A Comparison of Visually Evoked Saccadic Inhibition and Visually Evoked Potentials

by

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A thesis submitted in conformity with the requirements for the degree of Doctor of Philosophy, Graduate Department of Psychology, in the University of Toronto.

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Abstract

Participants read black text on a white background that was replaced for 33 ms with an irrelevant transient image, at random intervals of 300-400 ms. Visually evoked saccadic inhibition caused the frequency of saccades to be reduced in a narrow interval after the display change. A series of 7 experiments investigated the influence of stimulus characteristics of the display change on the latency and strength of the inhibition, and contrasted these with similar effects on visually evoked potentials (VEP). Display changes included luminance, contrast, color, spatial frequency content, and displacement. Size of the display change and the area of the visual field (foveal or peripheral) were also manipulated. Inhibition was evoked by all visual stimuli employed but not by an auditory stimulus. The fastest and strongest inhibition was evoked by large luminance changes. This inhibition began within 68 ms and suppressed up to 79% of saccades. Weaker and
slower inhibition was evoked by displacement, changes in high frequency content and low contrast and isoluminant color changes. Latency of inhibition was longer for display changes in high-frequency content compared to changes in low-frequency content. The magnitude of inhibition increased and its latency decreased for higher contrast stimuli and for larger displacements. Saccadic inhibition was only weakly dependent on stimulus size, with latency increasing and magnitude decreasing only for small stimuli. Unlike VEP, saccadic inhibition did not scale as a function of cortical area activated by the stimulus. The effects of stimulus characteristics on saccadic inhibition appear to be similar to those reported for VEP when the source of these effects is subcortical rather than cortical in origin. These findings are discussed in relation to the hypothesis that the neural locus of saccadic inhibition is the superior colliculus.
Acknowledgements

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Overview

Recently, Reingold and Stampe (in press) reported a new paradigm for measuring the effect of changes in visual input on saccade production. In this procedure, participants perform a task such as reading or visual search that produces saccades at a rate of 3 to 4 per second. At random intervals, the task display is replaced with another image for 33 ms. For example, in a reading task the normal display of text on a white background might be replaced by a black screen. The result is an irregular flickering that the participants are told to ignore. Effects of the change in the display on saccade production are analyzed by creating a histogram of the number of saccades by latency from the visual change.

Figure 1. Idealized saccade frequency histogram following a visual change. Saccades are unaffected in the baseline period immediately following the visual change, then become less likely during the period of inhibition induced by the change. This is followed by an increased number of saccades during the recovery.
A typical histogram of saccade frequency is shown in Figure 1. The rate of saccade production is unaffected for a short period after the visual change, immediately followed by a reduction in saccade frequency. This pattern suggests that the first effect of the visual change on the saccadic system is to inhibit the production of saccades. Figure 1 also shows how the time course of the inhibition can be reliably determined, by measuring the period when the dip in the histogram exceeds half its depth as measured relative to the unaffected period (baseline) immediately after the visual change. Using this criterion, the inhibition observed by Reingold and Stampe (in press) using a reading task began 70 ms after the visual change, and lasted for 48 ms. Inhibition reached its maximum strength 92 ms after the visual change, at which time the number of saccades had decreased by 75% compared to the baseline. The time course of the inhibition was also found to be highly reproducible between participants.

The quick onset of visually evoked saccadic inhibition suggests that it is a low-level, reflexive neurological response to the presence of new visual information. This is analogous to visually evoked potentials (VEP) in the electroencephalogram, that arise from the mass action of cortical neurons as they respond to changes in visual input. The latency of the peaks in the VEP are thought to be determined in part by neural delays in the visual system. Some or all of these delays may also contribute to the latency of visually-evoked saccadic inhibition. As numerous studies have found that the latency and magnitude of early peaks in the VEP are determined by low-level stimulus characteristics such as brightness and size (e.g., Osaka & Yamamoto, 1978; Meredith & Celesia, 1982), it is likely that similar effects will be found for visually evoked saccadic inhibition as well.
The goal of this thesis will be to investigate the effects of manipulation of low-level stimulus characteristics on visually evoked saccadic inhibition, and to contrast these with previous findings in the VEP literature using similar manipulations of luminance, contrast, spatial frequency, color, and area and retinal location of the visual change. The role of saccadic inhibition in psychological research will be explored by surveying previous findings of saccadic inhibition and discussing studies whose results may be explained by such inhibition. Neurological mechanisms of perception and saccade production that may be relevant to visually evoked saccadic inhibition will then be reviewed. A short introduction to VEP research and analysis will then be provided to facilitate the discussion of findings from the VEP literature. The experimental methodology employed in the current study of saccadic inhibition will be described, and measures of strength and time course of inhibition will be defined. This will be followed by individual experiments that investigate the effects of manipulations of stimulus and visual changes characteristics on visually evoked saccadic inhibition, and the results will be compared to analogous findings from the VEP literature. Finally, the experimental results of these studies will discussed, as will their implications for the nature of saccadic inhibition, and tentative constraints on its neurological substrates.
Previous Findings Related to Saccadic Inhibition

If visually evoked saccadic inhibition is a general phenomenon, then it should also be a factor in studies of stimulus-elicited saccades. It may already have been observed indirectly in such studies, for example by increasing the saccadic reaction time (SRT) in response to the onset of a target. The studies that are likely to have induced visually evoked saccadic inhibition would have included a visual change simultaneous with or delayed from the onset of the saccade target, such as the onset of a fixation target or of one or more distractors. Several such studies are reviewed below.

Saccadic inhibition should influence the distribution of saccadic reaction times (SRT) as illustrated in Figure 2. Each panel of this figure shows the expected distribution of SRTs that results from a visual change occurring simultaneously with or delayed from the saccade target onset. Panels A and B of the figure show the SRT distributions with no visual changes or distractors present. These distributions illustrate experimental conditions that produce short latency (panel A) or long-latency (panel B) saccades. For example, removing the fixation target 200 ms before target onset would create short latency saccades (gap paradigm), whereas leaving the fixation target on throughout the trial would create longer latency saccades (overlap paradigm) (e.g., Fischer & Weber, 1993).
Figure 2. Effect of visually evoked saccadic inhibition on distributions of saccadic reaction times. Vertical axis is proportion of saccades, and horizontal axis is time after the target appearance. The gray bars in each panel represents the period of saccadic inhibition. Average SRT is indicated by the arrows.Ticks are 100 ms apart. Panels A & B: SRT distributions with no visual change. Two conditions that produce short or long latency saccades are shown. Panels C & D: Visual change simultaneous with saccade target onset. Only the lowest-latency saccades in C are affected. Panels E & F: Visual change is delayed by 50 ms from target onset. The average SRT in both the short and long latency conditions are increased. Panels G & H: Visual change is delayed by 150 ms from target onset. All saccades in the short SRT condition have occurred so only the long SRT condition is affected.
When a second target or other detail is presented simultaneously with a saccadic target, it is called a distractor (e.g., Weber & Fischer, 1994). This can be considered to be a visual change that occurs simultaneously with the onset of the saccadic target, as is shown in panels C and D of Figure 2. The period of saccadic inhibition caused by the visual change is shown as a gray bar, that is from 75 to 125 ms after the visual change in this example. In panel C, the saccadic inhibition caused by the distractor inhibits only the lowest latency saccades that would have occurred. These saccades are substantially delayed, which increases the average SRT. In panel D, average SRT is not affected as no saccades with latencies short enough to be inhibited were present in the original SRT distribution (Figure 2, panel B).

When the visual change occurs after the target onset, saccades of longer latency begin to be inhibited. In panels E and H of Figure 2, the period of inhibition occurs near the peak of the original SRT distributions, producing a gap and causing the SRT distribution to become bimodal. As a result, a large proportion of the saccades are delayed by the saccadic inhibition and average SRT is substantially increased. If the delay from the target onset to the visual change is long enough, the inhibition will occur after all saccades have already occurred and SRT will be unaffected, as is the case in panel G.

Figure 2 suggests that the effect of saccadic inhibition on the average SRT should change with the timing of the visual change relative to the onset of the saccade target. This pattern of results was observed in a study by Ross and Ross (1980), that tested the effects of the timing of a warning signal on saccadic latencies. In this study, participants made saccades to a target 15° to the left or right of the fixation target. An ‘O’ appeared
or disappeared from the center of the fixation target as a warning signal, creating a visual change at the participant's fovea. This occurred before, simultaneous with, or after the saccadic target onset. The average SRT without the warning signal was 290 ms, which is a rather long saccadic latency and probably had a distribution similar to panel B of Figure 2. SRTs were found to be significantly increased when the onset of the warning signal was delayed by 50, 100, or 150 ms from the onset of the saccade target. Although no histograms of SRTs were reported, the timing would be similar to that in panels F and H in Figure 2. Simultaneous onset of the warning signal and the target did not cause a significant change in SRT, consistent with the situation in panel D in the figure. Ross and Ross interpreted their findings as an interfering effect associated with the warning stimulus onset.

In further experiments, Ross and Ross (1981) replaced the change in the fixation point with a 1000 Hz, 70 dBA tone and found no increase in SRT at any target-to-tone delay. Reaction time was not changed by the visual warning when participants responded with a manual movement of a lever instead of a saccade, confirming the purely visual and oculomotor nature of the effect. Finally, the onset of a pair of peripheral targets located 5.5° either above and below or to the left and right of the fixation point was used as a warning signal instead of the change in the fixation point. Significantly increased SRTs were seen when the onset of the peripheral targets was delayed by 100 ms relative to the saccade target, suggesting that saccadic inhibition does not require the visual change to occur at the fovea.

A similar manipulation of delay by Walker, Kentridge, and Findlay (1995) presented a saccade target at 4.5° or 8.5° to the right of the fixation point, and a distractor target to
the left of the fixation point. The distractor appeared at several delays either before, simultaneous with, or after the target onset. Targets were also presented without distractors to determine the baseline SRT, which was 168 ms in this study. Average SRT was significantly increased for distractor onset delays of 0 (simultaneous), 20 and 40 ms, probably similar to panels C and E in Figure 2. SRT was not significantly increased for a delay of 100 ms, as the inhibition may have occurred after all saccades had already been made, as illustrated by panel G of Figure 2. This same delay of 100 ms caused a large increase in SRT in the study of Ross and Ross (1980), which may be due to the longer average SRT in this study, similar to panel H of Figure 2. Walker et al. (1995) also included SRT histograms for their no-distractor and simultaneous distractor onset conditions. Comparison of these histograms shows a substantial decrease in the number of saccades with latencies of 150 ms or less, as in panels A and C of Figure 2.

A more complete set of SRT histograms is available from a study by Braun and Breitmeyer (1990). Here two red LEDs separated by 4° were used as the saccade target and the fixation point. The fixation point was switched off 300 ms before the target onset, and was left off or turned on again before, simultaneous with, or after the target appeared. The published SRT histograms for the conditions where the fixation LED onset was delayed by 50 ms and 100 ms from the target LED onset ("OFF 350" and "OFF 400" panels in Figure 3 of Braun & Breitmeyer, 1990) have a clear gap, similar to panel E of Figure 2. The location of this gap was shifted by 50 ms between these histograms, showing that the gap is time-locked to the onset of the fixation LED. In both histograms the gap began at 80 ms and ended at 160 ms after the onset of the fixation LED. When the onset of the fixation and target LEDs were simultaneous ("OFF 300"
panel of Figure 3 of Braun & Breitmeyer, 1990), few saccades with latencies of less than 160 ms were seen. This 160 ms saccade cutoff corresponds to the end of the period of inhibition (80 to 160 ms after fixation LED onset) as estimated from the histograms for the 50 and 100 ms delay conditions, and reproducing the situation in panel C of Figure 2.

The SRT histograms of Braun and Breitmeyer appear to support the role of saccadic inhibition in reducing the probability of low latency saccades when a second target or other detail is presented simultaneously with the onset of the saccadic target. When this simultaneous presentation is used, the non-target stimulus is usually called a distractor (e.g., Weber & Fischer, 1994). In the first study of this type, Levy-Schoen (1969) presented two targets simultaneously on opposite sides of the fixation point, resulting in an increase in SRT of 40 ms compared to that observed for a single target. In this study, participants were allowed to make a saccade to either target, so it is not clear that saccadic inhibition was responsible for the increase in SRT. Saccadic inhibition can only be implicated as the cause of increased SRTs when the study does not allow ambiguity about the identity of the target, as this could also increase SRT.

Saccadic inhibition in a distractor paradigm should selectively reduce the probability of the lowest latency saccades. In a study by Weber and Fischer (1994), the size and location of the distractor stimulus was varied to study its effect on the proportion of the fastest ("express") saccades produced. This was reported as the proportion of express saccades, computed by fitting three gaussian distributions to the distribution of saccadic reaction times and reporting the proportion of saccades in the earliest gaussian component. Neither average SRT or histograms of SRT distributions were reported, but
the express saccade proportion measure should reflect the strength of saccadic inhibition on the lowest latency saccades.

The proportion of fast saccades were enhanced by using a gap paradigm, in which the fixation point is removed 200 ms before the target onset. Low-latency saccade production was further encouraged by always placing the saccade target 4° to the right of the fixation point, thereby removing spatial uncertainty. Distractors of 0.1°, 0.2° and 0.4° in size were presented 4° to the opposite (contralateral) side of the fixation point from the target. The proportion of fast saccades was almost unaffected for the 0.1° distractor, but decreased with size for the 0.2° and 0.4° distractors, suggesting that the strength of saccadic inhibition increases with the size of the distractor. The 0.4° distractor was presented at both 4° and 12° contralateral to the target, and more fast saccades were observed at the larger distance, suggesting that inhibition decreases with increasing distance of the distractor from the fovea. A similar effect was observed using a larger distractor consisting of a column of 23 right slashes ('/'), presented on the same side (ipsilateral) of the fixation point as the target, at distances of 8.5°, 4.5°, 2.5°, and 0.5° from the fixation point. The proportion of fast saccades decreased as the distractor approached the fixation point, again suggesting that inhibition is inversely proportional to distance from the fovea.

The relation between the location of the distractor and SRTs was examined in detail in experiments by Walker, Deubel, Schneider, and Findlay (1997). Targets were presented at distances ranging from 0.5° to 8° from the fixation point, and distractors were at the fixation point, or ipsilateral or contralateral to the target and from 0.5° to 8° from the fixation point. The average SRT was longest when the distractor was located at
the fixation point, and decreased for contralateral distractors as distance from the fixation point increased. Assuming saccadic inhibition was responsible for the increased average SRT, this again suggests that the strength of saccadic inhibition is inversely proportional to distance from the fovea.

Visually evoked saccadic inhibition may also be found in other studies where visual changes occur near the time when a saccade is expected. An example is the double-step saccade paradigm, where a target is initially displayed at the fixation position, jumps to a peripheral location and then after a short delay to a second location (Becker & Jurgens, 1979). In a study of double-step saccades using small target displacements, Findlay and Harris (1984) found that a period of “intense inhibition” occurred in some of their target step sequences. This paper included histograms of saccade latencies, some of which show a gap from 80 to 160 ms after the second target step. The timing of this gap suggests that visually evoked saccadic inhibition triggered by the second step in the target position was responsible.

All of the studies reviewed so far have measured the reaction time of saccades made in response to the onset or motion of targets. Saccadic inhibition as measured by Reingold and Stampe (in press) was observed during reading and visual search of images, where the participant controls where and when to make a saccade. In this same study, Reingold and Stampe linked saccadic inhibition to a change in the distribution of fixation durations observed in gaze contingent masking tasks. In these tasks, a display change, such as the onset of a mask, was made at a fixed delay after the start of each of the participant’s fixations. This was observed to sharply reduce the number of fixations with durations equal to 80-120 ms plus the delay from the start of the fixation to the display
change. This produced a sharp dip in histograms of fixation durations, that was first observed in reading (Blanchard, McConkie, Zola, & Wolverton, 1984; McConkie, Underwood, Zola, & Wolverton, 1985; McConkie, Reddix, & Zola, 1992) and later in visual search of pictures (van Diepen, de Graef, & d’Ydewalle, 1995; van Diepen & Wampers, in press; van Diepen, 1998a, 1998b). These dips in the fixation duration distribution were shown by Reingold and Stampe (in press) to occur at a fixed delay from the display change and to be unaffected by the timing of the display change within the fixation. Using the paradigm described at the beginning of this thesis, it was clearly established that these dips are caused by visually evoked saccadic inhibition.

In summary, a number of previous studies of saccades to visual targets appear to support a role for visually evoked saccadic inhibition in increasing saccadic reaction times. Saccadic inhibition was evoked in these studies by the onset of warning signals, fixation targets, and distractors. Gaps in the distribution of SRTs were clearly seen when the visual change was delayed by 50 to 100 ms after the target onset (Braun & Breitmeyer, 1990), matching the expected time course of saccadic inhibition as illustrated in Figure 2. Delayed visual changes also increase average SRT, roughly following the pattern predicted for saccadic inhibition (Ross & Ross, 1980, 1981; Walker et al., 1995). In addition, distractors that appear at the same time as the saccade target were found to reduce the proportion of low-latency saccades and to increase average SRT (Weber & Fischer, 1994; Braun & Breitmeyer, 1990; Walker et al., 1995; Walker et al., 1997). Finally, saccadic inhibition was shown to be present in other tasks such as the double-step saccade paradigm (Findlay & Harris, 1984), and in gaze-contingent masking in reading (Blanchard et al., 1984; McConkie et al., 1985; McConkie et al., 1992) and in
visual search of pictures (van Diepen et al., 1995; van Diepen & Wampers, in press; van Diepen, 1998a, 1998b).
Neurophysiology of the Saccadic System

To provide insight into the phenomenon of visually evoked saccadic inhibition, it is necessary to review relevant neurological findings on the primate saccadic and visual systems. The review will be guided by the fact that saccadic inhibition is a very fast process, and probably involves the fastest, most direct neural pathways in the visual and saccadic systems. In humans, the fastest saccades are produced to bright targets when the fixation target has been removed at least 200 ms previously (Kingstone & Klein, 1993) and where oculomotor preparation is at its highest level (Dorris et al, 1997), with an average latency of approximately 100 ms. These have also been called “express” saccades (e.g. Fischer & Ramsperger, 1984), and are thought to approach the limits imposed by the latencies of the visual and saccadic system (Fischer & Weber, 1993; Pare & Munoz, 1996). By comparison, the latency of the onset of saccadic inhibition evoked by a full-screen luminance change was only 70 ms (Reingold and Stampe, in press).

An important topic in this review will therefore be the neural delay in the saccadic system. If available, the latency of neural activity from visual input and the latency of saccades produced by electrical stimulation will be reported for each neural substrate. These are summarized in Table 1 and Table 2 respectively. Caution must be used when comparing the visual latencies summarized in Table 1, as these are known to decrease as the size and contrast of stimuli are increased. For example, the latency of cat omnipause neuron responses to a flashed stimulus was found to decrease with increasing luminance from 100 ms at 0.032 cd/m² to 33 ms at 320 cd/m² (Evinger, Kaneko, & Fuchs, 1982). In addition, almost all information on neural latencies in primates comes from studies on
monkeys, which show significantly lower saccadic latencies than humans. Therefore the neural latencies in this review must be treated as lower limits when applied to the human nervous system. An example of these interspecies differences is the latency of the fastest (express) saccades for humans and rhesus monkeys under identical experimental conditions, which were found to be 100 ms for humans and 70 ms for monkeys (Weber, Latanov, & Fischer, 1993). Part of this difference may be due to longer delays in the human visual system, as an early positive peak in the visual evoked potential (VEP) was found to be 80 ms for humans and 65 ms for monkeys (Moller, Burgess, & Sekhar, 1987). The differences may be due to the larger brain and skull size of humans, requiring longer neural pathways and therefore longer delays in the optic and cranial nerves.

Figure 3 shows the neural structures and connections of the saccadic system that will be reviewed. These are limited to relatively direct and low-latency visual input pathways, the most direct pathways that can produce saccades, and control pathways that can act to suppress saccades. The flow of information in the figure proceeds clockwise through visual input that receives and processes visual information, saccade control structures that integrate visual and higher level input and determine where and when to make saccades, and the brainstem saccade generator that translates saccade commands into motor responses. Visual input structures reviewed include the retina, dorsal lateral geniculate nucleus (dLGN), striate and extrastriate cortex, and the pulvinar nucleus of the thalamus.
Figure 3. Selected neural structures and connections that are known to be involved in saccade production, selected on the basis of latency and function. Bold lines mark low-latency and dense projections; other lines mark slow or sparse connections. Data flow in saccade generation proceeds clockwise, proceeding through visual input, saccade control, and the saccade generator. Structures involved with feedback and visual attention are not shown, such as the internal medullary lamina of the thalamus. Abbreviations: dLGN, dorsal lateral geniculate nucleus; LIP, lateral intraparietal area; LLBN, long-lead burst neurons; MLBN, medium-lead burst neurons; OPN, omnipause neurons; OMN, oculomotor neurons; SNr, substantia nigra pars reticulata; MT, medial temporal area.
Table 1. Summary of visual latencies in the visual and saccadic system. Latencies are means (single numbers) or ranges as reported by authors.

<table>
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<th>Area</th>
<th>Cell Type</th>
<th>Visual latency (ms)</th>
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<td>Schmolensky et al. (1998)</td>
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<td>44-56</td>
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<tr>
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<td>Schmolensky et al. (1998)</td>
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<td>Parvocellular (4Cβ)</td>
<td>60-72</td>
<td>Schmolensky et al. (1998)</td>
</tr>
<tr>
<td>SC</td>
<td>Pandirectional cells</td>
<td>40-50 (small spot)</td>
<td>Goldberg &amp; Wurtz (1972)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>35-47 (bright flash)</td>
<td>Rizzolatti et al. (1980)</td>
</tr>
<tr>
<td></td>
<td>Buildup cells</td>
<td>~70 (2.0 cd/m² LED)</td>
<td>Dorris et al. (1997)</td>
</tr>
<tr>
<td>FEF</td>
<td>Visuomovement cells</td>
<td>67</td>
<td>Schall (1991)</td>
</tr>
<tr>
<td></td>
<td>Visual cells</td>
<td>77</td>
<td>Schall (1991)</td>
</tr>
<tr>
<td>SEF</td>
<td>Visuomovement cells</td>
<td>92</td>
<td>Schall (1991)</td>
</tr>
<tr>
<td>LIP</td>
<td>Visuomovement cells</td>
<td>60-140</td>
<td>Yin &amp; Mountcastle (1977)</td>
</tr>
<tr>
<td>PPRF</td>
<td>Omnipause neurons</td>
<td>~70 (2.0 cd/m² LED)</td>
<td>Everling et al. (1998)</td>
</tr>
</tbody>
</table>

We will discuss the visual input and saccade generator stages first, as these define the input and output connections and latencies of the system. The superficial visual layer and the intermediate layer of the superior colliculus, which play a key role in low-latency saccades and possibly visually evoked saccadic inhibition, are reviewed in detail. Finally, cortical areas including the frontal eye fields, supplementary eye fields, and lateral intraparietal area (LIP) will be reviewed.
Table 2. Latencies of saccades or other effects evoked by electrical stimulation within the saccadic system. Latencies are means (single numbers) or ranges as reported by authors.

<table>
<thead>
<tr>
<th>Stimulated Area</th>
<th>Effect</th>
<th>Latency (ms)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>SC (fixation zone)</td>
<td>Saccade deceleration</td>
<td>10-15</td>
<td>Munoz &amp; Wurtz (1993b)</td>
</tr>
<tr>
<td></td>
<td>Saccade delayed</td>
<td>&gt;20 before saccade onset</td>
<td>Munoz et al. (1996)</td>
</tr>
<tr>
<td>Caudal SC</td>
<td>Saccade evoked</td>
<td>20-30</td>
<td>Robinson (1972)</td>
</tr>
<tr>
<td></td>
<td>Change in saccade trajectory</td>
<td>8-10</td>
<td>Munoz et al. (1996)</td>
</tr>
<tr>
<td>FEF</td>
<td>Saccade evoked</td>
<td>18 (high intensity)</td>
<td>Seagraves (1992)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>40 (low intensity)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Omnipse inhibited</td>
<td>5 (high intensity)</td>
<td>Seagraves (1992)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30 (low intensity)</td>
<td></td>
</tr>
<tr>
<td>SEF</td>
<td>Saccade evoked (increases for small saccades)</td>
<td>30-500</td>
<td>Schlag &amp; Schlag-Rey (1987)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Tehovnik &amp; Lee (1993)</td>
</tr>
<tr>
<td>LIP</td>
<td>saccade evoked</td>
<td>40-120</td>
<td>Kurylo (1991)</td>
</tr>
<tr>
<td></td>
<td>blocks visual saccade</td>
<td>&lt;40</td>
<td>Computed from Kurylo (1991)</td>
</tr>
<tr>
<td>PPRF (Omnipause)</td>
<td>pause to saccade onset</td>
<td>10-22</td>
<td>Raybourn &amp; Keller (1977)</td>
</tr>
<tr>
<td></td>
<td>stimulation to saccade deceleration</td>
<td>10</td>
<td>Keller &amp; Edelman (1994)</td>
</tr>
</tbody>
</table>
**Visual Inputs and Processing**

Visual input specifies the destination of a saccade, and in most experimental tasks also supplies the signal to make a saccade. The time course of events in the visual system, such as the transduction of light into neural signals and the transmission and processing of these signals, determines the latency of neural events in the superior colliculus and other neural structures, and thus the latencies of saccades and of saccadic inhibition. A side effect of the processing of visual input in the cerebral cortex is the generation of visual evoked potentials (VEP). Measurements of the amplitude and latency of the VEP reflect the magnitude of cortical neural signals and the time course of neural processing respectively. These measures of VEPs will be compared with similar measures of visually-evoked saccadic inhibition in the experimental section of this thesis.

**Retinal Ganglion Cells**

Transduction of light to neural signals takes place in the retina, with retinal ganglion cells processing the rod and cone photoreceptor outputs and transmitting visual information through the optic nerve. Three classes of retinal ganglion cells have been identified, each creating a separate visual channel. In primates, the M cells, analogous to Y cells in the cat, have large cell bodies and thick axons with high conduction velocities that minimize delays. M cells respond equally well to all wavelengths of light and are insensitive to color. They respond to changes in visual input with a strong but transient burst of activity, and are sensitive to contrasts as low as 1% (Hicks, Lee & Vidyasagar, 1983). These cells also have large receptive fields and are most sensitive to low spatial frequencies. Primate P cells, similar to cat X cells, have smaller bodies and receptive
fields and thinner axons with conduction velocities half that of M cells. A large proportion of P cells have color-opponent receptive fields, and respond well to color differences such as a border between red and green areas. Their response to changes in visual input is slower than that of M cells, but is more sustained. P cells also have lower contrast sensitivity than M cells, but are more sensitive to high spatial frequencies. W cells have heterogeneous receptive fields, slow conduction velocities and are not color sensitive. The proportions of these types of ganglion cells in the retina is about 80% P cells, 15% M cells, and 5% W cells (Schiller & Malpeli, 1977).

**Lateral Geniculate Nucleus and Cortex**

Retinal ganglion cells project largely to the dorsal lateral geniculate nucleus (dLGN) of the thalamus. Retinal inputs to the dLGN are segregated, with M cells projecting to the magnocellular layers, and P cells projecting to the parvocellular layers. Visual latencies measured in the magnocellular layers are about 10-20 ms faster than those in the parvocellular layers (Schmolensky et al., 1998). Both magnocellular and parvocellular channels show 20-30 ms increases in latency at higher spatial frequencies (Sestokas & Lehmkuhle, 1986). The dLGN projects almost exclusively to the striate visual cortex, also called V1 or area 17. Most of the area of the striate cortex is involved with processing the foveal and parafoveal regions of the visual field, reflecting the higher acuity and denser projections from these regions of the retina. The input to striate cortex from the dLGN is also segregated by type, with magnocellular layers projecting to layer 4Cα of striate cortex, and the parvocellular layers to 4Cβ.
The segregation of magnocellular and parvocellular input appears to hold throughout much of the visual system. The parvocellular input proceeds through several regions of extrastriate cortex to the inferior temporal cortex, forming the ventral visual pathway that appears to specialize in object recognition. Processing in this pathway proceeds serially, resulting in relatively long and variable visual latencies. An example is area V4 in inferior temporal cortex, where latencies of 72-159 ms were observed (Schmolensky et al., 1998). The magnocellular input proceeds through extrastriate cortex into the posterior parietal lobe, forming the dorsal visual pathway involved in location and motion processing. Latencies in this pathway are relatively fast. For example, latencies of 62-82 ms (Schmolensky et al., 1998) were observed in area MT (also called V5), that appears to specialize in motion perception (Newsome, Wurtz, Dursteler, & Mikami, 1985).

Subcortical Visual Pathways

The retina also has direct projections to subcortical visual pathways. Retinal input to the superficial layer of the superior colliculus supply very short latency input to the SC, that will be discussed later. The pulvinar and lateral posterior nucleus of the thalamus receive visual input largely through the superior colliculus and perhaps directly from the retina and parvocellular dLGN (for a review see Robinson & McClurkin, 1989). The pulvinar is reciprocally connected to extrastriate, frontal, and parietal areas of cortex, and may provide fast cortical input for visual alerting and attention. Because little is known of the function of the pulvinar, it will not be discussed further here.
Brainstem Saccade Generator

The saccade generator transforms a saccade command into a precisely defined burst of activity from the oculomotor neurons, producing rapid contractions of the ocular muscles (Robinson, 1970). The direction of the saccade is largely determined by the relative activation of each of the six ocular muscles (King, Lisberger, & Fuchs, 1986). The amplitude of the saccade is highly correlated with the total number of spikes in the saccade burst (Hepp & Henn, 1985), which is determined by the duration and firing rate during the burst. Therefore in order to produce a saccade of the correct amplitude and direction, the saccade generator must control both the duration of the saccade burst and its magnitude for each eye muscle.

A simplified version of the saccade generator is shown in Figure 3, consisting of the long-lead burst neurons (LLBNs), medium-lead burst neurons (MLBNs), and omnipause neurons (OPNs). These are located in the brainstem, in or adjacent to the paramedian pontine reticular formation (PPRF). Other parts of the oculomotor system associated with vestibular reflexes and pursuit and the cerebellar feedback that ensures accurate saccades are not discussed here (for a review, see Wurtz & Goldberg, 1989).

The LLBNs receive excitatory input from several saccade control areas in the brain. The major source of this input is from the superior colliculus, but sparse projections are also present from the frontal eye fields (FEF) and supplementary eye fields (SEF). LLBNs fire for a range of saccade directions and amplitudes that terminate in a small area of the visual field, forming a “movement field” for each neuron (Hepp & Henn, 1985). These “movement fields” are also seen in neurons in the intermediate layers of
the superior colliculus and in the frontal eye fields. This may indicate that LLBNs represent the desired saccade target in the same way as these structures do. LLBNs begin firing 15-30 ms in advance of a saccade, and continue to fire until the end of the saccade (Hepp & Henn, 1985).

The LLBNs provide excitatory input to the MLBNs, that in turn drive the oculomotor neurons (OMNs). The MLBNs consist of a group of excitatory and inhibitory burst neurons for each eye muscle. The duration and characteristics of the burst produced by these neurons closely matches the time course of the saccade. The timing of the burst is imposed by the omnipause neurons (OPNs), which strongly inhibit all MLBNs. The OPNs fire at a high tonic rate during fixation and pursuit, and pause to define the MLBN burst that drives the saccade. Omnipsause neurons receive both monosynaptic excitatory and polysynaptic inhibitory input from the superior colliculus, and inhibitory inputs from both the SEF and FEF. It is thought that the same neural pathways that excite the LLBNs and specify the goal of a saccade also inhibit the OPNs to start the saccade burst (Raybourn & Keller, 1977; Seagraves, 1992). In monkeys, the OPNs begin to pause 10-22 ms before the onset of the saccade (Raybourn & Keller, 1977). It is not yet known where the signal to end the burst and stop the saccade comes from, but the cerebellum (Everling, Pare, Dorris, & Munoz, 1998) and superior colliculus (Optican, 1995) have been suggested.

Could saccadic inhibition be mediated via the omnipause neurons? Saccades can be prevented or even interrupted by electrical stimulation of the omnipause neurons in monkeys (Keller & Edelman, 1994) and cats (Pare & Guitton, 1994). In cats, the output of the OPNs is modulated by visual, auditory and tactile sensory input, and very bright
flashes of light can stop saccades in progress within 60 ms (Evinger et al., 1982). The visual responses of cat OPNs appear to arise from the superior colliculus, as the bilateral ablation of the SC removed this visual modulation (King, Precht & Dieringer, 1980). Omnipause neurons in monkeys show much weaker visual modulation: in the cat, a 3.2 cd/m² flash produced a 100% increase in the firing rate of the OPNs (Evinger et al., 1982), whereas in the monkey a 2 cd/m² LED at 10° eccentricity caused a 7% increase in the firing rate (Everling et al., 1998). The visual response of monkey OPNs is probably too weak to permit a visual stimulus, however strong, to interrupt saccades or to cause the robust saccadic inhibition observed by Reingold and Stampe (in press).

**Saccade Control Structures**

A network of cortical areas and subcortical structures acts to control saccades and visual attention. This review will be limited to those areas that produce saccades when electrically stimulated. The latency of such electrically-evoked saccades are summarized for each area in Table 2. In the prefrontal cortex, the frontal eye fields (FEF) and the supplementary eye fields (SEF) act to generate voluntary saccades. In parietal cortex, the lateral intraparietal area (LIP) can generate saccades as well. All these cortical regions are connected by direct projections, and indirectly via the pulvinar nucleus and internal medullary lamina of the thalamus, in a network that may coordinate visual attention as well as saccades. The FEF and SEF have direct projections to the saccade generator in the brainstem. Each of the cortical areas also has a direct projection to the superior colliculus, and an indirect projection through the basal ganglia (caudate nucleus and substantia nigra pars reticulata). The superior colliculus is the central element in
subcortical saccade production. It receives projections from most other saccade-producing areas, has fast visual input, and its neurological mechanisms are beginning to be understood in detail.

This review will discuss the superior colliculus first and in some detail as more is known about how it functions than any other saccade control structure. The frontal eye fields, supplementary eye fields, and LIP will next be discussed. Each of these areas will be discussed in terms of structure, visual inputs, saccade-related outputs, and their possible functions. Any known inhibitory effects on saccades will be discussed, as well as their suitability for producing the observed visually-evoked saccadic inhibition.

**Superior Colliculus**

The superior colliculus (SC) is uniquely situated to provide fast oculomotor responses to visual inputs. The SC receives direct retinal input, and collicular output directly activates the saccade generator in the brainstem. Lesions of the superior colliculus abolish the fastest (express) saccades in monkeys (Schiller, Sandell, & Maunsell, 1987), the latency of which is thought to approach the limits imposed by delays in the visual and saccadic system (Fischer & Weber, 1993; Pare & Munoz, 1996). The SC is a central structure in the saccade control network, receiving converging projections from many cortical areas involved with visual attention and saccades, and collicular output is sent to the cortex through the internal medullary lamina (IML) of the thalamus, that may serve to coordinate visual attention as well as saccades. The SC also has connections with many of the subcortical structures involved with saccades and pursuit, including the cerebellum.
**Structure of the Superior Colliculus**

The superior colliculus is divided on its midline into left and right colliculi, each of that receives visual input from and commands saccades to the contralateral (opposite) visual hemifield. Each SC consists of several alternating layers of gray and white tissue, which have been grouped by function into the superficial, intermediate, and deep layers. The superficial SC contains largely visually responsive cells. The upper part of the superficial SC receives direct retinal input, whereas the input to the lower part is largely from striate and extrastriate visual cortex (Schiller, Malpeli, & Schein, 1979). The lower part of the superficial SC also sends projections to the pulvinar nucleus of the thalamus, possibly carrying low-latency information on changes in visual input (Robinson & McClurkin, 1989).

The intermediate SC contains buildup and burst neurons (Munoz & Wurtz, 1995a), that fire in advance of and during saccades and project to the saccade generator in the brainstem. This layer receives projections from several cortical regions that are involved in saccade planning and visual attention: the frontal eye fields (Seagraves & Goldberg, 1987), supplementary eye fields (Shook, Schlag-Rey, & Schlag, 1990), and lateral intraparietal area (Lynch, Graybiel, & Lobeck, 1985). Input is also received from subcortical structures including the substantia nigra and cerebellum. The deep SC receives extensive motor, premotor, and multimodal sensory projections, and projects to the thalamus and the brainstem (for a review, see Sparks & Hartwich-Young, 1989). The deep SC may be involved in multimodal orienting behaviors and in combined eye-head gaze shifts.
Both the visual inputs to the superficial SC and the motor responses of the intermediate SC are organized in a retinotopic map (Robinson, 1972). Figure 4 shows an idealized mapping of the visual field onto the surface of the SC, using a mathematical model of the relation between the retinotopic and collicular coordinates (Ottes, van Gisbergen & Eggermont, 1986; Optican, 1995). The foveal and parafoveal region of the visual field is magnified in this map, with much of the rostral collicular surface representing the central 10° of the visual field. This is similar to the foveal magnification seen in striate visual cortex.

Figure 4. Topographical mapping between the surface of the superior colliculus and the contralateral visual hemifield. Coordinates in millimeters on the collicular surface are given. Note the magnification of the foveal region of the collicular surface and the compression of the peripheral visual field. (Derived from Optican, 1995).
The motor map in intermediate SC and the visual map in superficial SC appear to be aligned. It has been found (e.g., Kadonya, Wolin, & Massopust, 1971; Goldberg & Wurtz, 1972) that cells in the superficial SC respond to locations in the visual field that are close to those to which the cells immediately below them in the intermediate layer command saccades. The foveal area of the visual field is represented at the rostrolateral pole of the SC, and stimulation of the intermediate layer of the SC in this area evokes small saccades (Robinson, 1972) or prevent the production of saccades (Munoz & Wurtz, 1993). The peripheral visual field is represented in the caudal SC, and larger saccades are evoked by stimulation of this region. Excitatory synaptic connections have been demonstrated between the superficial and intermediate layers of the SC (Mooney et al., 1988; Moschovakis, Karabelas, & Highsteen, 1988; Lee, Helms, Augustine, & Hall, 1997), suggesting the vertical columnar organization of the SC illustrated in Figure 5.

**Superficial Layers of the Superior Colliculus**

The superficial layer of the superior colliculus receives direct inputs from M and W cells in the retina (Schiller & Malpeli, 1977), as well as projections from the striate cortex and sparse projections from extrastriate cortex (Fries, 1984). The retinal input comes from both peripheral and foveal areas of the visual field (Cowey & Perry, 1980), and projects to the uppermost 0.3mm of the superficial SC (Schiller, Malpeli, & Schein, 1979).

Several findings suggest that the cortical projections to the superficial SC from the visual cortex is not the major source of visual input. Removal of the cortical input by
cooling or ablating striate cortex does not appear to change the properties of most visually responsive neurons in the upper part of the superficial SC (Schiller, Stryker, Cynader, & Berman, 1974). In addition, the properties of the cells in striate cortex that project to the SC show orientation and motion direction specificity, which is unlike the nonselective response of the pandirectional cells that form the majority (87%, Goldberg & Wurtz, 1972) of visually responsive cells in the SC. A small number of direction-sensitive cells are present in the superficial SC (<10%, Goldberg & Wurtz, 1972), which may reflect the striate cortical input. An alternative role for the cortical input may be to modulate the responsiveness of the SC to visual input (Finlay, Schiller, & Volman, 1976; Lee et al., 1997). An example of this modulation is the enhanced visual responsiveness shown by neurons in the lower part of the superficial SC when a saccade is being prepared to a target that falls within the cell’s receptive field (Wurtz & Mohler, 1976). It has been suggested that this enhancement is controlled either by descending cortical input (Lee et al., 1997) or by ascending input from eye-movement cells in the intermediate layers of the SC (Wurtz & Mohler, 1976).

Pandirectional cells in the superficial SC respond with a transient burst of activity to flashing spots or to stimuli moving in any direction (Goldberg & Wurtz, 1972). The visual latency of these neurons is as low as 35 ms for bright flashed stimuli (Rizzolatti, Buchtel, Camarda, & Scandolara, 1980) and stimulus contrast has only a small effect on their response (Marrocco & Li, 1977). Pandirectional cells respond best to stimuli of optimal size, which varies from <1° at the fovea to >15° at 40° eccentricity (Marrocco & Li, 1977). Stimuli of smaller than the optimal size produce only slightly attenuated responses, with a 16-fold decrease in stimulus area reducing the number of spikes in the
burst by only a factor of 2 (figure 3 of Moors & Vendrik, 1979). Most pandirectional cells also show reduced responses to stimuli larger than their optimal size, and no response to stimuli larger than their receptive fields. However, about 20% of pandirectional cells continue to respond well to large flashed stimuli (Goldberg & Wurtz, 1972). Visual latency decreases for larger stimuli, reaching its minimum at the cell’s optimal stimulus size (Moors & Vendrik, 1979).

Numerous findings suggest that the SC may be insensitive to color. Retinal inputs to the SC are from broadband color-insensitive M and W ganglion cells. The cortical inputs to the superficial SC also appear to be derived from the color-insensitive magnocellular input to visual cortex, as inactivating the magnocellular layers of the dLGN caused loss of all visual activity in anesthetized monkeys, except for the retinal inputs (Schiller, Malpeli, & Schein, 1979). No cells in the superficial SC of macaque monkeys have been found that respond strongly to moving isoluminant color edges (Marrocco and Li, 1977). Some SC neurons do show small differences in the color they best respond to, which might explain weak responses to isoluminant stimuli (Marrocco and Li, 1977; Kadonya et al., 1971).

**Intermediate Layers of the Superior Colliculus**

The intermediate layer of the SC contains several types of premotor cells, that fire before and during saccades. These cells have been classified according to their pattern of activity, and by their position and depth in the intermediate layer (Munoz & Wurtz, 1995a, 1995b). The upper part of the intermediate SC contains burst neurons (SCBNs), that fire strongly just before and during saccades but are otherwise inactive. The deep part of the intermediate SC contains buildup neurons (SCBUNs), that show increasing
activity before saccades and may also fire strongly during saccades. The most rostral part of the deep layer of the intermediate SC contains fixation cells (SCFNs) (Munoz & Wurtz, 1993a, 1993b) that are active during fixations and pursuit but pause just before and during saccades. These cells are limited to the fixation zone, that has been estimated to cover the rostral 0.72mm of the SC (Munoz & Wurtz, 1995b), which is the region associated with saccades of less than 2° in amplitude (see the collicular map in Figure 4). Some SCFNs also appear to encode small errors in eye position during smooth pursuit (Krauzlis, Basso, & Wurtz, 1997). The location and connections of these three classes of cells are illustrated in Figure 5.

All classes of cells in the intermediate SC may show transient bursts of activity in response to visual events. This visual input may be generated by the visual cells in the superficial SC, and carried through vertical connections to the intermediate SC. Neurons in the intermediate SC can have both a visual receptive field and a movement field. The movement field is the set of saccade directions and amplitudes that a neuron fires to command, which typically moves the eye to a small region of the visual field. The visual receptive field and movement field typically overlap, and the combination of these has been called the neuron’s response field (Dorris, Pare & Munoz, 1997).

Visual activity in both the superficial and intermediate SC may be modulated by descending projections from striate and extrastriate cortex (Wurtz & Mohler, 1976; Lee et al.,1997). The intermediate SC also receives projections from several cortical regions that are involved in saccade planning and visual attention: the frontal eye fields (Seagraves & Goldberg, 1987), supplementary eye fields (Shook et al., 1990), and lateral intraparietal area (Lynch et al., 1985). These projections may serve to suppress unwanted
saccades and to command saccades by imposing the desired pattern of activation onto the intermediate layers of the SC. The frontal eye field connections are known to achieve this by a pattern of inhibition and excitation of the intermediate SC (Schlag-Rey, Schlag, & Dassonville, 1992). Movement neurons in the frontal eye fields have excitatory connections to neurons in the intermediate SC that produce saccades similar in amplitude and direction to those produced by the FEF neurons that project to them. Other SC neurons that fire during saccades that are incongruent to those that the FEF neuron fires for will be inhibited by the activity of that FEF neuron (Schlag-Rey et al. 1992; Stanton, Goldberg, & Bruce, 1988).

Another control pathway to the SC is via the basal ganglia. Both the FEF and SEF project to the caudate nucleus (CN), that sends inhibitory projections to the substantia nigra pars reticulata (SNr), which in turn sends inhibitory projections to the intermediate SC. Cells in the SNr appear to change activity in a sequential fashion as required to carry out saccadic tasks, firing tonically and pausing to allow SC activity and saccades. The caudate nucleus appears to be active only during performance of well-learned tasks, with neurons firing to inhibit the SNr and disinhibit the SC (for a review, see Hikosaka & Wurtz, 1989).

The intermediate SC commands saccades through its connections to the brainstem saccade generator. The connections of each type of intermediate SC neuron are shown in Figure 5, which follows those proposed by Everling et al. (1998). In this scheme, both SCFNs and SCBUNs have excitatory connections to the OPNs. Rapid inhibition of the OPNs from the SC has also been demonstrated (Chimoto, Iwamoto, Shimazu, & Yoshida, 1996; Raybourn & Keller, 1977), presumably by the SCBNs (Everling et al., 1998).
Excitatory connections from the intermediate layers of the SC have been demonstrated to the LLBNs in the saccade generator (Raybourn & Keller, 1977; Istvan, Dorris, & Munoz, 1994), but it is not yet clear if the excitation is from SCBNs or both SCBNs and SCBUNs (Munoz & Wurtz, 1995a).

Figure 5. A model of inputs, internal connectivity, layers and outputs of the superior colliculus. Visual input from the retina is processed and gated in the superficial layer by visual neurons (V), then excites burst (B) and buildup (BU) or fixation (F) cells in the intermediate layers. Descending cortical projections inhibit or excite selected columns. Output from fixation, buildup, and burst cells then drives the saccadic burst generator in the brainstem. Inhibitory connections are mediated by interneurons.
Figure 6. Schematic of excitatory and inhibitory interactions in the superior colliculus. Fixation zones of the left and right colliculi excite each other to act as a unit, whereas inhibition between and within caudal areas of the colliculi acts as a winner-take-all competition network to select a saccade target. Local excitatory zones act to produce active regions to encode saccades. Connections in the figure after Munoz & Istvan, 1998. F: Fixation neurons; S: saccade neurons (SCBN or SCBUN)

A network of excitatory and inhibitory interneurons within the superior colliculus shapes neural activity before and during saccades. The current understanding of the collicular circuitry is shown in Figures 5 and 6, which are derived from Munoz and Istvan (1998). These authors found that electrical stimulation of SCFNs in the fixation zone at the rostral pole of the SC strongly inhibited SCBNs and also inhibited SCBUNs to a lesser degree. In turn, stimulation of both SCBNs and SCBUNs inhibited the SCFNs. This suggests a pattern of mutual inhibitory connections which may prevent the SCFNs and SCBNs from being active at the same time, thus forcing the SC to switch quickly
between saccade (SCBNs active) and fixation (SCFNs active) states. SCFNs in the left and right colliculi are connected by strong excitatory connections, ensuring that both colliculi are in the same state. The lesser inhibition of SCBUNs by SCFNs allows moderate preparatory activity in these neurons before saccades (Munoz & Istvan, 1998).

Inhibition also occurs between SCBNs and SCBUNs in different regions of the SC, as shown in Figure 6. The majority of neurons inhibit each other, and inhibitory connections are also present between neurons in the left and right colliculi. However, there is strong evidence that each neuron also excites nearby neurons within 1 to 2mm (McIlwain, 1982; Munoz & Istvan, 1998; Meredith & Ramoa, 1998). Models using this pattern of excitation of nearby neurons and inhibition of distant neurons produced winner-take-all competition to select between distant saccade targets, while shaping the local region of neural activity seen during saccadic bursts (van Opstal & van Gisbergen, 1989a, 1989b). This connectivity also contributes to the merging of activity that leads to the global effect, in which saccades to two closely spaced visual targets tend to land midway between the targets (Findlay 1982; Ottes, van Gisbergen, & Eggermont, 1984; Edelman & Keller, 1998).

The activities of fixation, buildup, and burst cells before and during saccades to visual targets had been studied in detail (Munoz & Wurtz, 1993a, 1995a, 1995b; Dorris et al., 1997; Dorris & Munoz, 1998; Anderson, Keller, Gandhi, & Das, 1998). The pattern of activity during saccades and fixations is illustrated in Figure 7. During a fixation, SCFNs are active and SCBNs are silent. In preparation for a saccade, activity of SCBUNs in the area of the colliculus corresponding to the saccadic target increases and may be accompanied by a decrease in SCFN activity. About 20 ms prior to the saccade,
SCBUNs and SCBNs both burst to command the saccade and activity of SCFNs is almost completely suppressed.

Throughout the course of the saccade, the SCBN activity remains limited to an area of the SC about 1.4mm in diameter, the location of which encodes the saccade amplitude and direction (Munoz & Wurtz, 1995b). SCBUNs also burst in this area during the saccade, and lower levels of SCBUN activity spread rostrally towards the fixation zone as the saccade progresses (Munoz & Wurtz, 1995b; but see Anderson et al., 1998). Shortly before the end of the saccade, the fixation zone is reactivated and the SCBNs and SCBUNs stop bursting. One possible role for the spread of SCBUN activity during the saccade is as a neural integrator of the saccade pulse (Optican, 1995). According to this model, when SCBUN activity reaches the fixation zone, the SCFNs are reactivated and shut off the brainstem saccade generator via the OPNs (Munoz & Wurtz, 1995b; but see Aizawa & Wurtz, 1998).

Activity of SCBUNs before a saccade has been shown to be correlated with motor preparation, which can be manipulated by changing the predictability of the location of the saccade target (Dorris & Munoz, 1998) or by changing the number of potential targets (Basso & Wurtz, 1997). Presaccadic activity in monitored SCBUNs was highest when the location of the saccade target was predictable and was within the response field of the neuron. Conversely, presaccadic activity was lowest when the target location was uncertain or was unlikely to be in the response field of the neuron. The presaccadic SCBUN activity was also highly correlated with the saccadic reaction time on a trial-by-trial basis, with high activity preceding the shortest reaction times (Dorris & Munoz, 1998).
Figure 7. Behavior of intermediate superior colliculus neurons before, during, and after saccade. Height of curves show activity levels of neurons at various positions in the SC. Buildup neurons are active before the saccade to compute the goal, burst during the saccade and show spreading activity that reaches the fixation zone at the end of the saccade. Fixation neurons are active between saccades, but pause during the saccade to let SCBNs fire. (After Munoz & Wurtz, 1995b).

The probability of very low latency (express) saccades also increases when SCBUN activity is high (Dorris et al., 1997). These saccades are thought to be triggered directly by visual input to the SCBUNs caused by the onset of the saccade target. When
presaccadic activity in the SCBUNs is high enough, this additional visual input may cause the total SCBUN activity to reach the threshold required to produce a saccade immediately (Edelman & Keller, 1996; Dorris et al., 1997).

Visually-evoked saccadic inhibition may also be a result of activity in the intermediate superior colliculus caused by changes in visual input. One mechanism is for visually evoked activity in SCBUNs to act through inhibitory connections within the intermediate SC to reduce presaccadic activity in other SCBUNs (Munoz & Istvan, 1998). This lowered activity of SCBUNs has been shown to delay the onset of saccades (Dorris & Munoz, 1998). Alternatively, the visual activity might stimulate SCFNs, which would also inhibit presaccadic activity in SCBUNs throughout the SC. The small size of the classical fixation zone (Munoz & Wurtz, 1993a, 1993b) would limit the source of this inhibition to visual changes near the fovea, although it has been proposed that SCFNs may be present in an extended fixation zone as far as 10° from the fovea (Gandhi & Keller, 1995, 1997; Findlay & Walker, in press). This extended fixation zone is based on the interruption of saccades by electrical stimulation of the SC outside the area of the classical fixation zone (Gandhi & Keller 1995), but this finding may also be explained by mutual inhibition between SCBUNs or by excitatory connections of SCBUNs to the OPNs.

The latency of saccadic inhibition caused by both SCBUN and SCFN excitation may be estimated from the latencies of visual activity in the intermediate SC, and the interval in which the reduction of presaccadic activity can act to delay the saccade. Visual latencies in the superficial SC are as low as 35 ms for strong stimuli (Table 1), and the delay in transmission between the superficial and intermediate SC is probably less than
10 ms (Lee et al., 1997) for a predicted visual latency of 45 ms in the intermediate SC. Visual latencies of 60-70 ms were reported for SCBUNs by Munoz & Wurtz (1995a), but this longer latency may due to the dim (2.0 cd/m²) LEDs used as stimuli in their study. The latency at which SCFN activity can act to delay saccades can be estimated from a study by Munoz and Wurtz (1993b), in which electrical stimulation of SCFNs could delay saccades when delivered as little as 20 ms before the saccade onset. The estimated latency of saccadic inhibition is then visual latency in the intermediate SC (45 ms) plus the latency at which such activity can delay saccades (20 ms), for an estimated latency of 65 ms. This is in good agreement with the 70 ms latency of onset of saccadic inhibition reported by Reingold and Stampe (in press). This timing example used findings of SCFN stimulation, but the latency of inhibition of SCBUNs within the SC is similar when either SCFNs (1.8 ms in ipsilateral SC, 3.6 ms in contralateral SC) or SCBUNs (1.3 ms for ipsilateral SC, 3.0 ms in contralateral SC) are stimulated (Munoz & Istvan, 1998). This suggests that saccadic inhibition produced by either of the two mechanisms would result in similar latencies.

**Frontal Eye Fields**

The frontal eye fields (FEF) receive visual input from both the dorsal (position and motion) and ventral (identity) streams of processing in extrastriate visual cortex (Schall, Morel, King, & Bullier, 1995). The FEF may also receive fast visual input from the superficial superior colliculus by way of the pulvinar (Lo, Cork, & Mize, 1998). Both visual inputs and saccades elicited by electrical stimulation are retinotopically organized, with the lateral FEF receiving foveal visual input and producing saccades of small amplitude, and the medial FEF receiving peripheral visual input and producing longer
saccades (Bruce & Goldberg, 1985). The projections from the FEF to the intermediate layers of the superior colliculus are also organized by saccade size and direction, connecting areas of the SC and FEF that produce similar saccades (Komatsu & Suzuki, 1985; Stanton et al., 1988; Schlag-Rey et al., 1992).

The majority of FEF neurons produce bursts of activity in response to visual input, and about 50% of these show enhanced visual responses if a saccade is to be made to targets in their receptive fields (Bruce & Goldberg, 1985). These cells may also fire just before a saccade is made (visuomovement cells) or may respond only to visual input (visual cells). A small number of neurons (movement cells) do not respond to visual input at all. The latency of the visually responsive cells is substantially greater than that of cells in the superficial superior colliculus or in striate cortex (Bruce & Goldberg, 1985; Schall, 1991). Pure visual cells respond to target onset with an average latency of 77 ms, although some cells may respond within 50 ms (Figure 3C of Schall, 1991). Transient visuomovement cells produce a short burst of activity to the onset of a target with an average latency of 65 ms, whereas sustained visuomovement cells show an average response latency of 98 ms and continue their activity while the target remains in the cell’s receptive field. It should be noted that these latencies are from the start of the change in firing rate of the neurons, with peak activity occurring about 50 ms later (Schall, 1991).

Each movement or visuomovement cell fires before saccades with a range of amplitudes and directions, the mean of which defines the preferred saccadic target for the cell (Bruce & Goldberg, 1985). This implements a movement field for each cell, similar to those of neurons in the superior colliculus and the LLBNs of the saccade generator in the brainstem. High-current electrical stimulation of the FEF evokes saccades within 18
ms, acting through direct projections to excite the LLBN in the brainstem in 5-10 ms and inhibiting the OPN in less than 5 ms (Seagraves, 1992). More typical current strengths produce saccades within 40 ms, perhaps through the projections to the intermediate layers of the superior colliculus (Schlag-Rey et al. 1992). Unlike the SC, electrical stimulation of the FEF does not appear to cause excitation of the OPNs.

A small number of cells (7%, Bruce & Goldberg, 1985) have foveal receptive fields and are active at times when a task requires attentive fixation, or when such a period of fixation ends (Seagraves & Goldberg, 1987). These foveal-fixation cells are also active during smooth pursuit. A study by Seagraves (1992) of electrical stimulation in the FEF was unable to show that these cells stimulate the OPNs, unlike fixation cells in the superior colliculus. These foveal-fixation cells are scattered throughout the frontal eye field, and appear to project to all areas of the superior colliculus (Seagraves & Goldberg, 1987) and not specifically to the fixation cells in its rostral pole.

The frontal eye fields may include a fixation zone similar to that of the superior colliculus. An area just lateral to the classical FEF contains largely fixation cells and, when stimulated, acts to suppress saccades (Burman & Bruce, 1997). The frontal eye fields may be involved with controlling unwanted saccades, as patients with frontal lesions often have difficulty suppressing saccades towards a visual target in an antisaccade task (Guitton, Buchtel, & Douglas, 1985). Burman and Bruce (1997) demonstrated that low-level electrical stimulation of the FEF can also suppress saccades. Monkeys were trained to make saccades to the location of targets after the fixation target was turned off. The target remained visible (visually guided saccade) or disappeared before the signal to make the saccade (memory guided saccade). Low-level electrical
stimulation was applied for 450 ms immediately after the fixation target disappeared. At some FEF sites, contralateral saccades were delayed by the stimulation (i.e. right saccades were suppressed when the left cortex was stimulated). When memory guided saccades were made, saccades were completely suppressed until the end of stimulation, and were reduced in amplitude by 25%. Visually guided saccades were delayed by 100 ms by the stimulation and were of normal amplitude.

Stimulation of most sites in the FEF elicits short-latency saccades. At purely suppressive sites, however, strong stimulation did not elicit saccades, although some smooth pursuit eye movements were observed. These sites were often associated with foveal-fixation cells or cells with postsaccadic activity. Most purely suppressive sites were located in the lateral FEF, slightly beyond sites where stimulation elicited small saccades. This region of the FEF projects largely to the rostral pole of the superior colliculus (Stanton et al., 1988); that also inhibits saccades when stimulated (Munoz & Wurtz, 1993a, 1993b).

Suppression was also observed at sites where saccades could be elicited by stimulation. Electrical stimulation too weak to elicit saccades was found to suppress visual saccades whose directions were different than those of saccades elicited by stronger stimulation at that site. This suppression is most likely due to competition between FEF saccadic movement cells, similar to that found between buildup and burst neurons in the superior colliculus (Munoz & Wurtz, 1995a, 1995b; Munoz & Istvan 1998; Meredith & Ramoa, 1998).

The frontal eye fields might play a role in visually evoked saccadic inhibition, because they have the ability to suppress saccades. However, the latency of visual input
and the unknown latency of saccadic suppression from this area would result in longer latencies of inhibition onset than reported in Reingold and Stampe (in press). The FEF might still have a role in inhibition with slower onset, or in extending the duration of inhibition. This inhibition might also be task-dependent, as memory-guided saccades and other tasks that rely on the FEF for execution (Deng, Goldberg, Seagraves, Ungerleider, & Mishkin, 1986) would be affected more than visually-guided saccades (Burman & Bruce, 1997).

**Supplementary Eye Fields**

The supplementary eye field (SEF) is located adjacent to the supplementary motor area (SMA) in dorsomedial frontal cortex (Schlag & Schlag-Rey, 1987). This area receives inputs from visual areas similar to those of the frontal eye fields and has similar projections to the saccade generator in the brainstem and to the superior colliculus (Shook et al., 1990). It also has strong connections to the frontal eye fields. Visual inputs to the SEF are slower than to the FEF, with average latencies of 92 ms (Schall, 1991), and only the slower sustained visuomovement cells are present. The representation of visual space and of saccades appears to be organized by orbital position of the eye in the head, rather than retinotopically as in the FEF. Extended electrical stimulation drives the eye to a fixed position in its orbit, whereas similar stimulation of the FEF produces a series of repetitive saccades (Schlag & Schlag-Rey, 1987; Schall, 1991; Tehovnik & Lee, 1993). The latency of saccades produced by stimulation is greater than that for stimulation of the FEF, and is lowest when the stimulation results in large changes in eye position. Many cells in the SEF fire during active fixation of a
target at the corresponding orbital location, whereas SEF saccade cells fire for specific activities during tasks (Bon & Lucchetti, 1992).

The long latency of visual input to the SEF and their task-oriented nature do not appear to support a role in saccadic inhibition, which is fast and may be less task dependent.

**Lateral Intraparietal Area (LIP)**

The lateral intraparietal area (LIP) is a region of parietal cortex from which saccades can be elicited by low levels of electrical stimulation (Shibutani, Sakata, & Hyvarinen, 1984). It projects directly to the intermediate layers of the superior colliculus (Lynch et al., 1985; Pare & Wurtz, 1997) and to the frontal eye fields (Andersen, Asanuma, Essick, & Siegel, 1990) but unlike the FEF and SEF, does not project to the saccade generator in the brainstem. The latency of electrically evoked saccades in the LIP is about 50 ms (Shibutani et al., 1984), which is longer than the 18-40 ms latency of similar saccades evoked from the FEF (Seagraves, 1992). The LIP receives visual input from both the ventral and dorsal streams of visual processing, and neurons show visual latencies of 60-140 ms (Yin & Mountcastle, 1977; Robinson, Goldberg, & Stanton, 1978), which is similar to visually-responsive cells in the FEF (Schall, 1991). Receptive fields in LIP are large, from 5° at the fovea to 30° in the periphery, and form a roughly retinotopically map (Blatt, Andersen, & Stoner, 1990). However, some cells in LIP appear to modify their response according to head and eye position, and the LIP may be part of a progression of spatial mappings from retinotopic to extrapersonal space (Thier & Andersen, 1996; Snyder, Grieve, Brotchie, & Andersen, 1998).
Neurons in LIP invariably respond to the onset of a target in their receptive field. This response occurs whether or not the target is relevant to the task. The response is enhanced both when a saccade is planned to the target, and when the target is attended covertly (Colby, Duhamel, & Goldberg, 1996). This enhancement is therefore due to visual attention, whereas similar enhancements seen in the superficial superior colliculus and FEF only occur when a saccade is to be made (Wurtz & Mohler, 1976). Targets brought into a neuron’s receptive field by a saccade do not activate the neuron unless the target has behavioral significance. It has been suggested that the LIP encodes a map of visual salience (Gottlieb, Kusunoki, & Goldberg, 1998), and the response to the abrupt onset of a target can then be explained by the saliency of its onset. In support of this model, the activity of neurons in LIP was found to be modulated by changes in the relevance of stimuli (Platt & Glimcher, 1997). In this study, two target LEDs were presented, one of which was in the cell’s receptive field. Either target could be designated as a saccadic target by a change in the color of the fixation LED. The neuron responded to the onset of the LED in its receptive field, but its response decreased after the LED was designated as a distractor by the change in the fixation LED’s color.

LIP neurons also fire when saccades are made, most strongly when a visual target is present. When a saccade to a learned location or a memory-guided saccade to a previously displayed target is made, the response is weaker. Covert attention to the target also produces a stronger response than either the learned or memory-guided saccades, suggesting that LIP is tied to visuospatial behavior and not saccades per se (Colby et al., 1996). No neurons in LIP have been found that fire for saccades that do not also show visual responses (Colby et al., 1996; Pare & Wurtz, 1997), although such neurons are
present in both the superior colliculus and FEF (Bruce & Goldberg, 1985; Seagraves & Goldberg, 1987). In addition, LIP neurons that project to the intermediate layers of the SC show sustained activity between the target presentation and the saccade in memory-guided saccade tasks (Pare & Wurtz, 1997). This presaccadic activity is similar to the behavior of buildup neurons in the SC. This suggests that the LIP projection to the SC encodes visual salience, whereas the FEF input to SC specifies the movement characteristics of desired saccades (Pare & Wurtz, 1997).

Kurylo (1991) has demonstrated that stimulation of the LIP can interfere with the production of saccades. Saccades can be elicited by electrical stimulation of the LIP, FEF or superior colliculus. When such an electrical stimulation is delivered during a visually guided saccade to either the FEF or the SC, the trajectory of the resulting saccade is a combination of the saccade that would have been evoked by the stimulation alone and the intended saccade to the target (Schlag-Rey, Schlag & Shook, 1989; Schiller & Sandell, 1983). In an attempt to duplicate this phenomenon in the LIP, Kurylo (1991) presented monkeys with a target at an eccentricity of 10° or 20° for 150 ms, after which the target was extinguished. Monkeys produced saccades to the target with an average latency of 241 ms when no stimulation was applied. Electrically evoked saccades with latencies of 40-120 ms were produced by stimulation of the LIP for a period of 200 ms. Visual target position was adjusted to produce saccades of orthogonal or opposite direction to the electrically evoked saccades.

Electrical stimulation was then delivered for 200 ms beginning at times from 300 ms before to 400 ms after the onset of the visual saccadic target. No mixing of the electrically and visually evoked saccades was seen. Instead, the visually evoked saccades
were either not produced or were delayed until 179 ms after the electrical stimulation ended. This effect occurred when the onset of electrical stimulation was delayed by 0 to 250 ms from the visual target appearance. The electrically evoked saccade was usually produced in this condition, whereas the probability of a visually guided saccade was reduced by 70%-90%. When the visually guided saccade was made, it remained accurately targeted to the position of the now-extinguished target.

It is possible to estimate the latency of the inhibition following the electrical stimulation from the reported data. The average latency of a visual saccade from target onset was 241 ms (SD=43) and the minimum number of visually guided saccades were observed when the delay from the target onset to electrical stimulation was 150 ms (Figure 3 of Kurylo, 1991). Therefore the stimulation inhibited most of the saccades that would have been made at a latency of 241-150, or 91 ms after the stimulation. At this latency, 90% of visual saccades were suppressed. About half of the expected number of visual saccades were observed for the stimulation delay of 250 ms. At this delay, the stimulation would have began when approximately half of the visual saccades were already initiated, implying that the latency to cancel a saccade must be very low, probably much less than the 43 ms standard deviation of the saccadic reaction times recorded with no stimulation applied.

Could LIP produce visually evoked saccadic inhibition, as observed by Reingold and Stampe (in press)? The evidence from Kurylo (1991) suggests that LIP can indeed cancel or delay saccades, but the latency at which this occurred could only be estimated from the reported data. Since the visually guided and electrically evoked saccades in this study were always incongruent, it is unknown whether interference would occur between
congruent visual and stimulation saccade vectors. It is also uncertain whether visual flashes would act in the same way as electrical stimulation upon the LIP. The latency of onset of saccadic inhibition for LIP would be the visual latency of 60-140 ms measured for visually responsive for neurons in LIP, plus to the unknown but small latency from electrical stimulation at which half of visually guided saccades were cancelled. This is unlikely to be as low as the 70 ms latency for the onset of saccadic inhibition reported by Reingold and Stampe. As with the frontal eye fields, the LIP might still have a role in inhibition with slower onset, or in extending the duration of inhibition
Introduction to Visually Evoked Potentials (VEP)

This section reviews the principles of visual evoked potential (VEP) research, and defines measures and a standard terminology for the various VEP peaks. Each VEP peak is discussed in terms of its characteristics and probable neural source. Typical stimuli and known low-level stimulus characteristics that affect the VEP are summarized. In the experimental section of this thesis, these effects will be discussed in greater detail and compared to analogous stimulus manipulations in visually-evoked saccadic inhibition studies.

The visually evoked potential is the spatial and temporal pattern of electrical voltages on the scalp induced by an abrupt changes in a visual stimulus. Scalp voltages are produced by the aggregate activity of large numbers of neurons in one or more regions of the cerebral cortex. Changes in these voltages following a change in visual input therefore reflects the time course and intensity of neural activity in the brain as it processes the new visual information. These potentials are recorded by scalp electrodes, with the number and placement of electrodes and the topography of the underlying cortex determining the contribution of each cortical area to the total signal. The most common electrode placement used in the studies reviewed in this thesis consisted of a single electrode placed 2.5 to 3 cm above the inion (the protuberance at the back of the skull), with a reference electrode attached to one or both earlobe or in the mid-frontal area. These correspond to the Oz, Ex, and Fz locations respectively, according to the conventions of the international 10-20 system (Jasper, 1958).
**VEP Methodology and Analysis**

The VEP is recorded by averaging the evoked scalp potentials over many repetitions (typically 100 to 200) of the visual stimulus. This reduces the effects of noise and of neural activity unrelated to the processing of the visual stimulus. The resulting plot of the potential in the period following the change in the visual stimulus consists of several positive and negative peaks. The timing and amplitude of each peak are interpreted as the latency and strength of the neural signal evoked by the visual stimulus at progressively later stages of cortical processing. An idealized VEP pattern in response to a flashed stimulus as measured at the Oz electrode placement is shown in Figure 8, with the most common peaks labeled.

![Idealized VEP pattern](image)

**Figure 8. Idealized VEP pattern, with labeled peaks.** Negative potentials are towards the top of the plot. A flat baseline immediately follows the visual event. C1 is the first peak caused by cortical activity in striate cortex, followed by peak P1 generated by extrastriate cortex. Following peaks N1, P2, and N2 indicate later neural activity. Amplitude and latency measures are shown for the N1 peak.
There is unfortunately much variability in the naming systems used for the various VEP peaks. The naming system adopted in this thesis is that used by Clark and Hillyard (1996). This is shown in Figure 8, and is common in contemporary papers. All of the results of the reviewed experiments will be reported using this naming system. Peaks from studies that used older nomenclatures have been renamed in this thesis.

Each of the peaks in the VEP can be recognized by its sequence, latency from the stimulus presentation, and polarity (positive or negative). This identification may not be straightforward, as changes in the stimulus characteristics or in electrode placement may cause peaks to disappear or to be inverted in polarity. The latency of each peak is generally measured by the time of its maximum or minimum voltage. The amplitude of each peak is measured in microvolts (μV), referenced to the baseline computed from the average voltage immediately following the display change. Some authors report amplitude as the difference between the peak voltages of a negative and positive peak, for example between P1 and N1.

**Characteristics and Sources of VEP Peaks**

Each peak in the VEP is generated by neural activity in one or more cortical areas. The precise cortical generator of each peak is difficult to determine, especially from recordings with one or two electrodes. When large ensembles of electrodes are used, computer algorithms such as BESA (Clark, Fan, & Hillyard, 1995) may be used to model the possible location of neural sources that might be responsible for the observed pattern of scalp potentials. The location of the generator for each peak is discussed below, as determined by Clark and Hillyard (1996) using BESA.
The first peak in the VEP pattern is C1, which is negative in polarity and has a latency of 50 to 100 ms from the visual event. This peak has been labeled N1, NP80, N100, and C0 by other authors. C1 appears to arise from the first cortical processing of the visual change in striate (primary visual) cortex (Clark & Hillyard, 1996) and therefore is the best measure of subcortical effects such as delays in the optic nerve. This peak is often of low amplitude, and is therefore not analyzed in many older studies. It may be recorded more reliably by presenting the stimulus to the left or right visual hemifield only, and subtracting the signals from electrodes placed to the left and right of the Oz position (Musselwhite & Jeffreys, 1982).

The second peak in the VEP is P1, which is positive in polarity and has a latency of 100 to 150 ms. Some authors have used other labels for this peak, including P120, C2, and CII. It appears to be generated in extrastriate cortex, and its latency and amplitude therefore reflect the effects of several stages of visual processing. The N1 peak that follows is negative in polarity, has a latency from 150 to 200 ms, and may be generated from multiple cortical areas, including the motion processing centers in area MT and by posterior temporal cortex. This peak has also been named N2 and N200. The late peaks P2 and N2 may also be identified, which show significant task-related and attention modulation.

**VEP Stimuli**

The stimuli used by the VEP studies reviewed in this thesis were simple flashes of light, square wave or sinusoidal gratings, or checkerboard patterns. Abrupt onset, reversal, or motion of these stimuli were used to induce the VEP response. When
patterned stimuli were used, luminance changes were avoided by abruptly replacing a blank background with the stimulus (pattern onset) or by replacing the stimulus with its negative image (pattern reversal). The amplitude and latency of early VEP peaks, especially C1 and P1, are known to be influenced by manipulation of elemental stimulus characteristics such as contrast and spatial frequency content. Similar manipulations of the display change will be made in the visually evoked saccadic inhibition paradigm in the experimental section of this thesis, and compared to the findings from previous VEP research.

The manipulations selected for these studies are luminance changes and stimulus contrast (Osaka & Yamamoto, 1978; Kubova, Kuba, Spekreijse, & Blakemore, 1995; Musselwhite & Jeffreys, 1985), size and retinal eccentricity of the stimulus (Osaka and Yamamoto, 1978; Brecelj & Prevec, 1983; Meredith & Celesia, 1982; Schlykowa, van Dijk, & Ehrenstein, 1993; Baseler, Sutter, Klein, & Carney, 1994), amplitude of stimulus displacement (Fenwick & Turner, 1977; Spekreijse, Dagnelie, Maier, & Regan, 1985), spatial frequency content (Parker & Salzen, 1977; Vassilev, Manahilov, & Mitov, 1983; Musselwhite & Jeffreys, 1985; Vassilev & Stomonyakov, 1987), and effect of isoluminant stimuli (Tobimatsu, Tomodo, & Kato, 1995; Wijers, Lange, Mulder, & Mulder, 1997).
Experiments

This section of the thesis documents a series of experiments that test the effects of stimulus characteristics on visually evoked saccadic inhibition. The General Method section outlines the procedure and text stimuli used for all studies, and defines the measures of saccadic inhibition used. This is followed by introduction, method, results, and discussion sections for each of the seven experiments.

General Method

Participants

A group of 10 participants were tested in each experiment. All participants had normal or corrected to normal vision, and were paid $10.00 for each one hour session. Each experiment required one to three sessions per participant, depending on the number of conditions.

Apparatus

The SR Research Ltd. EyeLink eye tracking system used in this research has high spatial resolution (0.005°) and a sampling rate of 250 Hz (4 ms temporal resolution). The three cameras on the EyeLink headband allow simultaneous tracking of both eyes and of head position, computing true gaze position with unrestrained head motion. By default, only the participant's dominant eye was tracked in our studies. The EyeLink system uses an Ethernet link between the eyetracker and display computers to supply real-time gaze position data for the generation of the gaze-contingent window. The on-line saccade
detector of the eye tracker was set to detect saccades with an amplitude of 0.5° or greater, using an acceleration threshold of 9500°/sec² and a velocity threshold of 30°/sec.

Participants viewed a 17" ViewSonic 17PS monitor from a distance of 60 centimeters, which subtended a visual angle of 30° horizontally and 22.5° vertically. The display was generated using an S3 VGA card and special software that supplied 120 Hz frame rates, fast switching between several images, and gaze-contingent windows. The average delay between an eye movement and the update of the gaze-contingent window was 14 ms.

Materials and Randomization

Participants read a short story for comprehension. The text was presented in black on a bright background. The luminance of the background was determined by the stimulus requirements of each experiment, but was constant for all trials within each experiment. Anti-aliased, proportional spaced text was used which had an average of 2.2 characters per degree of visual angle and an average of 10 lines of text per page.

One page of text in the story was displayed in each trial, with pages presented in the same order to all participants. The pairing of pages to conditions was determined randomly for each participant, constrained to allow no more than 3 contiguous trials of the same condition. The conditions and number of trials for each experiment are specified in detail later in this thesis.

Procedure

A 9-point calibration was performed at the start of each block of 18 to 32 trials, followed by 9-point calibration accuracy test. Calibration was repeated if the error at any
point was in error by more than 1°, or if the average error for all points was greater than 0.5°. Before each trial, a black fixation target was displayed at the center of the display. The participant fixated this target and the measured gaze position during this fixation was used to correct any post-calibration drift errors. In order to minimize pupil size changes, the background of the display during fixation had the same luminance as the normal text image to be displayed during the following trial.

One page of text was presented during each experimental trial. A transient image, determined by the experimental condition, was flashed for 33 ms at random intervals of 300 to 400 ms during trials. These transient images could be black, blank with the same luminance as the normal image, a sinusoidal grating, or a modified version of the normal image. In some experiments, a gaze-contingent window was used to limit the transient image to a small region of the participant's visual field centered on the participant’s current point of gaze. The time of the display of the transient image was recorded along with eye-movement data for later analysis. After reading each page, participants pressed a button to end the trial and proceed to the next page of the story.

Data Analysis

Eye tracker data files were processed to produce histograms of saccade frequency for the period following each display change. A separate histogram was compiled for each participant and condition, and analyzed to produce measures of the evoked saccadic inhibition. Composite histograms were generated using the saccades of all participants, and are reproduced in this thesis to illustrate the results of each experiment.
**Histogram Generation**

The times of display changes and of the onset of all saccades with amplitudes of 0.5° or greater were extracted from the eye tracker data files. Saccades occurring during display changes were discarded, and the remaining saccades were used to compile the histograms. The number of saccades in each histogram will be reported for each experimental condition, as a measure of the stability of the data set. A minimum of 800 saccades were required to generate reliable saccadic frequency histograms, which required about 5 minutes of reading per condition.

![Diagram](image)

Figure 9. A typical saccadic frequency plot, showing the dip and peak due to visually evoked saccadic inhibition. Boxes outline the four measures of saccadic inhibition.
A histogram of saccade frequency as a function of time after each display change was compiled for each participant and condition, as well as a composite histogram containing saccades from all participants. The histogram bin width was 4 ms, which was determined by the maximum temporal resolution of the eye tracker. These narrow bins provide high temporal resolution at the cost of few saccades (typically 10 to 30) in each bin and noisy individual participant histograms. To reduce this noise, a 7-bin running average filter was applied to each histogram, that replaced each bin with the average of that bin, the 3 previous, and the 3 following bins.

**Baseline and Normalization**

Figure 9 shows an idealized histogram, with the latency of saccades after a display change on the horizontal axis, and saccadic frequency on the vertical axis. The frequency of saccades remains unaffected by the display change for at least 48 ms following the display change (the baseline period). At longer latencies, a reduction in frequency caused by saccadic inhibition is followed by a peak associated with higher saccadic frequency during recovery from inhibition, after which saccadic frequency returns to the baseline rate.

The baseline period immediately following the display change is used to estimate the baseline or expected saccadic frequency, which is computed as the average saccadic frequency in the first 48 ms following the display change. All bins of the histogram were then normalized by dividing by the baseline saccadic frequency. This converts the histogram units into the ratio of measured to expected saccadic frequency, as shown on the vertical axis of Figure 9. The strength of saccadic inhibition, which is the proportion
of expected saccades eliminated by inhibition, can be computed for any time after the
display change by subtracting the normalized saccadic frequency from 1.0.

The average of the normalized saccadic frequency throughout the trial should be 1.0,
if the total number of saccades was not changed by the inhibition. A baseline saccadic
proportion measure can be computed to test this by dividing the number of saccades in
the first 48 ms following the display change by the total number of saccades in the first
300 ms of the histogram. This measure allows the assumption that saccadic probability is
unaffected in the baseline period to be tested, as its value should be relatively constant at
48/300 or ~0.15. This measure has been reported for each condition of each experiment.

Measures of Inhibition

Quantitative measures of the latency and strength of the saccadic inhibition are
computed from the sharp reduction in saccadic frequency ("dip") caused by inhibition in
the saccadic frequency plot. These measures are magnitude, \( L_{\text{ONSET}} \), \( L_{\text{MIN}} \), and duration,
and are shown in relation to the saccadic frequency curve in Figure 9 and described in
detail below.

**Period of Minimum Saccadic Frequency**

The first step in analysis of the histogram is to locate the bottom of the dip caused by
the inhibition. This is the time of the minimum saccadic frequency, which is located by
finding the lowest-valued three-bin average at latencies between 55 and 175 ms. The
minimum itself is not used as a measure as noise strongly affects both its value and
latency. Instead, the entire bottom of the dip is located, defined as the period of lowest
saccadic frequency. This is determined by the range of latencies in which the saccadic
frequency is no greater than the minimum saccadic frequency plus 10% of the difference between the baseline and minimum saccadic frequencies. This period is not used as a measure of inhibition itself, but is used to compute the other measures of saccadic inhibition.

**Magnitude of Inhibition (magnitude)**

The *magnitude of inhibition* (hereafter magnitude) is the proportion of expected saccades that are missing during the period of lowest saccadic frequency. A magnitude of 1.0 would mean that no saccades were produced at some time after the display change. Because the histogram has been normalized, the magnitude is simply computed as 1.0 minus the average normalized saccadic frequency during the period of lowest saccadic frequency.

**Latency of Minimum Saccadic Frequency (L_{MIN})**

The *latency of minimum saccadic frequency* (L_{MIN}), is defined as the time from the display change to the time of the strongest saccadic inhibition. L_{MIN} is computed as the midpoint of the period of lowest saccadic frequency. This measure is highly correlated to the sum of the onset of inhibition plus half the duration of inhibition. Therefore L_{MIN} will not be statistically analyzed in this thesis, but will be included in the experimental results for descriptive purposes.

**Latency of the Onset of Inhibition (L_{ONSET})**

The *latency of the onset of inhibition* (L_{ONSET}), is defined as the latency from the display change at which inhibition first reaches 50% of its greatest strength. This
criterion minimizes sensitivity to noise and to the slope of the sides of the dip. To minimize the effects of noise, this measure is actually computed by averaging the latency of all bins in which inhibition is between 33\% and 67\% of its greatest strength. This average is computed by scanning leftwards (at decreasing latencies) from $L_{\text{MIN}}$ until three consecutive bins in which the inhibition is below 33\% of its greatest strength are found.

**Duration of Inhibition (duration)**

The *duration of inhibition* (duration), is defined as the duration of the period in which inhibition remains above 50\% of its greatest strength. The duration is computed from the difference between $L_{\text{ONSET}}$ and the end of the period of inhibition. The end of the period of inhibition is found in the same way as $L_{\text{ONSET}}$, but scanning proceeds rightwards (at increasing latencies) from $L_{\text{MIN}}$.

**Experiment 1**

The first experiment will compare the saccadic inhibition evoked by a visual display change and an auditory tone. A study by Ross and Ross (1981) showed that saccadic reaction time (SRT) was increased if a visual warning was presented after the saccadic target had appeared, but no increase was observed when an auditory warning tone was substituted for the visual warning. If saccadic inhibition was responsible for the observed increase in SRT, then the probability of saccades should be changed after a display change, but should be unaffected by an auditory stimulus.

In addition, a control condition will be included that produces a null (invisible) display change. During analysis, the expected saccadic frequency (that would have been
seen if no display change had occurred) is estimated from the mean saccadic frequency in the baseline period (the 48 ms following the display change). The validity of this estimate will be tested by comparing the saccadic frequencies in the baseline periods of the real and null display change conditions using the baseline saccadic proportion measure.

**Method**

In the black display change condition, a black (4.0 cd/m²) transient image momentarily replaced the normal text image as described in the General Method section. The control condition used a transient image identical to the normal image, resulting in no visible display change. These visual stimulus conditions are shown in Figure 10. In the auditory stimulus (beep) condition, a 2000 Hz square-wave tone was produced during
the period in which the transient image would have been displayed. Display changes and beeps both lasted for 33 ms and occurred at random intervals between 300 and 400 ms. The average brightness of the normal image was 40 cd/m². A total of 72 pages of text were presented, with 24 randomly assigned to each condition.

![Figure 11. Saccadic frequency histograms from Experiment 1. No saccadic inhibition was evoked by the Beep and Control conditions, whereas strong inhibition was observed in the Black condition.](image)

**Results and Discussion**

Eye movement data were analyzed as described in the General Method section. An average of 2190 saccades were used to create the histogram of saccade frequency in each condition for each participant. Saccadic frequency following the display change (black
or control) or beep are plotted in Figure 11. No dip due to saccadic inhibition was present in either the control or the beep conditions, confirming that a visual change is required to evoke saccadic inhibition and that the auditory tone was an ineffective stimulus. The baseline saccadic proportion was not significantly different between any condition ($F(18,2)=2.98$, $p=0.076$). This confirms that the baseline is a good estimate of the saccadic frequency that would be seen in the absence of display changes.

Table 3. Results of Experiment 1. Values in parenthesis are standard errors.

<table>
<thead>
<tr>
<th>Condition</th>
<th>$L_{\text{onset}}$ ms</th>
<th>Duration ms</th>
<th>$L_{\text{min}}$ ms</th>
<th>Magnitude proportion</th>
<th>Saccades total count</th>
<th>Baseline Saccadic Proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>2213 (179)</td>
<td>0.147 (0.003)</td>
</tr>
<tr>
<td>Beep</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>2222 (163)</td>
<td>0.143 (0.002)</td>
</tr>
<tr>
<td>Black</td>
<td>68.2 (1.1) 52.8 (1.5)</td>
<td>93.6 (2.2)</td>
<td>.791 (.027)</td>
<td>2135 (163)</td>
<td>0.153 (0.003)</td>
<td></td>
</tr>
</tbody>
</table>

Table 3 lists the measured magnitude and latencies of the inhibition evoked by the black display change. The strongest inhibition occurred 93.6 ms after the display change, when 79.1% of expected saccades were missing (magnitude=.791). The inhibition first reached 50% of its maximum strength at 68.2 ms after the display change, as measured by $L_{\text{onset}}$, and remained at greater than 50% of its maximum strength for 52.8 ms. The latency of the onset of saccadic inhibition is similar to the 70 ms seen by Reingold and Stampe (in press), where similar text and a black transient image were also used. Low-latency saccadic inhibition was produced by all participants in this experiment, despite instructions to ignore the flickering of the display.
Experiment 2

The salience of a change in visual input has been observed to affect the amplitude of the VEP response. For example, the amplitude of early (C1 and P1) VEP peaks was found to increase with luminance (Osaka & Yamamoto, 1978) and pattern contrast (Kubova et al., 1995; Musselwhite & Jeffreys, 1985). A similar effect has been demonstrated using step displacements of a pattern. Fenwick and Turner (1977) displayed black and white checkerboard patterns with 0.33° squares to their participants. The pattern was shifted to the left or right once per second, by ¼ of the width of a square (0.083°), ½ square (0.17°), or ¾ square (0.25°). VEP amplitude was measured as the difference between the P1 and N1 peaks in this study, and increased from 4.96 μV for the 0.083° displacement to 7.13 μV for the 0.17° displacement and 9.25 μV for the 0.25° displacement. No significant shifts in latency of the P1 peak were seen, which was approximately 93 ms for all displacements. A similar pattern of results was observed in a study by Spekreijse et al. (1985) using displacements from 0.05° to 0.175° of a 20% contrast checkerboard with 0.2° squares. The amplitude of the C1 VEP peak increased linearly with increasing displacements, whereas the latency of the C1 peak remained constant at 81 ms for all displacements.

Method

This experiment will use a similar manipulation of displacement size with visually evoked saccadic inhibition. Step displacements of the display of text could not be used, as this was found to cause microsaccades at low latencies following the displacement.
Instead, a transient image consisting of a vertically shifted version of the normal image was displayed for 33 ms, producing an intermittent jitter of the entire display. Because of the short presentation of the shifted text, reading was not affected and extraneous saccades were avoided.

Figure 12. Displacement and Black display change stimuli used in Experiment 2.

The normal image consisted of pages of text as in Experiment 1 (average brightness 40 cd/m²). Transient images were shifted down by 0.1°, 0.3°, or 0.6°, with the largest
shift displacing the text by the height of a lowercase character. In addition, a black (4.0 cd/m²) transient image was included, as used in Experiment 1. This allowed within-participant comparisons between displacement and black stimulus types. Each condition is illustrated in Figure 12, and was randomly assigned to 20 pages of text, for a total of 80 trials.

![Saccade frequency histograms from Experiment 2.](image)

**Figure 13:** Saccade frequency histograms from Experiment 2. Magnitude is seen to increase for larger displacements, while latency decreases. Inhibition in the Black condition is faster than in any displacement condition.

**Results and Discussion**

An average of 1573 saccades were analyzed from each participant in each condition. Measures of the evoked inhibition for each condition are listed in Table 4. Examining the
plot of saccadic frequency in Figure 13, it can be clearly seen than the magnitude of saccadic inhibition increases for larger displacements \( F(2,18)=61.6, p<.001 \), with all comparisons significant \( (p<.05 \text{ for all}) \). This is similar to the findings for amplitude of the VEP responses found by Fenwick & Turner (1977) and Spekreijse et al. (1985).

The latency of inhibition was found to decrease with larger displacements, an effect that was not seen in the VEP studies. There was a significant overall effect for \( L_{\text{onset}} \), \( F(2,18)=3.76, p<.05 \), but only the 0.3° and 0.6° displacement conditions differed significantly \( (t(9)=2.89, p<.05) \), whereas comparison of the 0.1° and 0.6° displacement conditions showed a marginal effect \( (t(9)=2.01, p=.075) \). This suggests that there is a consistent effect of displacement size on latency, but that the power of this study \( (n=10) \) was insufficient to bring all pairwise comparisons to significance.

Table 4. Results of Experiment 2. Values in parenthesis are standard errors.

<table>
<thead>
<tr>
<th>Condition</th>
<th>( L_{\text{onset}} )</th>
<th>Duration</th>
<th>( L_{\text{min}} )</th>
<th>Magnitude proportion</th>
<th>Saccades total count</th>
<th>Baseline Saccadic Proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ms</td>
<td>ms</td>
<td>ms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.1° offset</td>
<td>87.5 (3.8)</td>
<td>34.3 (2.8)</td>
<td>109.2 (2.5)</td>
<td>0.380 (0.026)</td>
<td>1590 (109)</td>
<td>0.149 (0.001)</td>
</tr>
<tr>
<td>0.3° offset</td>
<td>84.6 (2.0)</td>
<td>35.4 (1.6)</td>
<td>104.8 (1.5)</td>
<td>0.597 (0.042)</td>
<td>1566 (107)</td>
<td>0.147 (0.003)</td>
</tr>
<tr>
<td>0.6° offset</td>
<td>78.8 (1.7)</td>
<td>43.0 (2.1)</td>
<td>103.0 (1.5)</td>
<td>0.718 (0.046)</td>
<td>1591 (109)</td>
<td>0.148 (0.003)</td>
</tr>
<tr>
<td>Black</td>
<td>68.4 (1.8)</td>
<td>48.9 (2.9)</td>
<td>93.0 (1.9)</td>
<td>0.765 (0.034)</td>
<td>1545 (103)</td>
<td>0.154 (0.003)</td>
</tr>
</tbody>
</table>

The duration of inhibition also increased for the larger displacements \( F(2,18)=5.11, p<.05 \). The 0.6° displacement condition differed significantly from both the 0.3° displacement \( (t(9)=2.87, p<.05) \) and 0.1° displacement conditions \( (t(9)=2.28, p<.05) \), but
there was no difference between the duration of the inhibition evoked by the 0.1° and 0.3° displacements ($t(9) < 1$).

Comparison of the saccadic inhibition evoked by the 0.6° displacement of the text with that produced by the black display change shows that the latency of the onset of inhibition was 10.4 ms faster for the black than the displacement stimulus. The change in $L_{\text{ONSET}}$ between these conditions was highly significant ($t(9) = 6.74, p < .001$). No significant difference in duration of inhibition ($t(9) = 1.84, p = .099$) or magnitude of inhibition ($t(9) = 1.45, p = .18$) were found between these conditions. The type of visual stimuli was different in these conditions: a luminance change caused by the black transient image and motion caused by the displaced transient images. Because magnitudes were similar in these two conditions, it is difficult to argue that inhibition caused by the black display change was faster because of higher salience. This suggests that different types of visual changes may evoke inhibition of different latencies, and that strong luminance changes produce very fast inhibition.

**Experiment 3**

In the previous experiment, stimulus salience was manipulated by changing the size of a transient displacement of the display. A more fundamental stimulus characteristic that determines salience is the luminance or contrast of the stimulus. In VEP studies, the amplitude of early VEP peaks C1 and P1 was found to be affected by the contrast or luminance of the stimulus. In one study of pattern contrast, Kubova et al. (1995) presented large (35°) pattern-reversal checkerboard stimuli with contrasts from 1.3% to 96%. The amplitude of the P1 peak was found to be proportional to the logarithm of the
stimulus contrast. A possible source of this relation may be the nonlinear response of M ganglion cells in the retina (e.g., Hicks, Lee, & Vidyasagar, 1983). The latency of P1 was also found to increase by about 25 ms as contrast decreased. This change in latency with contrast has also been seen for both X (parvocellular) and Y (magnocellular) ganglion cells in the cat (Sestokas & Lehmkuhle, 1986). Similar changes in latency with contrast were observed in the C1 peak (Musselwhite & Jeffreys, 1985), and the amplitude of this peak also appeared to be proportional to the logarithm of contrast, as estimated from VEP waveforms from a single participant (Figure 7 of Musselwhite & Jeffreys, 1985).

A similar effect of stimulus luminance was observed by Osaka and Yamamoto (1978), who flashed 1° targets for 50 ms on a dark background. P1 latency was found to increase by 15 ms as luminance was decreased from 2600 cd/m² to 26 cd/m². Although no amplitude data were reported, estimates from the VEP waveforms for a single participant (Figure 1 of Osaka & Yamamoto, 1978) appear to confirm a logarithmic relation between the luminance of the target and P1 amplitude.
Figure 14. Stimuli used for Experiment 3. These are small 1° squares that are flashed at the participant’s fovea using a gaze-contingent window. The three levels of contrast (11%, 33%, and 100%) used in this study are shown.

Method

In this experiment, the effects of contrast and luminance on saccadic inhibition evoked by a small stimulus were investigated. The stimulus used was a 1° square, positioned on the participant’s fovea by a gaze contingent window. Both luminance and contrast of the stimulus were changed relative to the normal text image, by mixing a black transient image with the normal image in different proportions as shown in Figure 14. The contrast of the stimulus can be computed from the average luminance inside and outside the gaze-contingent window while the transient image is displayed, using the formula (outside-inside)/(outside+inside). The mixture levels were selected to create contrasts of 11%, 33% and 100%. Each contrast level differed from the previous by a
factor of 3, resulting in equal logarithmic contrast steps. A total of 78 pages of text were presented, with 26 pages in each contrast condition.

![Graph showing saccadic frequency histograms for Experiment 3. Magnitude of inhibition increases and latency decreases with increasing contrast.](image)

**Figure 15:** Saccadic frequency histograms for Experiment 3. Magnitude of inhibition increases and latency decreases with increasing contrast.

**Results and Discussion**

An average of 1540 saccades were analyzed from each participant for each of the contrast conditions. Inhibition measures are reported in Table 5, and Figure 15 shows the saccadic frequency histograms for each condition. The magnitude of the inhibition was found to be significantly affected by the overall manipulation of contrast ($F(2,18)=6.59$, $p<.05$). The magnitude of inhibition in the 100% contrast condition differed significantly
from the 33% contrast ($t(9)=6.41, p<.001$) and 11% contrast conditions ($t(9)=3.07,$
p<.05$). However, the magnitude of the inhibition in the 33% contrast and 11% contrast
conditions did not significantly differ ($t(9)=1.19, p=.23$).

Table 5. Results of Experiment 3. Values in parenthesis are standard errors.

<table>
<thead>
<tr>
<th>Condition</th>
<th>$L_{\text{onset}}$ ms</th>
<th>Duration ms</th>
<th>$L_{\text{min}}$ ms</th>
<th>Magnitude proportion</th>
<th>Saccades total count</th>
<th>Baseline Saccadic Proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td>11% contrast</td>
<td>94.8 (1.1)</td>
<td>32.4 (2.1)</td>
<td>111.4 (2.0)</td>
<td>0.564 (0.046)</td>
<td>1528 (166)</td>
<td>0.139 (0.003)</td>
</tr>
<tr>
<td>33% contrast</td>
<td>86.8 (3.2)</td>
<td>35.4 (2.9)</td>
<td>106.4 (1.8)</td>
<td>0.614 (0.031)</td>
<td>1550 (151)</td>
<td>0.144 (0.004)</td>
</tr>
<tr>
<td>100% contrast</td>
<td>81.4 (1.7)</td>
<td>35.8 (2.8)</td>
<td>100.8 (1.9)</td>
<td>0.687 (0.031)</td>
<td>1542 (169)</td>
<td>0.147 (0.002)</td>
</tr>
</tbody>
</table>

This increase in magnitude with increasing contrast is qualitatively similar to the
relation between amplitude and contrast or luminance observed in VEP studies, where a
logarithmic relation between contrast and amplitude of response was seen.
Unfortunately, the relatively small changes in magnitude observed in this study were not
sufficient to quantitatively discriminate between a linear or logarithmic relationship to
contrast for saccadic inhibition.

The latency of evoked saccadic inhibition decreased with increasing contrast, as was
seen in the VEP studies. The change in $L_{\text{onset}}$ was significant ($F(2,18)=11.81, p<.01$),
however the comparison between the 100% and 33% contrast conditions did not reach
significance ($t(9)=1.58, p=.15$). $L_{\text{onset}}$ in 11% contrast condition differed significantly
from both the 33% contrast ($t(9)=8.64, p<.001$) and 100% contrast condition ($t(9)=2.68, p<.05$). No effects of contrast on the duration of inhibition were found ($F(2,18)<1$).

In general, the effect of stimulus contrast on evoked saccadic inhibition was found to be similar to that seen in VEP studies, with stronger inhibition evoked by higher contrast display changes. However, this effect was much weaker than was seen in the VEP studies. High contrast stimuli also evoked lower latency inhibition than did lower contrast stimuli, an effect that was also present in the VEP studies. These findings are similar to those seen when stimulus saliency was manipulated by displacement amplitude in Experiment 2, suggesting that higher salience may evoke stronger and faster saccadic inhibition for all types of stimuli.

**Experiment 4**

A consistent finding in VEP studies is that high frequency grating stimuli produce increased response latency when compared to low frequency gratings (Parker & Salzen, 1977, 1982; Parker, Salzen & Lishman, 1982; Vassilev et al., 1983; Musselwhite & Jeffreys, 1985; Vassilev & Stomonyakov, 1987). The source of this latency effect may be subcortical, as both Y cells (magnocellular) and X cells (parvocellular) in the cat have been observed to show a clear correlation between the spatial frequency of the stimulus and the latency of the neural response at the LGN (Sestokas & Lehmkuhle, 1986). As this latency shift arises in the retina and optic nerve, it should affect the latency of all cortical and subcortical responses, including visually evoked saccadic inhibition.

The effect of stimulus frequency content on the latency of early peaks of the VEP appear to be universal, and has been observed for the onset, offset, or reversal of
sinusoidal gratings (Parker & Salzen, 1977, 1982); over a wide range of contrasts (Vassilev et al., 1983); and for both sinusoidal and square wave gratings (Musselwhite & Jeffreys, 1985). The size of the subcortical latency effect is best measured from the C1 peak, which is the earliest cortical component of the VEP and should be least contaminated by cortical processing effects. The latency of C1 was found to be relatively constant for spatial frequencies below 2 cycles per degree (c/deg), then rose by 15 to 30 ms by 10 c/deg, which corresponds to about 5 to 10 ms per octave of frequency (Musselwhite & Jeffreys, 1985). A similar latency shift was observed by Vassilev and Stomonyakov (1987) using sinusoidal gratings at 4 and 6 times the psychophysical contrast threshold, with C1 latency increasing by 30 ms between 2 and 16 c/deg, or about 10 ms per octave of frequency.

This study will attempt to reproduce the spatial frequency effect with visually evoked saccadic inhibition. The display change to evoke transient inhibition will momentarily replace the normal text image with a high or low frequency luminance-matched grating transient image. If the effect of spatial frequency on the VEP latency is indeed due to neural delays at a subcortical level, then the latency of visually evoked saccadic inhibition should be affected in a similar fashion to the VEP latency, whether the locus of saccadic inhibition is cortical or subcortical. As a control for the removal of the text during the display of the gratings, a black and a blanked transient image were included.
Figure 16. Stimuli used in Experiment 4. These images show the display when the transient image is displayed, using a 6° gaze contingent window. The transient images are low and high frequency gratings, and black and blanked images.

**Method**

Low and high frequency sinusoidal gratings (0.5 cycles per degree and 6.0 cycles per degree respectively) were used as transient images. Contrast of the gratings were 50%, computed as \((max-min)/(max+min)\) where \(max\) and \(min\) are the maximum and minimum luminance of the gratings respectively. The average luminance of the gratings were adjusted to match that of the text image (34 cd/m²). A luminance-matched blank
transient image was used to remove the text without displaying a grating. Finally, a black transient image (4 cd/m²) both removed the text and changed the luminance of the display. Because high spatial frequencies are less salient in peripheral vision, the display changes were limited to the foveal and parafoveal areas of the visual field by a 6° square gaze-contingent window centered on the participant's point of gaze. A total of 112 pages of text were presented, with 28 pages randomly assigned to each of the four conditions illustrated in Figure 16.

**Results and Discussion**

An average of 2386 saccades were analyzed from each participant in each condition. Inhibition magnitude and latencies for each condition are reported in Table 6. The saccadic frequency plots in Figure 17 compare the inhibition evoked by the low and high frequency gratings separately from the black and blanked images.

Inhibition evoked by the high-frequency grating was of significantly longer latency than that caused by the low-frequency grating, by 12.0 ms for $L_{\text{ONSET}}$ ($t(9)=5.28$, $p<.01$) and 14.8 ms for $L_{\text{MIN}}$. The effect of spatial frequency on latency is approximately 4 ms per octave, which is half that observed for similar gratings in VEP studies (8 ms/octave, Parker and Salzen, 1977; 5-10 ms/octave, Musselwhite and Jeffreys, 1985; 10 ms/octave, Vassilev and Stomonyakov, 1987). The magnitude of the inhibition evoked by the low frequency grating was somewhat stronger than that produced by the high frequency grating, however this difference was only marginally significant ($t(9)=1.97$, $p=.08$). The duration of the inhibition did not differ significantly for any of the conditions ($F(3,27)=2.02$, $p=.16$).
Figure 17. Saccade frequency histograms from Experiment 4. Left: comparison of inhibition evoked by low and high-frequency gratings, which was of different latencies. Right: comparison of inhibition evoked by black and blanked transient images. The latency shift is similar to that observed for the gratings.

Table 6. Results of Experiment 4. Values in parenthesis are standard errors.

<table>
<thead>
<tr>
<th>Condition</th>
<th>L_{onset}</th>
<th>Duration</th>
<th>L_{min}</th>
<th>Magnitude proportion</th>
<th>Saccades total count</th>
<th>Baseline Saccadic Proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black</td>
<td>74.0 (1.1)</td>
<td>43.0 (2.5)</td>
<td>95.2 (1.9)</td>
<td>0.730 (0.027)</td>
<td>2386 (249)</td>
<td>0.152 (0.003)</td>
</tr>
<tr>
<td>Blanked</td>
<td>85.6 (1.4)</td>
<td>39.7 (2.2)</td>
<td>103.2 (1.6)</td>
<td>0.706 (0.028)</td>
<td>2394 (265)</td>
<td>0.147 (0.004)</td>
</tr>
<tr>
<td>0.5 cy° grating</td>
<td>76.6 (1.0)</td>
<td>42.0 (1.9)</td>
<td>95.0 (1.6)</td>
<td>0.800 (0.021)</td>
<td>2372 (246)</td>
<td>0.152 (0.004)</td>
</tr>
<tr>
<td>6.0 cy° grating</td>
<td>88.6 (2.2)</td>
<td>38.4 (2.4)</td>
<td>109.8 (1.9)</td>
<td>0.728 (0.045)</td>
<td>2380 (252)</td>
<td>0.142 (0.005)</td>
</tr>
</tbody>
</table>
A similar difference in latency was also observed for the inhibition evoked by the black transient image and the luminance-matched blank image. Inhibition was significantly faster when evoked by the black transient image than when produced by the blanked image, by 11.6 ms for \text{L}_{\text{ONSET}} (t(9)=8.77, p<.001) and 8.0 ms for \text{L}_{\text{MIN}}. The magnitude of inhibition (t(9)=1.27, p=.24) did not differ significantly between these conditions.

Low-latency saccadic inhibition was evoked by both the black and low-frequency grating transient images, that caused changes in display luminance and low-frequency content respectively. The luminance change caused by the black transient image evoked inhibition of lower latency than did the display of the low-frequency grating, as measured by \text{L}_{\text{ONSET}} (t(9)=2.75, p<.05). The low-frequency grating also caused significantly stronger inhibition than did the black image (t(9)=4.53, p<.01), suggesting that the presence of contours in the transient image enhanced the inhibition.

Inhibition of significantly longer latency was evoked by the display of both the blank image and the high frequency grating, as compared to that produced by the black or the low-frequency grating images. No significant difference in the latency of inhibition was found for \text{L}_{\text{ONSET}} (t(9)=1.31, p=.22). The magnitude of evoked inhibition did not differ significantly between the high-frequency grating and blank conditions (t(9)<1), suggesting that these transient images produced display changes of equal salience.

The similar latency effects observed for the high—low frequency grating and the black—blanked transient image comparisons in Figure 17 can be understood in terms of the change in spatial frequency content produced when the text was replaced by the transient image. The display of the blank image removed the text, which contained
mostly high spatial frequency detail. This resulted in a sudden decrease in the high frequency content of the image. When the high-frequency grating replaced the text, the much stronger grating replaced the high frequencies in the text, which increased the total high frequency content of the display. Only the high frequency content changed in both of these conditions, which resulted in saccadic inhibition of relatively long latency.

Presentation of the black image and the low-frequency grating also removed the text, and must have reduced the high-frequency content of the display in the same way that displaying the blanked transient image did. However, this change in the high-frequency content of the display did not appear to affect the latency of the inhibition evoked by the black or low-frequency grating transient images. The lack of effect of the high-frequency components in the presence of low-frequency or luminance components of the display change may be explained by a simple race model, in which saccadic inhibition is always initiated by the first component of the display change to arrive at the neural substrate of inhibition. Because the luminance and low-frequency components travel through the visual system faster than the high-frequency components, inhibition should always be initiated by the faster luminance and lower-frequency components if these are present.

**Experiment 5**

In Experiment 4, saccadic inhibition of relatively long latency was produced by momentarily replacing the text with a luminance-matched blanked image, which removed the text and thereby reduced the high frequency content of the display. The latency of the inhibition appeared to be determined by the difference in spatial frequency content between the normal and transient images, rather than the contents of either image.
individually. If this hypothesis is valid, then manipulating the frequency content of the normal image should change the latency of saccadic inhibition evoked by a blank transient image. Specifically, changing from a text image containing mainly low frequency components to a blank transient image should cause a change in low frequency content, and evoke low-latency inhibition. This same transient image when paired with a text image containing mostly high frequency components should cause a change in high frequency content, and evoke longer latency inhibition.

It was also observed in the previous experiment that the luminance change caused by a black transient image evoked low-latency inhibition, and that the change in high spatial frequency content caused by the removal of the text appeared to have no effect in the presence of the luminance change. If this is true, then the frequency content of the normal image should not affect the time course of the inhibition evoked by a black transient image. Both hypotheses will be tested in this experiment.

**Method**

Images of text similar to those used in previous experiments were manipulated in frequency content by high-pass or low-pass filtering. A Gaussian low-pass filter with a standard deviation of 2.0 cycles per degree in the frequency domain was applied to create low frequency text (LF text) images. High-frequency text (HF text) images were created by subtracting the LF images from the originals, and adding a constant to equalize the luminance of the HF and LF text images. The filtered text is shown in Figure 18, and is only slightly degraded in readability, with LF text appearing blurred and HF text appearing to have white borders around the characters. Two types of transient images were used: a blank image matched in luminance to the HF and LF text images (40 cd/m²),
and a black image (4 cd/m²). Each type of transient image was presented in combination with both HF and LF text images, as shown in Figure 18. A gaze-contingent window limited the display of the transient image to a 6° square centered on the participant's point of gaze. A total of 120 pages of text were presented, randomly assigned with 30 pages in each of the 4 conditions.

Figure 18. Stimuli used in Experiment 5. These images show the display when the transient image is displayed, using a 6° gaze contingent window. Normal images of low frequency and high frequency content text were fully crossed with blanked and black transient images to create the four conditions shown here.
Results and Discussion

An average of 1752 saccades were analyzed from each participant in each condition. The inhibition evoked by the black and blanked transient images when presented in combination with the LF and HF filtered text are plotted in Figure 19. Measures of the inhibition for each condition are reported in Table 7.

Figure 19. Saccade frequency histograms from Experiment 5. Left: comparison of inhibition evoked by black transient image replacing LF and HF text. No differences were seen in the time course of inhibition. Right: comparison of inhibition evoked by blanked transient images. A very large latency difference is observed between LF and HF text conditions.
Table 7. Results of Experiment 5. Values in parenthesis are standard errors.

<table>
<thead>
<tr>
<th>Condition</th>
<th>L&lt;sub&gt;onset&lt;/sub&gt;</th>
<th>Duration</th>
<th>L&lt;sub&gt;min&lt;/sub&gt;</th>
<th>Magnitude</th>
<th>Saccades</th>
<th>Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ms</td>
<td>ms</td>
<td>ms</td>
<td>proportion</td>
<td>total count</td>
<td>Proportion</td>
</tr>
<tr>
<td>LF, Black</td>
<td>72.8 (2.0)</td>
<td>44.4 (3.4)</td>
<td>93.2 (2.5)</td>
<td>0.674 (0.031)</td>
<td>1753 (209)</td>
<td>0.146 (0.003)</td>
</tr>
<tr>
<td>HF, Black</td>
<td>72.4 (1.9)</td>
<td>46.2 (3.7)</td>
<td>95.2 (2.7)</td>
<td>0.674 (0.038)</td>
<td>1779 (213)</td>
<td>0.153 (0.003)</td>
</tr>
<tr>
<td>LF, Blanked</td>
<td>82.0 (2.1)</td>
<td>41.8 (2.1)</td>
<td>102.8 (1.9)</td>
<td>0.726 (0.031)</td>
<td>1762 (201)</td>
<td>0.148 (0.003)</td>
</tr>
<tr>
<td>HF, Blanked</td>
<td>101.1 (1.9)</td>
<td>46.4 (2.2)</td>
<td>126.0 (2.9)</td>
<td>0.547 (0.038)</td>
<td>1714 (203)</td>
<td>0.143 (0.004)</td>
</tr>
</tbody>
</table>

No difference was observed in the time course of the saccadic inhibition evoked by the black transient image when used with the LF or HF text normal images, and no significant differences were found for any measure of latency or magnitude (t(9)<1 for all tests). In contrast, the blanked transient image evoked very different inhibition when paired with the LF compared to the HF text. This confirms that it is not the transient image alone that sets the time course of saccadic inhibition, but the difference between the normal and transient images. As predicted, the latency of inhibition was longer for the HF text, by 18.1 ms for L<sub>onset</sub> (t(9)=6.11, p<.001) and 23.2 ms for L<sub>min</sub>. No such difference was seen for inhibition evoked by the black transient image, confirming that the effects of changes in high-frequency content are suppressed by changes in luminance. The interaction between the effects of the black—blanked transient image and the HF—LF text normal images in determining the latency of inhibition is also significant.
The duration of the inhibition did not differ significantly for any of the conditions (F(3,27)=1.02, p=.40).

The blank transient image evoked much stronger inhibition in combination with the LF text than with the HF text (t(9)=6.01, p<.001). This is similar to the stronger inhibition evoked by the low frequency grating in Experiment 4, which may indicate that saccadic inhibition is more sensitive to the lower spatial frequencies.

**Experiment 6**

Tobimatsu et al. (1995) investigated the effect of isoluminant (equal luminance red and green), colored (red and green), and achromatic (gray) sinusoidal gratings on the VEP response. The measured latency of C1 for 2 cycle per degree sinusoidal gratings was found to be 92 ms for achromatic (black and white) gratings, 98 ms for chromatic (red/black) gratings, and 120 ms for isoluminant (red/green) gratings. The amplitude of C1 also increased from 2 μv for achromatic to 9 μv for isoluminant gratings, although this increase may be due to the significantly changed shape of the VEP evoked by isoluminant stimuli (Berninger & Arden, 1991). Isoluminant stimuli were also used by Wijers et al. (1997), who presented green letters on a black background (nonisoluminant condition) or on a luminance-matched grey background (isoluminant condition) while recording VEP responses. Latency of all VEP peaks increased at isoluminance, by 28 ms for the C1 peak and 47 ms for the P1 peak. Unlike Tobimatsu et al. (1995), no large changes in amplitude was seen. This may be due to choice of a mid-frontal reference electrode instead of the linked earlobe reference used by Tobimatsu et al. (1995).
The large increase in latency observed for isoluminant stimuli was hypothesized by these authors to be due to the inability of the fast magnocellular visual pathway to respond to isoluminant stimuli. Isoluminant stimuli are thought to be limited to the slower parvocellular visual pathway, which contains color-opponent channels (Hubel & Livingstone, 1990). It is known that isoluminant stimuli produce perceptual deficits in detection of texture, motion and stereopsis (Logothetis, Schiller, Charles, & Hurlbert, 1990; Schiller, Logothetis, & Charles, 1990), which may be due to the lack of activation of the magnocellular system by these stimuli.

Isoluminant display changes may provide clues to the neural locus of saccadic inhibition, as the superior colliculus does not appear to receive input from color-opponent ganglion cells in the retina either directly (Schiller & Malpeli, 1977) or via corticotectal projections (Schiller et al., 1979). In support of this, moving isoluminant color edges produce only weak responses in the superficial (visual) superior colliculus (Marrocco & Li, 1977). Therefore this experiment will investigate whether saccadic inhibition can be evoked by isoluminant display changes.

**Method**

Three conditions were included in this experiment: an achromatic luminance condition, a color isoluminance condition, and a mixture condition that combined color and luminance changes. Unlike most previous experiments, the text remained visible within the transient image, while only the color or luminance of the background changed. The luminance condition used a gray text background for the normal image (average luminance 24 cd/m²), and a white background in the transient image (46 cd/m²), to create a change in luminance. The isoluminance color condition used a red background for the
normal image (24 cd/m², CIE coordinates x=.524, y=.348) and a luminance-matched green background in the transient image (24 cd/m², CIE coordinates x=.295, y=.584). The mixed color and luminance condition used the same red normal image and a brighter green background in the transient image (46 cd/m², CIE coordinates x=.295, y=.584). A gaze-contingent window was used to limit the transient images to a 6° square centered on the participant's fovea, to avoid changes in color perception in the peripheral visual field. These stimuli are shown in Figure 20. A total of 72 pages of text were presented, with 24 pages randomly assigned to each condition.

![Luminance and Isoluminant Color](image)

**Figure 20.** Stimuli used in Experiment 6. Achromatic luminance and color isoluminant changes are presented, as well as a mixture condition that includes both luminance and color changes. The color conditions both have red normal images and green transient images.
Results and Discussion

An average of 1424 saccades were analyzed from each participant in each condition. Inhibition magnitude and latencies for each condition are reported in Table 8, and saccadic frequency plots are shown in Figure 21. Saccadic inhibition is clearly evoked by the isoluminant stimulus, although this inhibition was significantly weaker than that evoked by the luminance change ($t(9)=3.69, p<.01$). The onset of inhibition was delayed by 20.1 ms in the isoluminance condition compared to the luminance condition ($t(9)=12.04, p<.001$). This delay is similar to the 28 ms difference in VEP latency reported by Tobimatsu et al. (1995) for isoluminant and luminance grating stimuli. The duration of the inhibition is similar in the luminance and isoluminance conditions ($t(9)<1$).

In the mixture condition, both color and luminance display changes were present. The onset of inhibition was as fast in the mixture condition as in the luminance condition ($t(9)=1.60, p=.132$), suggesting that the luminance component of the display change initiated the saccadic inhibition, as was the case for the black transient image conditions in Experiments 4 and 5. The magnitude of inhibition in the mixture condition was also similar to that in the luminance condition ($t(9)=1.19, p=.266$), and was greater than in the isoluminance condition ($t(9)=4.99, p<.01$). In addition, the inhibition produced in the mixture condition was of greater duration than the inhibition in either the isoluminant ($t(9)=3.03, p<.05$) or luminance ($t(9)=2.26, p<.05$) stimulus conditions.
Figure 21. Saccade frequency histograms from Experiment 6. Saccadic inhibition is present in all condition, but is weaker and delayed when evoked by the isoluminant stimulus. Inhibition evoked by the mixed color and luminance stimulus has a rapid onset but is significantly prolonged compared to the luminance condition.

An important finding of this experiment is that saccadic inhibition was present in the isoluminant display condition. This does not prove that saccadic inhibition is not mediated through the superior colliculus, even though this structure has been shown not to receive color-opponent parvocellular input in monkeys. The superior colliculus does receive direct inputs from retinal M-cells, that have been shown to respond to abrupt isoluminant visual changes. Logothetis et al. (1990) showed that magnocellular neurons in the dLGN respond to abrupt transitions from red to green at isoluminance, even though these neurons do not receive color-opponent input. The response of these neurons at
isoluminance was about 40% of their response to non-isoluminant stimulation, which might explain the lower magnitude of the saccadic inhibition evoked at isoluminance.

It is also possible that saccadic inhibition was observed in the isoluminant condition because of slight mismatches from the isoluminant condition for each participant. It was shown in Experiment 3 that saccadic inhibition can be evoked with a robust magnitude of 0.59 by small stimuli with contrasts as low as 11%. This sensitivity to low luminance contrasts mean that true isoluminance may be difficult to achieve, especially given that neurons in the superior colliculus may respond optimally to slightly different colors (Marrocco & Li, 1977).

Table 8. Results of Experiment 6. Values in parenthesis are standard errors.

<table>
<thead>
<tr>
<th>Condition</th>
<th>( L_{\text{onset}} ) ms</th>
<th>Duration ms</th>
<th>( L_{\text{min}} ) ms</th>
<th>Magnitude proportion</th>
<th>Saccades total count</th>
<th>Baseline Saccadic Proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isoluminance</td>
<td>100.0 (2.5)</td>
<td>37.6 (2.6)</td>
<td>121.8 (2.6)</td>
<td>0.596 (0.039)</td>
<td>1429 (162)</td>
<td>0.138 (0.003)</td>
</tr>
<tr>
<td>Mixture</td>
<td>83.0 (1.6)</td>
<td>43.0 (2.1)</td>
<td>107.0 (1.6)</td>
<td>0.758 (0.035)</td>
<td>1438 (148)</td>
<td>0.145 (0.003)</td>
</tr>
<tr>
<td>Luminance</td>
<td>79.9 (1.8)</td>
<td>37.9 (1.7)</td>
<td>100.8 (1.8)</td>
<td>0.714 (0.048)</td>
<td>1405 (160)</td>
<td>0.144 (0.003)</td>
</tr>
</tbody>
</table>

In many of the previous experiments, the text was hidden during the display of the transient image. This might suggest an explanation for saccadic inhibition based on disruption of visual information processing during reading, caused by the transient loss of the text (McConkie et al., 1985; McConkie et al., 1992; but see Reingold & Stampe, in press). However, in the current experiment the text remained visible within the transient image, and saccadic inhibition was evoked by changing only the brightness or color of
the text background. This appears to be incompatible with a processing disruption model of saccadic inhibition, and instead suggests a fast reflexive mechanism sensitive to changes in the low level characteristics of visual input.

**Experiment 7**

Several different sizes of display changes were used in the previous experiments. Full-screen (30° by 22.5°) display changes were used in Experiment 1 and 2, and gaze contingent windows were used to present 1° (Experiment 3) and 6° (Experiments 4, 5, and 6) square display changes at the participant's point of gaze. Saccadic inhibition was present in all these experiments, suggesting that saccadic inhibition can be produced by a wide range of stimulus sizes. The effects of the size of the stimulus and of stimulation of the foveal, parafoveal, and peripheral regions of the visual field on the evoked saccadic inhibition will be investigated further in this experiment.

Such a study was performed by Brecelj and Prevec (1983) for the VEP response. Circular stimuli with a radius of 2.5° (stimulus area of 10 deg²) and 4° (area of 25 deg²), and rings with an outside radius of 16° and an inside radius of 4° (area of 325 deg²) or 8° (area of 300 deg²) were presented. Stimuli were centered on the fovea but only the half in the right visual hemifield was displayed, with the results reported here using reversing checkerboard patterns with 0.25° checks. The amplitude of the P1 component of the VEP was 5.3 μV for the 2.5° circular stimulus and 5.8 μV for the 4° circular stimulus, indicating that the response scaled nonlinearly with the stimulus area. It was also found that amplitude decreased for more peripheral stimulation, from 6.4 μV for the 4°-16° ring to 3.6 μV for the 8°-16° ring, despite the similar areas of these stimuli. It is clear that the
area of the stimulus alone did not determine the amplitude, because the 2.5°, 4°, and 4°-16° rings have very different areas yet produced similar amplitude VEP responses. Likewise, the two ring stimuli had similar areas yet produced very different VEP responses.

Meredith and Celesia (1982) performed a similar study using small stimuli at different distances from the center of the visual field. VEP responses were found to decrease rapidly with eccentricity: for example, a 2.3° square checkerboard stimulus that produced strong responses at the fovea produced no detectable response at eccentricities greater than 4° from the fovea. To obtain reliable VEP responses, stimulus size had to increase with eccentricity. Approximately equal response amplitudes were produced by a 0.22° stimulus at the fovea, a 3.3° stimulus at 8° eccentricity, and a 5.8° stimulus at 14° eccentricity. Meredith and Celesia showed that these stimuli were represented by equal areas of the visual striate cortex, by using the cortical magnification factor data of Cowey and Rolls (1974) and of Rovamo and Virsu (1979). Equal cortical area activated by a stimulus implies that equal numbers of cortical neurons contribute to the VEP response, supplying a physical link between the amplitude of the VEP response and cortical area.

The cortical magnification factor (CMF) is the relation of the size of a stimulus in the visual field and the size of its representation in visual striate cortex (Daniel & Witteridge, 1961). It is usually measured in millimeters of cortex per degree of visual angle. Several numerical estimates of the CMF as a function of retinal eccentricity have been published, using tabular data (Cowey & Rolls, 1974), third-order polynomial equations (Romano & Virsu, 1979), and logarithmic functions (Horton & Hoyt, 1991). These estimate agree well except for the central 1° of the visual field.
Five stimulus size conditions were used in this experiment. The display change covered the entire display, or was limited to a 1° or 6° square centered on the participant's fovea as in previous experiments. An 18° square gaze contingent window was also used that covered 50% of the area of the display, with the display change limited to the region inside the square (18° central condition) or outside the square (18° peripheral condition). This last condition did not stimulate the fovea, which is an important test of models of saccadic inhibition. All of these stimuli are symmetrical about the participant's point of gaze, in order to avoid any directional effects which might interfere with reading. These stimuli are shown in Figure 22, that also lists the proportion of the total display area and the estimated area of striate visual cortex activated by each stimulus.

Note that the 6° central and 18° peripheral conditions are matched for the cortical area activated by these stimuli, but differ by a factor of 10 in display area. Also, the 18° central and 18° peripheral conditions are matched in display area but differ by a factor of 3 in cortical area. Comparison of the magnitude of inhibition evoked by these conditions will be important in determining how saccadic inhibition scales with stimulus area.

Method

A black transient image (4.0 cd/m²) was used in this experiment, with the normal image consisting of text with an average luminance of 40 cd/m². A gaze contingent window was used to create all stimulus displays except in the full screen display change condition. A total of 125 pages of text were presented, with 25 pages randomly assigned to each condition.
Figure 22. Stimuli used in Experiment 7. Four sizes of stimuli are used that include the central part of the visual field, as well as a peripheral-only stimulus (bottom). The percentage of the display area and the estimated striate cortical area activated by each stimulus are given.
The CMF data can be used to estimate the cortical area activated by a given stimulus, using numerical integration as follows. Assuming isotropic magnification (i.e. equal resolution in horizontal and vertical directions), the area of striate visual cortex activated by a small stimulus is the area of that stimulus in the visual field, multiplied by the square of the CMF appropriate for the eccentricity of the stimulus from the fovea. The cortical area activated by a large stimulus can be estimated by breaking the stimulus into small patches, then summing the cortical area activated by small parts of the stimulus using the relationship stated above. This method was used to compute the estimates of cortical area for the stimuli in this experiment. The stimuli were divided into 0.1° squares, and the cortical area of each computed using the average of the CMFs for the four quadrants of the visual field derived by Romano and Virsu (1979).

Results and Discussion

An average of 1490 saccades were analyzed from each participant in each condition. Inhibition magnitude and latencies for each condition are reported in Table 9, and the saccadic frequency plots in Figure 23 show the time course of inhibition produced by each type of stimulus. Magnitude of inhibition showed an increasing trend as stimulus area increased progressively with the 1°, 6°, 18° central conditions and the full-screen condition, but the effect was only marginally significant (F(3,27)=2.70, p=.065). Pairwise t-tests between the 1°, 6°, and 18° central conditions did not approach significance (p>.10 for all), but the full-screen condition did differ significantly from the 1° central condition (t(9)=2.30, p<.05) and the 6° central condition (t(9)=2.70, p<.05).
Figure 23. Saccadic frequency histograms from Experiment 7. Left: comparisons of small area and full-screen display changes. Inhibition is somewhat weaker for both 1° and 6° conditions, and inhibition is slower in onset. Right: comparison of large area display changes. No significant changes in strength of inhibition are seen.

Inhibition was clearly evoked by the peripheral 18° condition, although no part of the visual field within 9° of the fovea was stimulated. Indeed, the magnitude of the inhibition produced in this condition did not differ from that in the full-screen or 18° central conditions (t(9)<1 for both). Inhibition in the 18° peripheral condition was significantly stronger than in the 6° central (t(9)=2.65, p<.05) and the 1° central (t(9)=3.16, p<.05).
Table 9. Results of Experiment 7. Values in parenthesis are standard errors.

<table>
<thead>
<tr>
<th>Condition</th>
<th>$L_{\text{onset}}$</th>
<th>Duration</th>
<th>$L_{\text{min}}$</th>
<th>Magnitude</th>
<th>Saccades</th>
<th>Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(ms)</td>
<td>(ms)</td>
<td>(ms)</td>
<td>proportion</td>
<td>total count</td>
<td>Saccadic Proportion</td>
</tr>
<tr>
<td>1° Central</td>
<td>86.2 (1.5)</td>
<td>32.6 (1.9)</td>
<td>103.8 (1.6)</td>
<td>0.576 (0.038)</td>
<td>1490 (134)</td>
<td>0.147 (0.003)</td>
</tr>
<tr>
<td>6° Central</td>
<td>74.7 (2.1)</td>
<td>52.9 (2.1)</td>
<td>101.0 (1.9)</td>
<td>0.613 (0.042)</td>
<td>1482 (133)</td>
<td>0.156 (0.005)</td>
</tr>
<tr>
<td>18° Central</td>
<td>73.0 (1.7)</td>
<td>51.5 (2.4)</td>
<td>98.4 (2.6)</td>
<td>0.650 (0.051)</td>
<td>1514 (129)</td>
<td>0.152 (0.004)</td>
</tr>
<tr>
<td>Full-screen</td>
<td>71.0 (1.8)</td>
<td>50.5 (2.3)</td>
<td>94.4 (2.1)</td>
<td>0.682 (0.027)</td>
<td>1453 (134)</td>
<td>0.157 (0.003)</td>
</tr>
<tr>
<td>18° Peripheral</td>
<td>72.0 (2.5)</td>
<td>42.4 (2.6)</td>
<td>94.4 (3.2)</td>
<td>0.684 (0.028)</td>
<td>1518 (141)</td>
<td>0.152 (0.002)</td>
</tr>
</tbody>
</table>

It can be concluded that the magnitude of saccadic inhibition is only weakly dependent on the size of the stimulus, at least when the stimulus is centered on the fovea. More importantly, magnitude of saccadic inhibition does not appear to scale with the cortical area activated by the stimulus, as was the case for VEP. Specifically, there was a significant difference in magnitude between the central 6° and peripheral 18° condition, which activate similar areas of striate cortex. Also, no difference was found between the 18° central and 18° peripheral conditions ($t(9)<1$), which were matched in display area but differed in cortical area by a factor of 3. In fact, the only significant differences in magnitude was found between the peripheral 18° and full-screen conditions on one hand and the 1° and 6° central conditions on the other, that differ in stimulus area by at least a factor of 10. This might mean that magnitude of inhibition is reduced only when small stimuli are used.

Significant differences in $L_{\text{onset}}$ were found between conditions ($F(4,36)=13.08$, p<.001). Only the 1° central condition was significantly different from the 6° central ($t(9)=5.96$, p<.001), 18° central ($t(9)=7.46$, p<.001), full-screen ($t(9)=6.62$, p<.001) and
peripheral 18° (t(9)=4.45, p<.01) conditions, with no other comparisons reaching significance (p>.17 for all). Thus there is a trend for the inhibition evoked by small stimuli to be of longer latency.

There was also a significant effect of stimulus size on the duration of inhibition (F(4,36)=23.39, p<.001). Inhibition was significantly less sustained in the 1° central condition compared to all other conditions: 6° central (t(9)=9.02, p<.001), 18° central (t(9)=10.20, p<.001), full-screen (t(9)=6.77, p<.001) and peripheral 18° (t(9)=3.40, p<.01). Inhibition for the peripheral 18° condition is also less sustained than in all other conditions excepting the central 1° condition: central 6° (t(9)=4.99, p<.01), central 18° (t(9)=3.12, p<.05), and full-screen (t(9)=4.02, p<.01). Duration did not differ between any other conditions (t(9)<1 for all). The increased latency for the 1° central condition may indicate that inhibition caused by foveal-only changes is a special case, or may simply be due to its small area. The shorter duration of inhibition for the 18° peripheral stimulus might be related to the higher temporal responsiveness of the peripheral visual field (Seiple, Greenstein, Holopigian & Carr, 1988).

The results of this experiment suggest that the magnitude of saccadic inhibition scales weakly with stimulus size, with magnitude increasing by only 13% when stimulus size increased from 1° to 18°. In contrast, the effect of stimulus size on VEP amplitude is very strong: Michael and Halliday (1971) reported that a 32° stimulus produced twice the VEP amplitude as an 8° stimulus. Stimulation limited to the fovea in the 1° central condition evoked saccadic inhibition, as did stimulation limited to regions at greater than 9° from the fovea in the 18° peripheral condition. Therefore changes in either the foveal or peripheral visual fields can produce saccadic inhibition. None of the measures of
latency and magnitude for the 18° peripheral condition differed significantly from those in either the full-screen or the central 18° condition, that stimulate both the peripheral visual and central visual field. In contrast, peripheral stimulation is much less effective than foveal and parafoveal stimulation in VEP studies. For example, Brecelj and Prevec (1983) observed that the VEP response to ring-shaped peripheral stimuli was much weaker than that seen for smaller central stimuli.
General Discussion

The experiments presented in this thesis investigated the effect of stimulus characteristics on the strength and time course of visually evoked saccadic inhibition. These were compared to the known effects of similar stimulus characteristics on visually evoked potentials (VEPs). In this discussion, the findings of these experiments will be summarized and discussed in terms of the similarities and differences in the neurological basis of VEP and saccadic inhibition. The stability of the measures of saccadic inhibition introduced in this thesis will also be analyzed, as will the advantages and limitations of the reading task used to study it. The implications of these findings for the hypothesis that the superior colliculus (SC) is the site of saccadic inhibition will be noted, and known neural mechanisms in the SC that might produce the stimulus effects found here will be discussed.

Empirical Findings

In Experiment 1, saccadic inhibition was found to be evoked by a visual display change, but not by an audio tone. This is similar to the findings of Ross and Ross (1981), who found that saccade reaction times (SRT) was increased by visual but not by auditory delayed warning signals.

Both the magnitude and latency of saccadic inhibition were found to be influenced by stimulus saliency. This was found for both for transient displacement (Experiment 2) and luminance and contrast changes (Experiment 3). For both types of stimuli, the magnitude of inhibition increased and the latency of inhibition decreased for stimuli with
higher saliency. This is similar to the results of VEP studies using contrast (Kubova et al., 1995) and luminance (Osaka & Yamamoto, 1978) stimulus manipulations. The latency of saccadic inhibition evoked by transient displacements decreased with increasing displacement size, whereas no latency change was observed in VEP studies using step-displacement stimuli (Fenwick & Turner, 1977; Spekreijse et al., 1985). The congruent effects of contrast and the incongruent effects of displacement size on the latencies of VEP and saccadic inhibition may be the result of the level of the visual system at which these effects arise. Changes in the latency of visual response in the dLGN have been observed for manipulations of contrast (Sestokas & Lehmkuhle, 1986), whereas no latency effects have been demonstrated for step displacement. The latency of early VEP peaks largely reflects these subcortical delays, whereas an additional effect unique to saccadic inhibition may be responsible for the decrease in latency observed for larger displacement sizes.

The latency of visually evoked saccadic inhibition was also found to be affected by the spatial frequency content of the stimulus in much the same way as has been found in VEP studies (e.g., Parker & Salzen, 1977; Vassilev & Stomonyakov, 1987). Transient display of low-frequency gratings evoked much faster inhibition than was produced by high-frequency gratings (Experiment 4). The latency of inhibition was determined by the change in spatial frequency content of the display (Experiment 5), and not by the frequency content of the transient image alone. Indeed, simply replacing text with a luminance-matched blanked display evoked inhibition that was similar to that caused by a high-frequency grating. The source of the effect of stimulus frequency content on latency
may be subcortical, as a similar effect has been observed for neural responses at the dLGN in the cat (Sestokas & Lehmkuhle, 1986).

The latency of the onset of saccadic inhibition ($L_{\text{onset}}$) was also found to be extremely rapid, with the fastest inhibition (latencies of ~68 ms) evoked by the large change in display luminance caused by full-screen black transient images. The inhibition evoked by stimuli that included luminance changes was consistently faster than observed for all other types of stimulus: 10.4 ms faster than displacement, 2.6 ms faster than a low-frequency grating, 14.6 ms faster than a high-frequency grating, and 20.1 ms faster than an isoluminant color change. When present, luminance changes also appeared to suppress the effects of other components of the display change in determining the time course of the evoked saccadic inhibition, perhaps by initiating saccadic inhibition before other stimulus components. Interestingly, the presence of a color change in addition to a luminance change may have acted to extend the duration of saccadic inhibition (Experiment 6), although no such effect was observed when high or low frequency changes were present (Experiment 5). No similar VEP studies were found for comparison with this phenomenon.

The effect of the size and region of the visual field of the stimulus differed between VEP and saccadic inhibition. For VEP, small stimuli at the fovea were found to be most effective, the amplitude of the VEP response increased with stimulus size, and stimulation of the peripheral visual field alone produced much weaker responses (Brecelj & Prevec, 1983). In contrast, saccadic inhibition evoked by similar stimulus manipulations showed only small variations in magnitude (Experiment 7). In particular, both full-screen and peripheral stimuli evoked saccadic inhibition of similar magnitude
and latency. The only significant differences were observed for the smallest stimulus sizes, that evoked somewhat weaker and delayed inhibition. The overall pattern of results was incompatible with scaling of response by either stimulus area, or with scaling by the area of striate visual cortex activated by the stimulus. In contrast, the scaling of response for VEP has been shown to be compatible with the area of cortex activated by the stimulus.

The overall pattern of similarities and differences between the effects of stimulus characteristics on VEP and visually evoked saccadic inhibition appears to suggest that both share latency and amplitude effects that are produced in subcortical visual pathways, specifically the optic nerve and retina. These effects include latency effects of contrast, luminance, spatial frequency content, and color. It is also possible that the nonlinear relation between stimulus contrast and VEP amplitude or magnitude of saccadic inhibition arises in this part of the visual system. In contrast, the effects of stimulus size on latency and of displacement size on the magnitude of saccadic inhibition differ sharply from those observed for VEP. This suggests that these effects arise separately at the neural locus of these two phenomena. VEP is known to arise from cortical sources, and this is congruent with the scaling of VEP response with striate visual cortical area. In the case of visually evoked saccadic inhibition, the lack of cortical scaling and its extremely low latency argue for a subcortical locus. In the absence of specific neurological findings, the most likely candidate for the locus of saccadic inhibition is the superior colliculus, that receives direct retinal input, projects directly to the saccade burst generator in the brainstem, and appears to be part of the pathway by which saccades of both visual and cortical origin are generated.
Finally, inhibition was evoked even when the text being read remained visible during the display of the transient image. In Experiment 6, a change in color or luminance of the text background evoked saccadic inhibition. Inhibition was also evoked by the peripheral 18° condition of Experiment 7, in which the display change was always 9° from the participant's point of gaze. Therefore a disturbance of the text being read was not required in order to evoke saccadic inhibition, contrary to the processing disruption model used to explain artifacts produced by saccadic inhibition in a study using gaze-contingent text masking (McConkie et al., 1985; McConkie et al., 1992; but see Reingold & Stampe, in press).

**Measures of Saccadic Inhibition**

Four measures of the strength and time course of saccadic inhibition were used in the experiments in this thesis. These were derived from a histogram of saccadic frequency by time after each display change, using narrow (4 ms) histogram bins and computational techniques that resulted in high temporal resolution. A fifth measure, baseline saccadic proportion, was introduced to monitor the variability of the baseline saccadic frequency, which is used in the computation of the magnitude of saccadic inhibition. Table 10 summarizes the range of means observed for these measures across all conditions of all experiments in this thesis. In addition, the standard deviation across conditions is reported.
Table 10. Range and standard deviations of measures of saccadic inhibition observed in the experiments.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Minimum</th>
<th>Maximum</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>$L_{onset}$</td>
<td>68.2 (expt 1)</td>
<td>101.1 (expt 5)</td>
<td>6.00</td>
</tr>
<tr>
<td>$L_{min}$</td>
<td>93.0 (expt 2)</td>
<td>126.0 (expt 5)</td>
<td>6.55</td>
</tr>
<tr>
<td>Duration</td>
<td>32.4 (expt 3)</td>
<td>52.9 (expt 7)</td>
<td>7.49</td>
</tr>
<tr>
<td>Magnitude</td>
<td>0.380 (expt 2)</td>
<td>0.800 (expt 4)</td>
<td>0.11</td>
</tr>
<tr>
<td>Baseline Saccadic Proportion</td>
<td>0.138 (expt 6)</td>
<td>0.157 (expt 7)</td>
<td>0.10</td>
</tr>
</tbody>
</table>

An inspection of Table 10 indicates that the experimental manipulations of stimulus characteristics caused substantial variation in all measures of saccadic inhibition. In contrast, the baseline saccadic proportion measure changed little between experiments, conditions, and participants, validating its use in computation of the magnitude measure. The standard deviations of the latency measures were remarkably small, indicating that the time course of saccadic inhibition was very reproducible between participants. In comparison, the standard deviation of the magnitude measure indicated greater variability between participants.

**Suitability of Reading Task**

In the studies of saccadic inhibition reported in this thesis, participants generated saccades by reading a short story. This task was very efficient in generating saccades,
and less than 10 minutes of reading were required for each experimental condition in order to collect the ~1500 saccades used in analysis. Participants had no difficulty in ignoring the flickering of the display caused by the transient image.

![Saccade Amplitude Distribution](image)

**Figure 24: Saccade amplitude distribution for reading.** This data was compiled from all conditions of all experiments in this thesis.

The drawback of the reading task is that a wide range of saccade amplitudes are generated. A very broad range of saccade amplitudes were present, as shown by the histogram in Figure 24. Data for this figure was compiled from all conditions of all experiments. There are two peaks of saccade amplitudes, with the peak at ~16° amplitude consisting of right-to-left return sweep saccades between the end on one line and the start of the next, whereas the majority of the saccades that made up the peak at ~4° amplitude were left-to-right saccades made during actual reading. It was not possible
to test if the strength or timing of saccadic inhibition were dependent on saccadic amplitude. It may be argued, however, that saccadic inhibition must have been present at a reasonable magnitude at all saccade amplitudes. If this were not the case, the observed magnitude of inhibition would have been reduced by the saccades for which inhibition was not present. The observed magnitudes of inhibition were too large (e.g., 0.687 for a 1° foveal black display change in Experiment 3 and 0.791 for a full-screen black display change in Experiment 1) to permit any range of saccade amplitudes from contributing to the effect. As reading cannot be used to investigate the possible interaction between saccade amplitude and visually evoked saccadic inhibition, a different task will have to be used. Such a task might involve more traditional saccadic paradigms, in which participants make saccades in response to the visual onset of targets, in combination with display changes similar to those used in the current series of experiments.

**Neurological Implications**

As noted earlier, the superior colliculus is the most probable locus of visually evoked saccadic inhibition. The implications of findings from the experiments presented here will be discussed in terms of their consistency with this hypothesis. In addition, the possible role of known neural mechanisms in the SC in producing the stimulus effects found here will be discussed.

Probably the strongest argument in favor of the SC as the site of saccadic inhibition is the speed of this phenomenon. The frequency of saccades is reduced by nearly half in as little as 68.2 ms ($L_{\text{onset}}$, Experiment 1) after the display of a full-screen black transient image. The time at which the drop in saccade production becomes significant can also be
computed from the composite histograms generated for each experiment. The threshold of significance was computed from the mean and standard deviation of the saccadic frequency during the 48-ms baseline period. Saccadic frequency was reduced by 3 standard deviations from the baseline at a latency of 64.8 ms after the display change for the full-screen black display change condition of Experiment 1. When applied to similar stimuli in other experiments, this same test yielded latencies of 67.6 ms for Experiment 2 and 67.2 ms for Experiment 7. The longer latencies of found for Experiments 2 and 7 may be due to the lower number of saccades and thus greater noise in the histograms from these experiments.

A possible model of saccadic inhibition based on the neurophysiology of the intermediate superior colliculus was discussed earlier in this thesis. In this model, changes in visual input cause activity in pandirectional cells in the superficial SC, which in turn stimulate neurons in the intermediate SC. These then act through the extensive network of inhibitory interneurons in the intermediate SC to reduce presaccadic activity in other buildup neurons (SCBUNs), which delays saccades. As saccadic inhibition was evoked by both foveal and peripheral stimuli (Experiment 7), both buildup and fixation cells in the intermediate SC would have to be involved in this inhibition. The latency at which saccades could be delayed by strong visual input was computed from measured latencies in the oculomotor system and found to be ~65 ms, which is in good agreement with the findings in this thesis.

Many of the stimulus-related effects observed here, including contrast or luminance, and spatial frequency, have already been explained in terms of delays in the retina and optic nerve. These delays occur before the branching of the axons of retinal M ganglion
cells to the SC and dLGN, and therefore should contribute to the latency of saccadic inhibition. Other effects may be explained by characteristics of the visual pandirectional cells, or by actions in the intermediate SC. For example, pandirectional cells respond well to stimuli over a wide range of contrasts (Marrocco & Li, 1977), which matches the rather weak modulation of saccadic inhibition by contrast. No data is available for the behavior of pandirectional cell in response to a transient displacement stimulus similar to that used in Experiment 3, but it is possible that the output would scale with the amount of change in the display, as is seen for VEP. In this case, the strong effect of displacement size on the magnitude of saccadic inhibition would be because of the increasing response of pandirectional cells to larger displacements.

The finding in Experiment 6 that isoluminant stimuli can evoke saccadic inhibition may appear to be evidence against the superior colliculus as the locus of saccadic inhibition. As discussed in Experiment 6, the superior colliculus does not appear to receive input from color-opponent ganglion cells in the retina, and only weak responses to moving isoluminant color edges have been observed in the SC (Marrocco & Li, 1977). However, the responsiveness of the magnocellular system to abrupt isoluminant visual changes (Logothetis et al., 1990) may provide a mechanism for saccadic inhibition to be evoked by such stimuli. In any case, it is difficult to exclude slight mismatches from the isoluminant condition as a cause for the observed saccadic inhibition, as Experiment 3 showed that robust saccadic inhibition can be evoked by rather weak luminance changes. Consequently, the finding of weakened and delayed inhibition in the isoluminant stimulus condition of Experiment 6 is inconclusive with regard to the possible role of the superior colliculus in visually evoked saccadic inhibition.
The scaling of the magnitude of saccadic inhibition with stimulus size was observed in Experiment 7 to be rather weak and clearly incompatible with the cortical area scaling that fits the results of VEP amplitude for similar stimuli (Brecelj & Prevec, 1983). This is surprising, given that visual stimuli map to the surface of the superior colliculus with a magnification function similar to that of striate visual cortex as shown in Figure 4 is (Ottes et al., 1986; Optican, 1995). However, the relation between the size of the visual stimulus and neural activity in the SC is complicated by the response characteristics of the pandirectional cells in the superficial SC. The receptive fields of these cells are very large, and may cover more than 20% of the area of the SC (McIlwain, 1975, 1986). In addition, the response of pandirectional cells increases only gradually with stimulus size, peaking at each neuron’s optimal stimulus size. The response gradually decreases for larger stimuli, although about 20% of pandirectional cells remain active for even the largest stimuli (Goldberg & Wurtz, 1972). All of these characteristics may act to reduce the effects of stimulus size and of the magnification of the foveal part of the visual field in the SC. For example, Moors and Vendrik (1979) found increased latency and decreased response for pandirectional cells when stimuli smaller than the optimal size for that cell were presented. This is in agreement with the findings of Experiment 7, in which the only significant changes were a decrease in magnitude and increase in latency of saccadic inhibition for the smallest sized stimuli.

Summary

The reviews, experiments, and discussions in this thesis have helped to elucidate the nature and characteristics of visually evoked saccadic inhibition. This phenomenon
appears to be a fast reflex of the oculomotor system, which acts in response to changes in visual input to suppress the production of saccades. The visual changes do not need to be relevant to the task being performed: for example, changing only the brightness or color of the background of text will evoke saccadic inhibition during reading. The exact characteristics of the visual change may influence the latency or magnitude of the saccadic inhibition, but these effects appear to be due to subcortical characteristics of the visual system. The locus of saccadic inhibition may be the superior colliculus, due to the extremely low latency between visual changes and the resulting reduction in saccade frequency.

What role could visually evoked saccadic inhibition play in everyday visual perception and oculomotor behavior? Visual perception is momentarily disrupted during saccades, during which the brain is unable to respond to new visual input. Saccadic inhibition may serve to give the brain time to process the arrival of changes in visual input, by delaying the execution of saccades. If the new visual input should prove irrelevant, the saccade can continue within ~70 ms, or, if the new input requires a change in behavior, the saccade might be cancelled or a different saccadic target chosen.
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