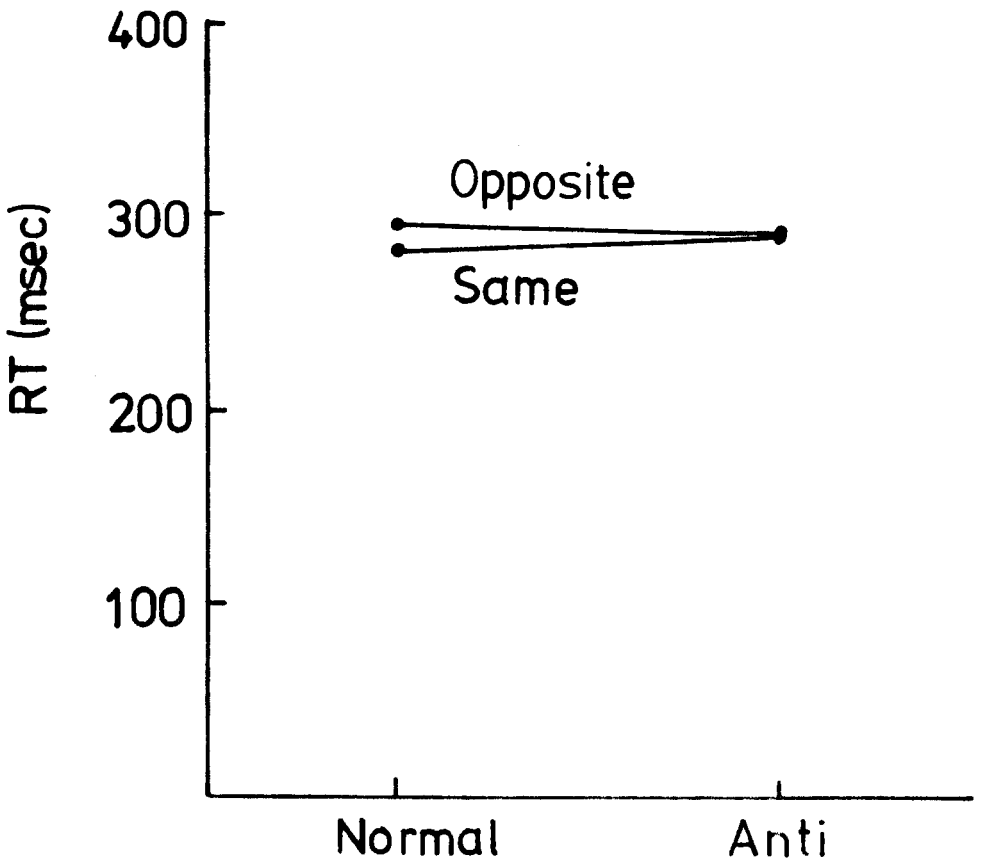


Figure 5. Reaction times (latencies of saccades) for four experimental conditions. Values shown are means of all 12 subjects' median RTs.

averages for the four RT conditions and the no saccade condition. One prominant feature in all the midline EEG averages is the negative (downward) peak which appears in both the RT and the no saccade conditions. For these grand averages the latency of this peak falls within the range of 167 to 175 msec for all conditions and electrodes.

Averages for each of the 12 subjects were visually inspected for a negative peak with a latency close to that found for the grand averages. Data for 11 of the 12 subjects showed a clear peak within the range of 150 to 200 msec. The other subject, MW, did not appear to produce a consistent negative EP component and was therefore excluded from the following analysis.

Prior to computation of the peak latencies and amplitudes the individual averages were filtered with a brick-wall digital low-pass filter of 51 points. This filter replaces each data point by the mean of 51 contiguous data values centered about the original point. The rather large window size chosen for this filter was used to reduce variability in amplitude produced by some of the more "spikey" peaks.

From the filtered averages the most negative point was found in the range of 150 to 200 msec latency. The amplitudes reported are defined as the difference between the value of this point and a baseline calculated as the mean of the first 400 data

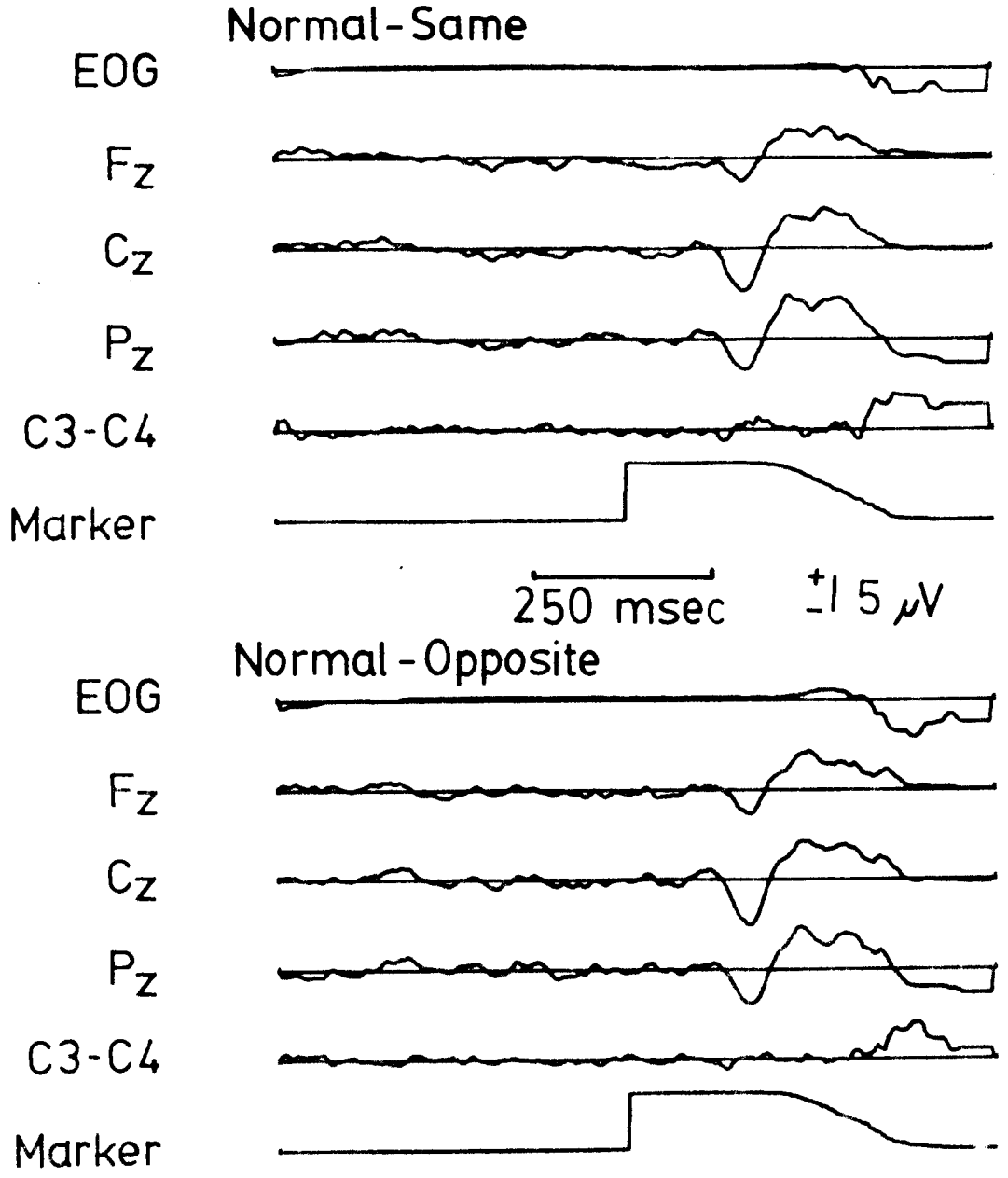
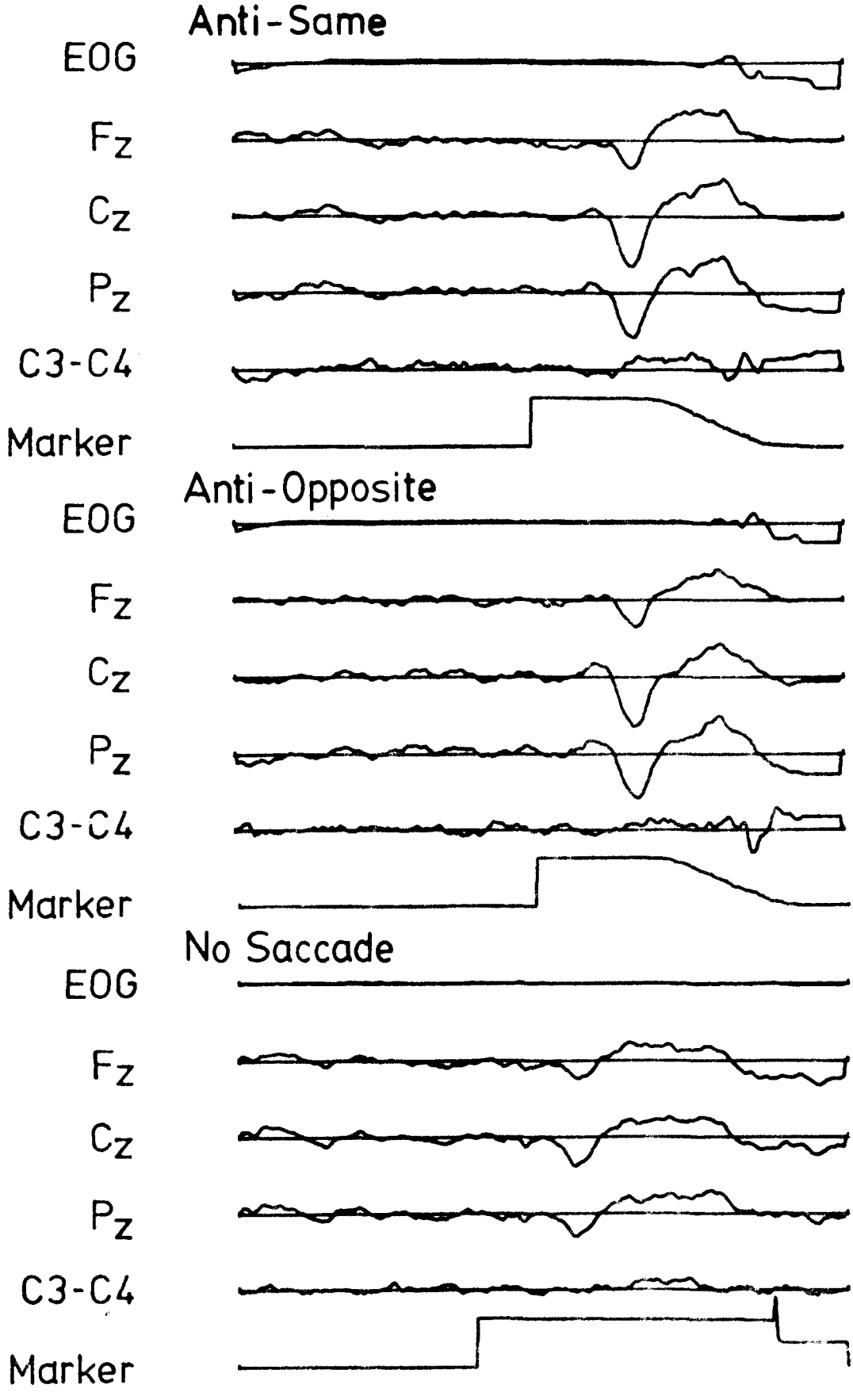


Figure 6. Grand averages for all 12 subjects of stimulus-oriented trials. Sweep duration is 1024 msec. Marker channel shows duration of initial stimulus light which was turned off at saccade onset. Calibration applies to all EEG channels. (Figure continued on following page.)


Anti-Same
EOG
Fz
Cz
Pz
C3-C4
Marker
Anti-Opposite
EOG
Fz
Cz
Pz
C3-C4
Marker
No Saccade
EOG
Fz
Cz
Pz
C3-C4
Marker

points in the average, which was the maximum number of points collected preceding the stimulus onset for all individual trials.

Mean evoked potential amplitudes for the 11 subjects are plotted in Figure 7 and the original measurements are included in Appendix C. Although there was a some amount of inter-subject variability in the latencies of the peaks found, the mean amplitudes plotted are very similar in form to the amplitudes of the peaks in the grand averages presented in Figure 6, and all the actual values are different by less than one microvolt. For both the grand averages and the subject means, the largest potentials were observed at Cz and the smallest at Fz. Also, this pattern of scalp distribution is the same for each of the five conditions.

In order to test the reliability of the task and stimulus-condition effects a 2x2 analysis of variance was performed on the data for each electrode separately (see Appendix D). The major finding was significantly greater amplitudes in the anti task than in the normal task for electrodes at Pz, $F(1,10) = 20.4$, $p < .005$; and at Cz, $F(1,10) = 7.9$, $p < .025$; while the difference at Fz was not significant $F(1,10) = 3.0$, $p > .1$. For no electrode was there a significant effect of the same vs. opposite stimulus condition, nor was there any significant interaction. The latencies of the N1

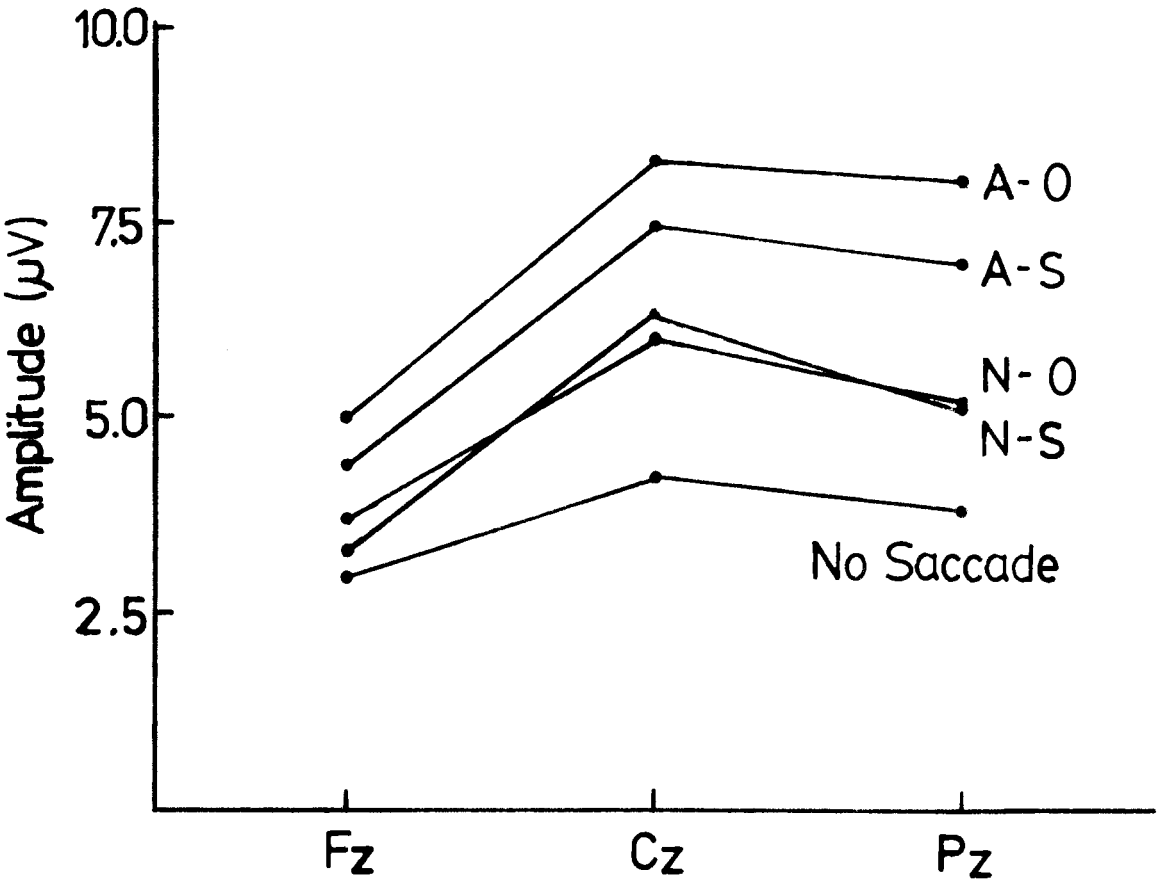


Figure 7. Negative evoked potential of 150 to 200 msec latency. Mean amplitudes for 11 of 12 subjects. Abbreviations: A - anti, N - normal, S - same, and O - opposite.

peaks were analyzed, but no reliable latency effects were found for the relatively small latency differences observed.

Some attempt was also made to identify and measure other components in the evoked response. In many of the plots in Figure 6 there is a positive peak which occurs before the larger negative peak. However, this component could not be reliably located in any more than the majority of the subjects' averages and therefore was not further pursued. The later positive-going wave also evident in the stimulus-averaged data was also not further investigated for the reason that its latency is longer than a minimum reaction time and it is therefore inextricably confounded with movement potentials, the topic of the following section. No consistent left/right asymmetries were observed from the C3-C4 derivation.

Movement potentials

Figure 8 presents the response-oriented grand averages for the four RT conditions, as well as the self-initiated condition in which the subject made a delayed response to a verbal instruction to move left or right. The most conspicuous feature of the potentials preceding eye movement in the self-initiated condition is the absence of much activity at all. For the method of this study there is no noticeable negative-going potential (the BP) which Becker et al. (1973) reported to be

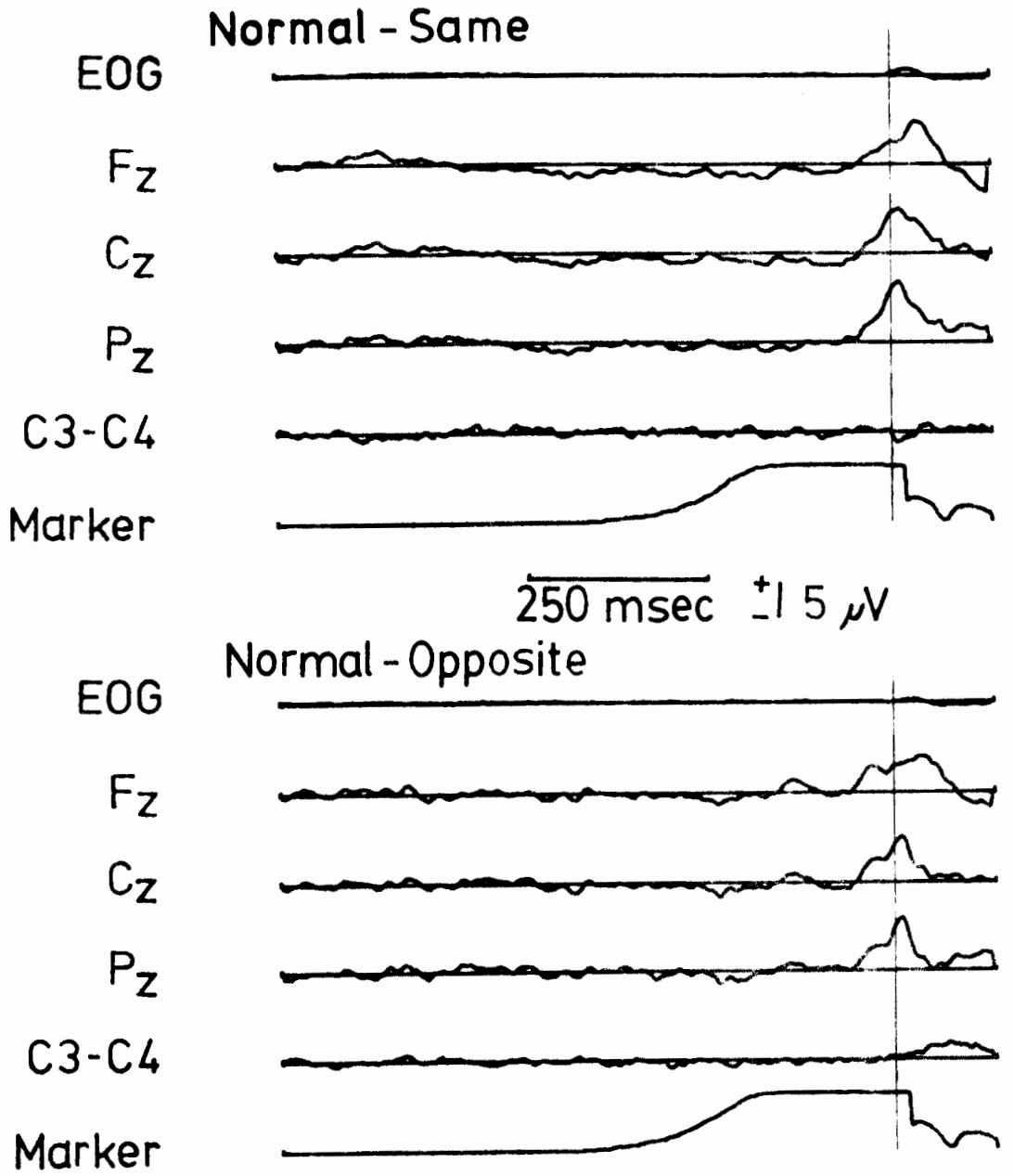
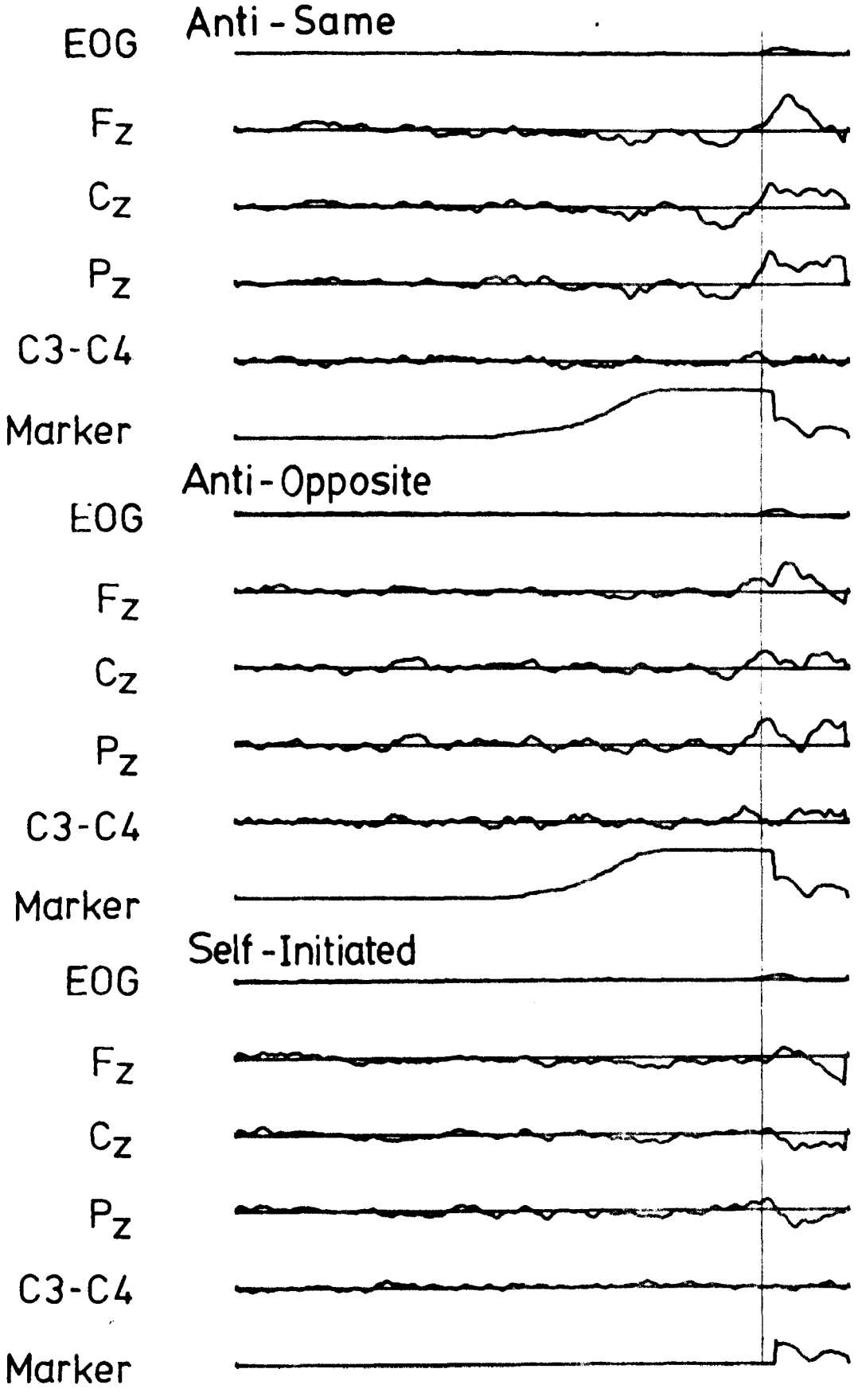


Figure 8. Grand averages for all 12 subjects of response-oriented trials. Sweep duration is 1024 msec. Marker channel shows stimulus light onset. Line indicates point of first EOG change, approximately 20 msec before trigger. Calibration applies to all EEG channels. (Figure continued on following page.)


Anti - Same
EOG
Fz
Cz
Pz
C3-C4
Marker
Anti - Opposite
EOG
Fz
Cz
Pz
C3-C4
Marker
Self - Initiated
EOG
Fz
Cz
Pz
C3-C4
Marker

almost 5 microvolts in amplitude at Cz.

Looking at the RT condition averages in Figure 8, the normal condition averages especially appear very similar to those from the "triggered" condition of Kurtzberg and Vaughan (1979). The averages show as a primary feature a positive-going potential which will be referred to in Becker et al.'s (1973) terminology as the Pre-Motion Positivity (PMP). The C3-C4 derivation in Figure 8 shows no left/right asymmetries, consistent with Becker et al.'s reports. The condition effects on the PMP were measured using the peak detection software already described. Visual inspection of separate averages of left and right movements from individual subjects determined that the EOG signal showed its first deflection approximately 20 msec prior to the response of the digital trigger recorded in the marker channel which was therefore taken to be the time of saccade onset. In order to quantify the PMP amplitudes the averages were first digitally filtered over a 21 point window, then the most positive point was found in the interval from 20 msec to 140 msec before the trigger occurred. The PMP was defined as the difference between this amplitude and a baseline calculated as the mean of the first 400 points in the average. The values obtained are presented in Appendix E and the means are graphed in Figure 9. The means of the PMP amplitudes calculated from the individual subjects' averages were as can be expected slightly greater than the PMP amplitudes shown in the grand

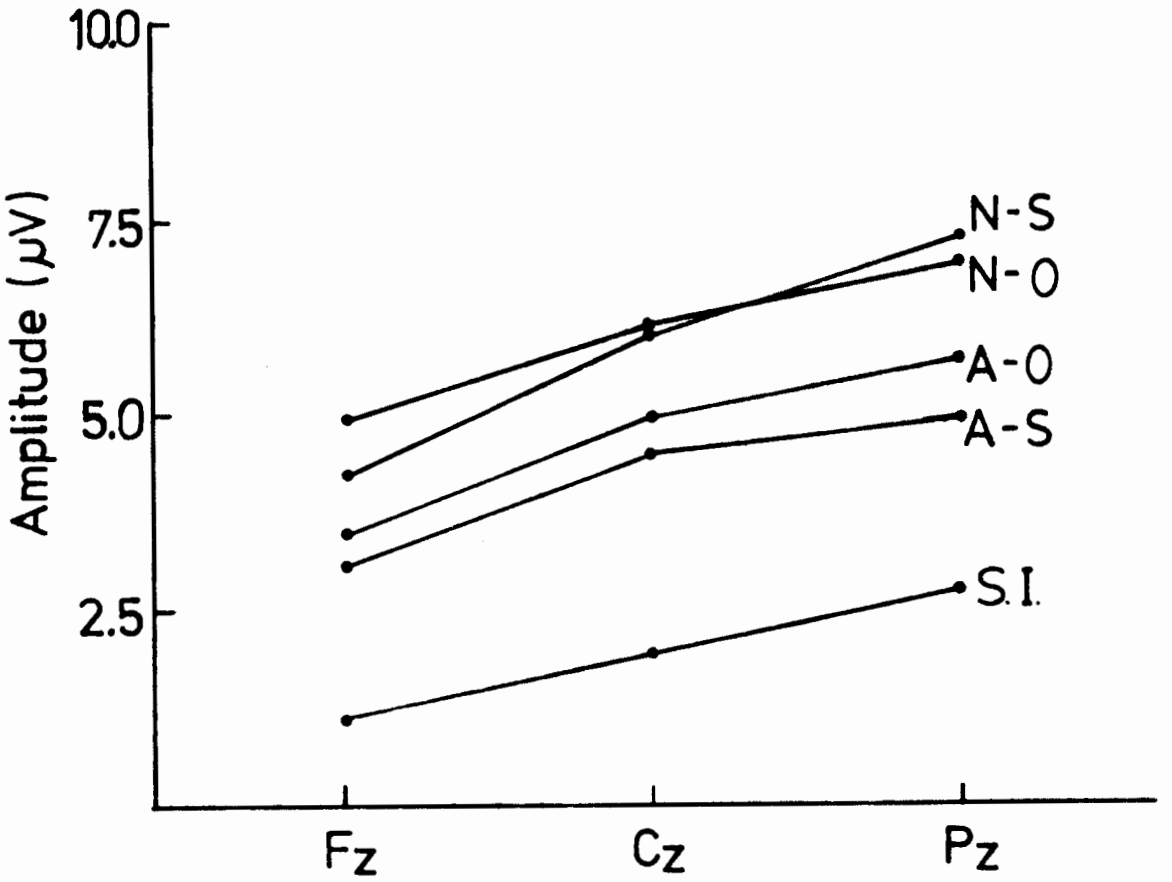


Figure 9. Maximum PMP amplitude in 120 msec interval preceding saccade onset. Data from all 12 subjects. Abbreviations: A - anti, N - normal, S - same, O - opposite, and S.I. - self initiated.

averages, especially for the Fz and Cz electrodes, but the relative condition effects were very similar.

Another 2x2 analysis of variance was performed on the data from the four RT conditions for each midline electrode. The Pz electrode showed significantly greater PMP amplitudes in the normal condition, $F(1,11) = 13.8$, $p < .005$; while the other two electrodes did not show the same effect significant at the .05 level: $F(1,11) = 4.2$, and $F(1,11) = 4.4$ for Fz and Cz, respectively. There was no other significant stimulus condition effect or interaction at any electrode (see Appendix F).

The scalp distribution for the PMP shows relatively the same form for all conditions: greatest amplitude at Pz, smallest at Fz. Based on the assumption that potentials recorded from different electrodes are not comparable on the same interval scale of measurement, analysis of variance was not used to test for differences in scalp topography. However, it may be noted that out of the 12 subjects, 11 of them showed greater Pz than Cz PMP amplitudes in most of the five conditions ($p < .02$, sign test). Also, the relative amplitudes observed at Cz and Pz are in the opposite direction for the negative EP and the PMP for all five relevant conditions, four of which are constituted from the same original data. Nine of eleven subjects show a greater Pz than Cz amplitude in more of the five relevant conditions for the PMP measure compared to the EP measure. Using a Wilcoxon

rank sum test, the value of T found was 11.5, which corresponds to just slightly more than the .05 level of significance.

Discussion

Reaction times

An unexpected result of this study was the absence of any significant difference in reaction times for the four experimental conditions. This non-effect is less of a surprise for the same/opposite stimulus conditions as there was no previous evidence that it did influence RT. For the normal and anti tasks, however, the present results were not expected based on Hallett's (1978) finding of much longer latencies for his anti condition.

The most plausible post-hoc explanation for these conflicting results concerns the complexity of the subject's task in the two experiments. The major difference in the methods is that Hallett's stimulus moved to one of eight equally likely positions. The anti condition in the simpler method of this study requires only that the eyes make a practiced movement to the single location at which the stimulus is absent. Hallett's method requires that the saccade is directed to the correct one of seven locations. In order to do this the subject must evaluate both the direction and the magnitude of the stimulus

step and program a saccade of equal magnitude but opposite direction. In the present experiment the required left and right movements are all of the same magnitude and can therefore be preprogrammed. Following the presentation of the stimulus the subject needs only to program the correct direction of movement based on the stimulus location.

An assumption inherent in the above analysis is that the direction and magnitude of saccades are not programmed simultaneously by the same mechanism but are at least in part independently programmed. This statement is consistent with other recent research using double step stimuli which found evidence for independent programming of direction and magnitude (Becker & Jurgens, 1979; Hou & Fender, 1979). In addition, the two studies cited have also indicated that the programming of direction takes less time and is therefore completed before the programming of magnitude. The increased RTs in Hallett's experiment can then be explained if the time required to program the magnitude but not the direction of a saccade is greater for the anti task.

Another possible explanation that must be considered for the difference in results is that the total number of possible responses was greater in Hallett's experiment and this factor produced greater latencies in the anti condition. Relevant to this, a study by Saslow (1967b) showed that for the normal task

separate blocks of trials within which the number of possible stimulus displacements was two, four or eight did not result in different RTs. Even if this is true for the normal task, it might be the case that the anti task is influenced by the number of possible responses even though the normal task in not.

An experimental test of these questions could be made by means of the following two simple experiments. The first suggested experiment would use the same method as the present study, except that the two stimulus positions used would both be on the same side of the fixation mark (left and right counterbalanced). In this situation the subject always makes a saccade in the same direction, but the correct magnitude must be determined from the stimulus location. If in this situation the results showed greater RTs for the anti task the hypothesis that the programming of saccade magnitude is lengthened in the anti task would be supported.

A second possible experiment would be to use four stimulus locations all at the same distance from the fixation point (up, down, left, and right). Here the prediction would be that the latencies would be equal for the anti task and the normal task because direction but not magnitude would have to be programmed.

One last comment on the RT results concerns the S-R compatibility effect discussed in the introduction. The

prediction of a simple version of S-R compatibility, i.e. due to the necessity of interhemispheric transfer, responses are slower when made by the side of the body opposite the stimulus, is not supported by the present RT results. Other recent research on manual responses has also shown that "S-R compatibility" is definitely not as simple as the above statement would indicate (Cotton, Tzeng, & Hardyck, 1980).

Event-related potentials

A positive aspect of the negative results discussed above is that there are no large RT differences that have to be considered when interpreting the other results.

One of the clearest findings of the present study which was not originally expected is the task effect on the negative evoked potential to the target onset. Generally, small peripheral lights which are not much brighter than the background do not produce very large evoked potentials (e.g. Regan, 1972). As was found earlier in pilot work and as the present results show the EP was not very large in absolute magnitude and few components were reliably observed in most subject's data. However, there was at least a single consistent negative peak which could be located within a narrow latency range for almost all subjects. This component appears to be the same as that referred to as N1 in the evoked potential studies cited in the introduction. The

actual latencies and waveforms obtained in the present experiment appear remarkably similar to those reported in the study by Eason et al. (1969) which used approximately the same type of stimulation.

The present results are consistent with those of Eason et al. (1969) and also Kurtzberg and Vaughan (1979) in that the evoked potential is larger for those conditions requiring a response in comparison to the no saccade condition in which the subject was told to "detect" the stimulus but not to respond with any movement. Not expected, however, was the significantly greater amplitudes for the anti task at Pz and Cz. The admittedly post-hoc explanation proposed for this effect is that the N1 component is related to the amount of selective attention directed to the stimulus (see literature cited in introduction) and that subjects are more attentive to the stimulus in the anti condition. Greater attention to the stimulus in the anti condition is supposedly caused because the task is more difficult. Indirect evidence for the last statement is the greater number of errors in the anti condition as well as the subjective responses of subjects (see also Hallett, 1978). One possibility is that if this hypothesis is true, greater attention might compensate to some degree for the greater difficulty of the anti task and decrease reaction time to values close to those for the normal task, as were observed.

The scalp distribution found for N1 was quite similar for all of the five relevant conditions and showed maximum amplitudes at vertex, less amplitude at Fz, and intermediate values at Pz. The most likely explanation for the widespread distribution of an EP component is, according to Goff et al. (1978), the existence of a potential generator which is either very large and close to the scalp or else smaller and farther away. In the present case the second situation seems more reasonable and is also consistent with the suggestion made above that the evoked response is mediated by an attentional mechanism which would more likely exist at subcortical levels.

The other major EEG measure of interest was the response-related potentials. For this measure as well as the evoked potential a single consistent waveform could be identified which was also small in amplitude. The waveform observed was similar to the PMP as described by Becker et al. (1972) and Kurtzberg and Vaughan (1979) in their more closely related study.

A detailed comparison of the results of the present study with those of Kurtzberg and Vaughan (1979) reveal numerous similarities but also major differences. In the present study all five response conditions produced the same scalp distribution which was similar to that reported for Kurtzberg and Vaughan's "triggered" condition, i.e. a parietal maximum which falls off evenly from Pz to Fz. The original expectation

was that the normal condition would show a PMP distribution such as this, but that the anti and self-initiated conditions would exhibit different distributions as Kurtzberg and Vaughan reported for their "return" and "self-initiated" conditions. These authors related the distribution differences found to hypothetical generators located in corresponding areas of cortex. Parietal cells studied by Hyvarinen and Poranen (1974), Lynch et al. (1977), and Mountcastle et al. (1975) which were activated prior to visually elicited saccades in monkeys were hypothesized to be the potential generators responsible for the largest PMP amplitudes at Pz. The other major cortical area involved with eye movements, the frontal eye fields, was thought to be more involved in generation of saccades in their self-initiated and return conditions which resulted in the more anteriorly-shifted topography observed.

The present study did not find any evidence of such a distribution difference between the normal condition and the supposedly more voluntary and less directly visually-elicited anti condition. The original expectation was that there would be a more frontal distribution shift for the anti condition. An important consideration is that the results for the self-initiated condition appears to have the same topography as both the normal and anti conditions. This would indicate that it is not simply the occurrence of the visual stimulus which produces the large amplitudes at Pz. Rather it seems that the

distribution (but not the amplitude of the PMP) is similar for all conditions in which exactly the same movement occurs. This statement is in line with the position expressed by Becker et al. (1973) that the PMP represents the actual efferent command for movement. If such is the case, the final command to contract particular extraocular muscles exactly the same way would likely originate in the same generator mechanism regardless of different sensory input and processing activities which precede it.

A possible source of some difference in the results of the present study and that of Kurtzberg and Vaughan (1979) is the actual method used for quantifying PMP amplitude. Kurtzberg and Vaughan's (1979) preliminary paper does not give any details of this and also expresses all results in terms of percent maximum amplitude. Due to this last-mentioned procedure it is not possible to compare the relative PMP amplitudes for different conditions between the two studies. The present investigation found significantly larger PMPs for the normal condition (at least at Pz). This result, together with the lack of appreciably faster RTs for the normal condition, does suggest that the PMP is sensitive to the particular task in which the eye movements are performed.

An alternate explanation for the task effect on the PMP amplitude that must be considered is that the earlier negative

evoked potential acts to reduce the size of the opposite-polarity PMP. It is the case that compared to the normal condition, the anti condition resulted in greater EP negativity and less PMP, indicating a possible dependency between the two waveforms. Not consistent with this hypothesis are the following four reasons. 1) From the results for the self-initiated condition it can be seen that when there is no negative EP the PMP is smallest in amplitude, therefore it cannot be just the EP alone which produces the observed PMP differences. 2) The data from the no saccade condition (Figure 6) show that EEG following the N1 peak tends to go positive and not remain negative. 3) The correlation of mean PMP and EP amplitude for all four experimental conditions combined, computed for the 11 subjects is -.07 (n.s.). 4) There is evidence of a difference in the scalp distribution of the EP and the PMP, which supports the idea of independence.

In conclusion, however, there is no absolutely convincing argument that there is no effect of the EP on the observed PMP amplitude, but there are, I think, plausible empirical and theoretical reasons to assume that the PMP differences are not found only because of influence from the evoked potential. Having made this assumption, what might be the physiological processes which produce these results? Following Becker et al.'s (1973) discussion of the PMP, it is suggested that the neural command to move the eyes is generated in some unknown but

possibly parietal location in all the conditions. The reduced amplitude in the self-initiated condition, in which there was no imperative stimulus the subject had to respond to, might result from less rapid generation of the command signal. For manual movements it has been reported that force, speed, and asscciated EMG activity are usually less for self-initiated movements than for RT responses (Rohrbaugh et al., 1980). Possibly this "less brisk" manner of responding, extrapolated back to before the onset of movement, would explain the reduced PMP.

The observed PMP amplitude difference found for the two task conditions is not associated with a corresponding difference in RT. The speculative hypothesis suggested here is that the anti task, because it is more difficult and less like usual stimulus-elicited saccades requires additional processing, such as activation of inhibitory mechanisms to suppress the normal response. This increased activity not directly related to the generation of the command to move the eyes results in greater cortical desyncronization of PMP-related neural events and a reduction in the summated potentials.

A final point of speculation concerns the relationship of the task-condition effect on PMP and EP amplitudes and the S-R compatibility effect discussed earlier. Might it be the case that for incompatible responses generally the sensory response to the stimulus is greater and the movement-asscciated

potentials are reduced?

Conclusion

The integration and processing of sensory information for the programming and generation of motor acts is, according to such authors as James, Sherrington, and Granit, the essential problem for psychology. In this author's opinion sensory-motor integration is demonstrated at the highest level of exquisite complexity in what Gibson refers to as the perceptual system of "seeing."

The present research has only investigated a single artificial example of an even trivial aspect of this system's capacity: left or right saccades to one of two possible target lights flashed on a screen. Yet because of the nervous system's unknown mechanisms for intricate interaction of sensory and motor function, this research has demonstrated what might be considered to be an effect of the higher-order relationship between the intended future movement and stimulus position on electrical correlates of sensory processing (the evoked potential) as well as the efferent command for movement (the PMP).

Appendices

Appendix A. Median Reaction Times (msec)

	Condition			
Subject	NS	NO	AS	AO
GH	268	314	266	298
BK	283	267	281	280
SJ	292	301	355	339
PG	278	273	275	287
DV	371	388	421	413
TA	265	325	304	299
SR	268	282	238	241
CS	265	266	303	284
KF	292	297	238	230
MW	337	342	314	348
PC	232	230	253	251
RM	227	270	231	237
MEAN	282	296	290	292

Appendix B. RT ANOVA

Labels: S - subjects
T - task (normal/anti)
C - stimulus condition (same/opposite)

	SOURCE	ERROR TERM	F	SUM OF SQUARES	DEG. OF FREEDOM
1	MEAN	S	511.1072	4036220.	1
2	S			86867.13	11
3	T	ST	0.0613	58.52083	1
4	C	SC	3.6225	875.5208	1
5	ST			10509.66	11
6	SC			2658.604	11
7	TC	STC	2.8129	462.5208	1
8	STC			1808.727	11

Appendix C. N1 evoked potential data.

Subject	Condition														
	No Sac.			NS			NO			AS			AO		
	Fz	Cz	Pz	Fz	Cz	Pz	Fz	Cz	Pz	Fz	Cz	Pz	Fz	Cz	Pz
GH	191	151	106	103	141	69	97	177	123	79	116	87	125	167	145
BK	78	154	105	78	222	230	107	170	124	242	307	271	217	284	216
SJ	100	114	109	108	169	137	144	156	127	111	132	98	126	180	189
PG	47	73	16	17	4	-70	83	93	32	4	17	-25	22	29	-29
DV	-24	2	48	102	170	207	77	152	189	66	216	264	109	266	318
TA	-22	14	33	5	53	45	-28	40	38	19	68	87	76	128	99
SR	162	141	170	140	171	204	151	67	120	199	186	233	132	142	182
CS	-52	-68	-21	6	21	0	4	6	56	-33	2	13	37	95	158
KF	-18	90	107	14	164	153	14	151	161	8	185	198	45	206	198
PC	56	94	89	62	160	123	45	117	92	89	153	140	86	145	152
RM	138	169	80	100	129	30	129	207	95	197	263	199	135	202	162
Mean	60	85	77	67	128	103	75	122	105	89	150	142	101	168	163

Note. All values are in raw A/D units (5 microvolts = 102.4).

Appendix D. N1 ANOVA

Fz Electrode

	SOURCE	ERROR TERM	F	SUM OF SQUARES	DEG. OF FREEDOM
1	MEAN	S	22.3804	302618.2	1
2	S			135215.4	10
3	T	ST	2.9892	6456.566	1
4	C	SC	2.3128	1070.204	1
5	ST			21600.00	10
6	SC			4627.355	10
7	TC	STC	0.0363	38.20581	1
8	STC			10512.11	10

Cz Electrode

	SOURCE	ERROR TERM	F	SUM OF SQUARES	DEG. OF FREEDOM
1	MEAN	S	46.2461	881828.0	1
2	S			190681.4	10
3	T	ST	7.8872	12750.02	1
4	C	SC	0.2726	390.0227	1
5	ST			16165.54	10
6	SC			14308.48	10
7	TC	STC	1.2670	1620.208	1
8	STC			12788.04	10

Pz Electrode

	SOURCE	ERROR TERM	F	SUM OF SQUARES	DEG. OF FREEDOM
1	MEAN	S	31.0550	722945.4	1
2	S			232794.9	10
3	T	ST	20.3876	26020.45	1
4	C	SC	0.5336	1466.273	1
5	ST			12762.86	10
6	SC			27479.98	10
7	TC	STC	0.7363	873.0906	1
8	STC			11857.97	10

Appendix E. PMP data.

Subject	Condition														
	SI			NS			NO			AS			AO		
	Fz	Cz	Pz	Fz	Cz	Pz	Fz	Cz	Pz	Fz	Cz	Pz	Fz	Cz	Pz
GH	79	50	62	-56	-37	29	127	62	78	-1	3	25	-13	60	69
BK	33	78	87	131	25	45	31	-15	10	-12	-20	-15	18	-27	-8
SJ	13	28	86	147	222	194	98	186	156	-5	5	49	82	117	120
PG	8	22	42	132	236	277	63	124	177	123	184	224	41	63	128
DV	50	82	68	210	236	252	237	261	266	229	284	270	161	169	185
TA	12	7	22	128	138	138	176	163	168	168	130	77	201	140	86
SR	19	117	120	-51	8	-32	-46	16	-10	-161	-34	-84	-92	-40	-82
CS	1	14	87	8	3	62	63	2	36	67	12	72	28	17	29
KF	14	-28	-18	80	3	42	115	50	67	16	-37	-51	43	60	64
MW	-18	39	12	21	287	384	117	370	403	117	308	363	124	301	370
PC	-3	38	55	110	105	110	49	50	76	70	113	134	62	96	118
RM	61	33	41	184	251	278	175	220	269	142	158	145	201	258	321
Mean	22	40	55	87	123	148	100	124	141	63	92	101	71	101	117

Note. Values are in raw A/D units (5 microvolts = 102.4).

Appendix F. PMP ANOVA

Fz Electrode

	SOURCE	ERROR TERM	F	SUM OF SQUARES	DEG. OF FREEDOM
1	MEAN	S	12.2598	310086.8	1
2	S			278221.5	11
3	T	ST	4.2146	8533.332	1
4	C	SC	0.7238	1452.000	1
5	ST			22271.92	11
6	SC			22066.31	11
7	TC	STC	0.0278	70.08203	1
8	STC			27727.44	11

Cz Electrode

	SOURCE	ERROR TERM	F	SUM OF SQUARES	DEG. OF FREEDOM
1	MEAN	S	12.2553	582120.6	1
2	S			522492.8	11
3	T	ST	4.4121	8694.082	1
4	C	SC	0.1006	300.0000	1
5	ST			21675.67	11
6	SC			32802.75	11
7	TC	STC	0.1152	192.0000	1
8	STC			18333.27	11

Pz Electrode

	SOURCE	ERROR TERM	F	SUM OF SQUARES	DEG. OF FREEDOM
1	MEAN	S	12.7143	771147.0	1
2	S			667170.5	11
3	T	ST	13.8123	1564.08	1
4	C	SC	0.0967	243.0000	1
5	ST			12442.92	11
6	SC			27632.50	11
7	TC	STC	1.1253	1564.082	1
8	STC			15288.61	11

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