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Technical Report
AFOSR # 89-0437

Multimodal Interactions
in Sensory-Motor Processing

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Abstract

We describe our progress in a) delineating the functional architecture of the human saccadic and attentional orienting systems (section 2) based on analyses of reaction times; b) development of accurate surface maps of the human neocortex *in vivo* from reconstructions of MR scans (section 3). Work carried out under AFOSR funding (2 in 90-91 year) provides the basis for our current model, which identifies two serially organized component processes in saccade generation (section 1.1). The early component is sensory; it's most noteworthy feature being the mode of convergence of visual and auditory information in the saccadic control system (section 1.2). In the subsequent pre-motor component, the processing time is partially determined by the state of fixation. Fixation point offsets facilitate saccade latencies by decreasing premotor processing times via disinhibition. These sensory and motor facilitatory mechanisms can be combined to optimize human saccadic performance (section 1.1). The ultimate goal is to provide a model which accounts for human oculomotor performance in terms of physiologically plausible component subprocesses.



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From the vast array of data available to our senses we can select information for detailed processing by directing our gaze or our attention to particular locations in the environment. The current project investigates the sensory and motor components of oculomotor control and attentional orienting in an effort to describe their anatomical bases and functional architecture, and to specify the conditions that will maximize the efficiency of human orienting.

The first section of this report includes three manuscripts describing our investigations of the effects of auditory and visual events on sensori-motor processes. In section 1.1 (Nozawa et al.) we present our current model of the component processes involved in saccade generation based on evidence from our other investigations funded under the present AFOSR grant (Reuter-Lorenz et al., 1991; Fendrich et al, section 1.3; Hughes et al., section 1.2). In this two-stage serial model of saccade generation, parallel auditory and visual inputs converge on a sensory processing stage to produce neural summation. A subsequent premotor programming stage is modulated by the observers state of fixation (i.e. fixation release). The model is tested within the additive factors framework by comparing the effects of unimodal and bimodal targets (i.e. combined visual and auditory stimuli) in the context of the fixation point offset paradigm. The data reported by Nozawa et al. provide new support for separable sensory and motor facilitatory components and indicate that these mechanisms can combine to optimize saccadic performance. Differences observed between the combined effects of fixation release and auditory versus visual signals, suggest that the relative timing of the sensory signal and fixation release may effect the magnitude of facilitation produced by fixation point offsets. We are currently investigating this possibility.

In section 1.2, Hughes et al., (submitted) demonstrate that the response facilitation produced by bimodal targets is more robust for saccades than for directed manual or simple manual responses. Only saccadic latencies show a benefit from bimodal targets exceeding that predicted by probability summation, a pattern which is consistent with the evidence for

auditory and visual convergence within the primate superior colliculus. In section 1.3, Fendrich et al. (in press) demonstrate that short latency saccades, referred to as express saccades, can be elicited by acoustic, as well as visual targets. These findings, together with our earlier work on express saccades (Reuter-Lorenz et al, 1991) provides the basis for the model presented in section 1.1, in which the inhibitory effect of fixating influences premotor rather than sensory components of saccadic initiation.

A central issue in understanding the functional architecture of selective orienting involves specifying the relationship between attention and saccadic eye movements. In section II of this report, Reuter-Lorenz & Fendrich (submitted) evaluate the hypothesis that saccadic motor programs are the basis for attentional orienting. This study provides evidence that directed attention and saccades may be controlled by separate mechanisms when orienting is elicited by a sudden peripheral event (an eccentric visual cue). However, when orienting is directed by a symbolic cue, attention and saccades appear to be more tightly coupled. A framework is proposed in which collicular components of the oculomotor system play a greater role in saccadic than attentional control in the case of peripheral cues, whereas a cortical network shared by both systems underlies orienting to central cues.

The third and final section includes our recent manuscript describing results from our new computerized method of determining regional cortical surface areas from 3D reconstructions of MR scans of the human brain. Comparisons of cortical surface areas in the left and right hemisphere in monozygotic twins and unrelated controls suggest that the left hemisphere may be far more genetically specified than the right hemisphere. The results are consistent with the view that high correlations between monozygotic twins on psychological and physiological variables may be related to reduced variance seen in major cortical structures, particularly in the left hemisphere.

SECTION 1.1

**Component Processes in Saccade Generation:
Intersensory Facilitation and Release from Fixation**

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Abstract

Previous work from our lab suggests that separable sensory and motor mechanisms may increase the efficiency of saccadic eye movements. To account for these results we present a two-stage serial model of saccade generation in which parallel auditory and visual inputs converge on a sensory processing stage to produce neural summation. A subsequent premotor programming stage is modulated by the observers state of fixation (i.e. fixation release). This model was tested within the additive factors framework by comparing the effects of unimodal and bimodal targets (i.e. combined visual and auditory stimuli) in the context of the fixation point offset paradigm. The data provides new support for separate sensory and motor facilitatory components and indicate that these mechanisms can combine to maximize the efficiency of saccadic responses. Differences between the combined effects of fixation release and auditory versus visual signals, suggests that the relative timing of the sensory signal and fixation release may effect the magnitude of facilitation produced by fixation point offsets.

Humans tend to explore their surroundings primarily through the visual modality. Thus, the speed of saccadic eye movements is an important determinant of the rate of information processing. Saccadic latencies can be influenced by the characteristics of the saccade target as well as the observer's state of fixation. As with other response systems, saccadic reaction times (SRTs) decrease monotonically with increasing target intensity (Wheless, Cohen & Boynton, 1967, Hughes & Kelsey, 1984). Saccadic RTs can also be reduced by as much as 70 msec simply by extinguishing the fixation stimulus prior to target onset (often called the "gap effect", Fischer & Ramsperger, 1984; Saslow, 1967).

The magnitude of the gap effect does not appear to vary with the luminance of the target (Reuter-Lorenz, Hughes & Fendrich, 1991), suggesting that target luminance and fixation point offsets influence different processes in saccade generation. Since fixation point offsets do not facilitate manual response latencies, the gap effect appears to influence sub-processes that are specific to saccade generation (Reuter-Lorenz et al., 1991). Thresholds for saccades elicited by electrical stimulation are reduced following fixation point offsets (Goldberg, Bushnell & Bruce, 1986). The gap effect may, therefore, reflect a disinhibition of saccadic responses which results from removal of the fixation stimulus. We refer to this facilitatory effect of fixation point offsets as "fixation release".

The additivity between target luminance and fixation point offsets led Reuter-Lorenz et al. (1991) to suggest a serial-stage model in which fixation release facilitates a premotor stage of saccade generation, whereas the effects of target luminance are confined to an earlier sensory stage of processing. Reuter-Lorenz et al. (1991) point out that these functional processing stages could be implemented within the superior colliculus (SC), a suggestion consistent with recent evidence that the gap effect depends on the integrity of the SC (Schiller, True & Conway, 1980). In addition, Fendrich, Hughes & Reuter-Lorenz (in press) have shown that fixation point offsets facilitate saccades to acoustic as well as

visual targets. Since the deeper layers of the SC receive converging auditory and visual afferents (Jay & Sparks, 1987; Meredith & Stein, 1983), the data from Fendrich et al. is consistent with the proposal that fixation point offsets facilitate saccadic processing within the SC (Reuter-Lorenz et al., 1991).

The recent evidence (Jay & Sparks, 1987; Meredith & Stein, 1983) that visual and auditory inputs convergence onto common neurons in the SC suggests that information conveyed within these modalities might be summed prior to initiating saccades; an architecture which would facilitate sensory components of pre-saccadic processing. Hughes, Reuter-Lorenz, Nozawa & Fendrich (1991) have demonstrated that combined visual and auditory targets reduce saccadic latencies beyond levels predicted by the statistical advantage conferred by having two targets rather than one. Furthermore, this bimodal target advantage was robust only for saccades; neither directed-manual nor simple-manual responses were faster than probability summation. The bimodal facilitation observed for saccadic responses provides evidence for neural summation of the two sensory inputs and may be a behavioral correlate of the auditory-visual convergence demonstrated in the SC.

These findings suggest separable sensory and motor mechanisms that serve to optimize the speed of saccadic responses. Distinguishing between "sensory" and "motor" processes in the colliculus is difficult because many collicular neurons have both sensory and motor response properties. However, functional distinctions between sensory and motor components of saccade generation may be revealed by an additive factors analysis of SRT (e.g., Sternberg, 1969; Townsend & Ashby, 1983). Auditory-visual convergence may enhance sensory processing via neural summation while fixation release might facilitate a premotor stage of processing. If, as suggested by Reuter-Lorenz et al. (1991), these component processes are serially organized, then the combination of neural summation and fixation release should produce additive effects on saccadic latencies.

Moreover, the combination of these factors should maximize the efficiency of human performance. The present experiment was designed to address these issues.

Figure 1 about here

Figure 1 presents a two-stage model representing our working hypothesis. The auditory and visual channels have parallel inputs to a summation stage which then sends its output to a premotor stage. The time course of the premotor stage is influenced by a fixation signal. Active fixation provides an inhibitory input to the pre-motor processor. This inhibition is reduced upon removal of the fixation point. Under the assumptions that 1). the auditory and visual channels are stochastically independent, and 2) selective influence holds, i.e., auditory inputs do not affect visual processing and visual inputs do not affect auditory processing, parallel processing within these channels will produce an intensity-dependent interaction (see Townsend and Ashby [1983] for a derivation). Nozawa & Townsend (1991) have proved that, in a parallel processing system, processes which operate on either the minimum completion times of the parallel channels or neural summation between these channels will show a particular form of interaction with target intensity termed "superadditivity". Thus, superadditivity is a signature of parallel processing in the models currently under consideration. Once superadditivity is obtained, additional analyses can determine whether the parallel inputs are summed, as suggested in Figure 1 (see Hughes et al., 1991). Therefore, the model predicts that variations in the intensity of auditory and visual components of bimodal signals should produce a superadditive interaction on saccade latency. However, since fixation offsets influence a subsequent stage, the effect of fixation condition should be additive with the effects of unimodal target intensity and with the neural summation effects produced by bimodal targets.

In order to test these predictions, the present experiment combined the fixation point offset paradigm with an expanded version of the bimodal target paradigm used by Hughes et al. (1991). Auditory and visual targets of two different intensities were presented either unimodally or bimodally. The unimodal targets were factorially combined yielding four bimodal target conditions (low visual-low auditory; low visual-high auditory; high visual-low auditory; high visual-high auditory). These data provide a basis for a rigorous evaluation of the two-stage serial model.

Methods

A centrally located green light-emitting diode (LED) provided a fixation point. Red LEDs served as the visual targets. The acoustic targets consisted of brief (100 ms) white noise bursts delivered through 4 cm. speakers located directly beneath the target LEDs. Target eccentricity was 20°. The entire apparatus was housed in a large (1.54 m. x 1.54 m. x 0.9 m.) enclosure lined with a sound-absorbing foam material (Sonex™). Eye position was digitized at 250 Hz using a scleral infra-red reflection device. Data collection followed 5 min. of dark adaptation, during which time the eye tracker was calibrated. Head movements were minimized using a bite-plate. All subjects were emmetropic and had normal hearing. Saccades were detected using a velocity criterion (generally 50 deg. sec⁻¹). Data analysis was performed off-line.

As the magnitude of summation effects should depend upon central simultaneity of the two inputs (Stein et al., 1989; Miller, 1986), preliminary testing identified a high and a low stimulus intensity that produced equivalent auditory and visual response latencies in each subject. Each observer then participated in 4 experimental sessions. Five trial blocks consisting of 80 trials each were run per session. Auditory, visual and bimodal targets were presented with equal frequency and randomized with respect to both order and location. On half of the trials, the fixation stimulus was extinguished 200 msec prior to the onset of the target (gap condition); the fixation stimulus remained on in the other half

(overlap condition). Acoustic warning signals (1000 Hz, 100 ms duration) were presented through a center speaker 300 ms before target onset. Thus, the offset of the warning signal was synchronous with the offset of the fixation point in gap trials. Twenty percent of the trials were catch trials, in which no saccade target was presented. Catch trials were evenly distributed between the gap and overlap conditions. The data reported below are based on at least 50 observations for each stimulus condition six naive observers.

Results

A variety of analyses were performed on these data. First, we report the results of Analyses of Variance (ANOVAs) on the individual subject means for each experimental condition. These ANOVAs were performed in three parts: the unimodal visual target X fixation condition, the unimodal auditory target X fixation condition, and the bimodal target X fixation condition.

The visual ANOVA suggests strong effects of both target intensity and fixation condition ($F(1,5) = 18.18$; $p < .01$; $F(1,5) = 55.87$; $p < .01$, respectively). However, the interaction between target intensity and fixation condition was not significant ($F(1,5) = 0.19$; n.s.). These data replicate the results of Reuter-Lorenz et al. (1991), and are represented in Figure 2. This confirmation of the additivity between target luminance and fixation offset effects support the serial stage model.

Figures 2 & 3 about here

The auditory ANOVA also reveals strong target intensity and fixation effects ($F(1,5) = 22.6$; $p < .01$; $F(1,5) = 12.28$; $p < .01$, respectively). These results confirm our previous finding that fixation point offset facilitate saccades to acoustic as well as visual targets. The present analysis also reveals an interaction between auditory target intensity and fixation

up until the time at which fixation release is complete. Since the time course of sensory processing is known to vary with signal intensity (e.g. Miller & Glickstein, 1967), it is also possible that as signal intensity increases so does the probability that, for a given gap duration, fixation release is not yet complete. This could reduce the gap effect for higher intensity signals.

Yet the present data show evidence of this only for the auditory modality. Since saccadic response latencies provide only a crude estimate of central arrival times, the possibility remains that acoustic signals arrive at the critical locus earlier than visual signals even when the overall RTs to these signals are matched. It follows that there should also be a gap duration at which the magnitude of the gap effect also varies with target luminance. We are currently testing this idea by systematically varying gap duration together with auditory and visual signal intensity.

condition ($F(1,5)=7.59$; $p<.05$). This interaction is not consistent with a simple serial stage model of auditory processing and fixation release, and therefore requires some modification of the model to accommodate auditory processing in the oculomotor system. These data are presented in Figure 3.

The results of the bimodal target X fixation condition ANOVA confirms significant effects of both visual and auditory intensity ($F(1,5)=9.9$; $p<.03$; $F(1,5)=10.17$; $p<.03$, respectively). Moreover, the interaction between visual and auditory intensity was significant ($F(1,5)=5.85$; $p<.06$) and superadditive. As shown by Nozawa and Townsend (1991), this superadditive interaction is the signature of a parallel process. This analysis is therefore consistent with the suggestion that the auditory and visual inputs are processed in parallel (also see Hughes et al., 1991). The gap effect was also observed ($F(1,5)=17.11$; $p<.01$). As the serial stage model of visual processing and fixation release predicts, the interaction of the gap effect and the visual intensity was not reliable ($F(1,5)=0.34$; n.s.). The interaction of the gap and the auditory intensity was found to be significant ($F(1,5)=6.23$; $p<.06$) but the triple interaction between auditory intensity, visual intensity and gap condition was not obtained ($F(1,5)=3.07$; $p>.13$). Although the failure of this triple interaction between bimodal intensity effects and fixation release is consistent with a serial stage model of bimodal convergence followed by pre-motor facilitation, the auditory X fixation interaction is problematic for this simple processing architecture. We return to this issue in the Discussion.

Figure 4 about here

These data confirm our previous work indicating that 1) visual intensity and fixation release produce additive effects on saccade latencies (Reuter-Lorenz et al., 1991) and the 2) fixation point offsets facilitate saccadic latencies for either visual or acoustic targets

(Fendrich et al., 1991). Furthermore, the significant interaction of auditory and visual intensity is consistent with the idea that these signals are combined by neural summation.

This possibility was tested by using the analysis developed by Nozawa and Townsend (1991; see also Hughes et al., 1991) to compute estimates of neural summation for bimodal targets in both the gap and overlap conditions. Evidence for neural summation was obtained in both conditions (Figure 4), confirming our previous results (Hughes et al., 1991).

Discussion

These data confirm and extend our previous findings concerning the effects of target luminance and fixation point offsets on saccade latencies. The data also replicate the results of Hughes et al. (1991) by demonstrating strong neural summation for combined auditory and visual targets. In general, the results can be accounted for by a serially organized, two-stage model of saccade generation. The first stage is viewed as a sensory processing stage in which visual and auditory inputs are summed via convergence onto common neural elements. The second stage involves premotor processing and its duration is partly determined by the presence or absence of a fixation signal.

The presence of an interaction between auditory intensity and fixation condition, however, does not entirely fit with the model presented in Figure 1. While this outcome raises the possibility that fixation release and auditory target intensity affect a common processing stage, such an interpretation would be incompatible with the effects observed for the visual modality and for the bimodal stimulus conditions. We favor an alternative explanation based on the relative timing of the fixation release state and the arrival of the sensory stage output at the premotor stage.

Figure 4 represents the possibility that the degree of fixation release varies over time from the offset of fixation stimulus. If this idea is correct, then for a given stimulus intensity, the magnitude of the gap effect should vary with the duration of the gap interval.

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Figure Captions

Figure 1. Diagram depicting a two-stage serial model of saccade generation in which auditory and visual inputs converge in a neural summation stage and a fixation release signal influences the time course of premotor processing. The inset graph represents how the time course associated with the transition from the engaged to the released state may influence the speed of premotor processing. Premotor processing time becomes faster with increasing time after fixation point offset.

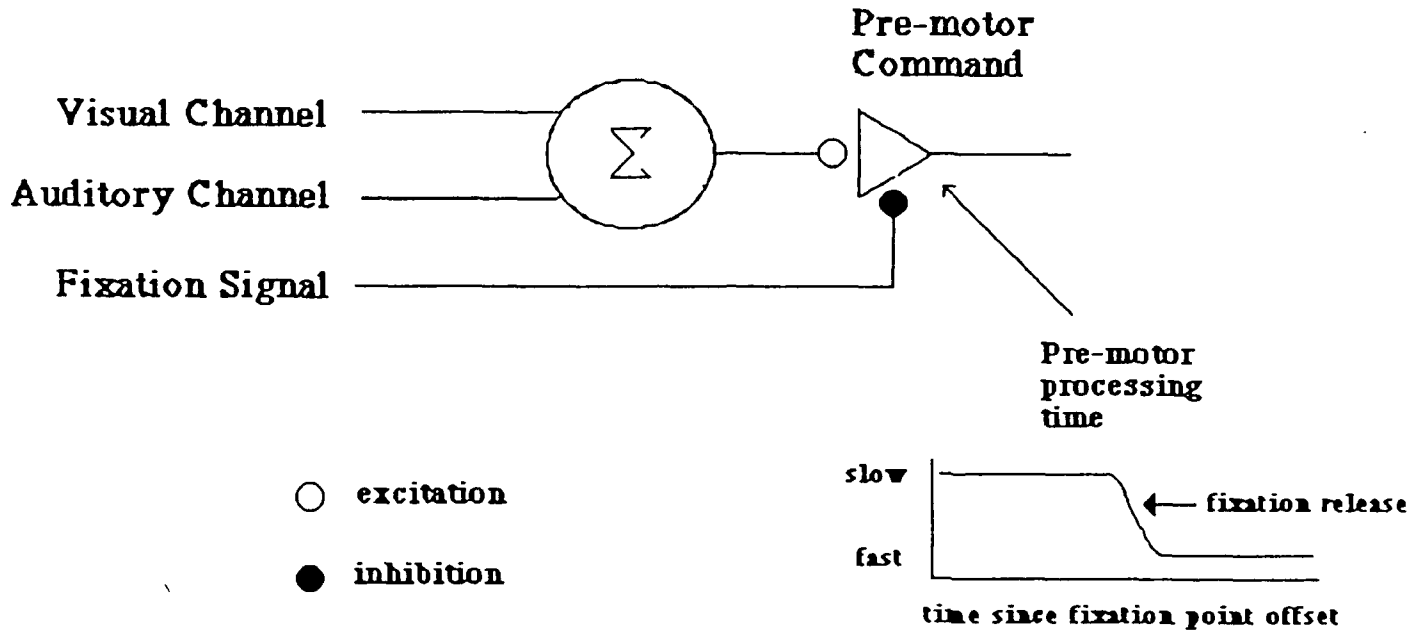
Figure 2. Graph representing the averaged SRT to high and low luminance lights in the gap and overlap conditions.

Figure 3. Graph representing the averaged SRT to high and low intensity auditory signals in the gap and overlap conditions.

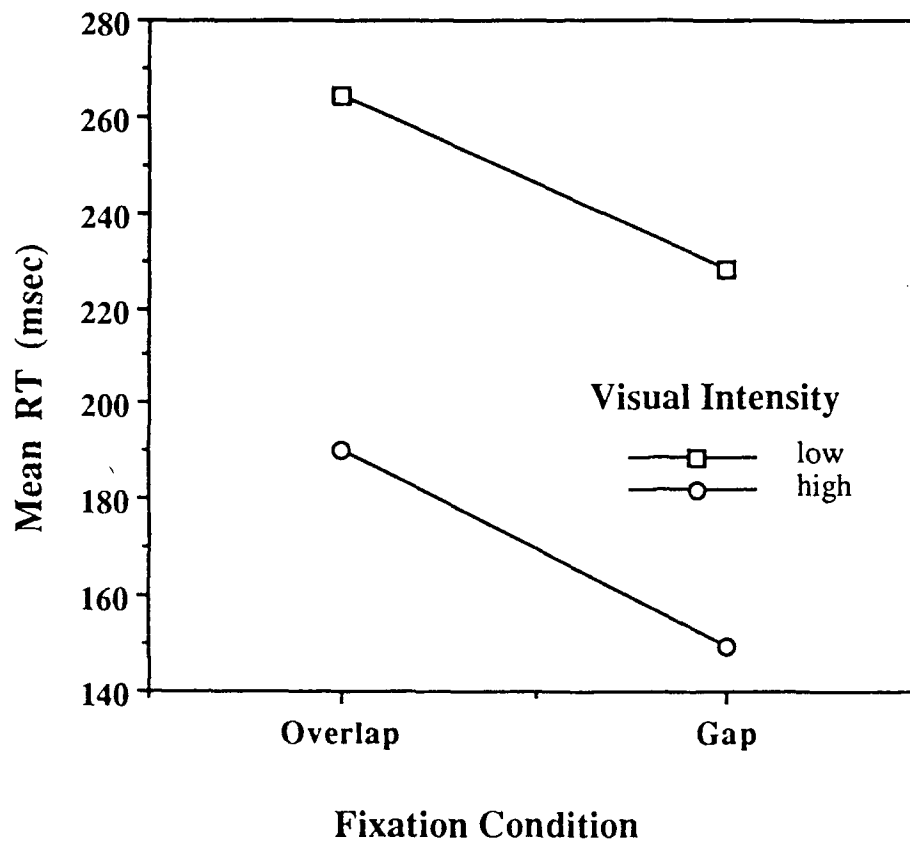
Figure 4. S-measures for the gap and overlap conditions averaged across all subjects. The S-measure represents the difference between obtained and predicted performance in the bimodal stimulus conditions. Negative S-measures indicate neural summation whereas probability summation is indicated when the S-measure equals 0. See text and Section 1.2 for details.

Figure 5. Diagram representing the possible time course of fixation release relative to the timing of outputs from the sensory processing stage. The figure presents the idea that in the case of a high intensity auditory target activity may arrive at the premotor stage before fixation release is complete. This could result in less facilitation in the gap condition for high than for low intensity signal, as obtained in the auditory condition (see Fig. 3).

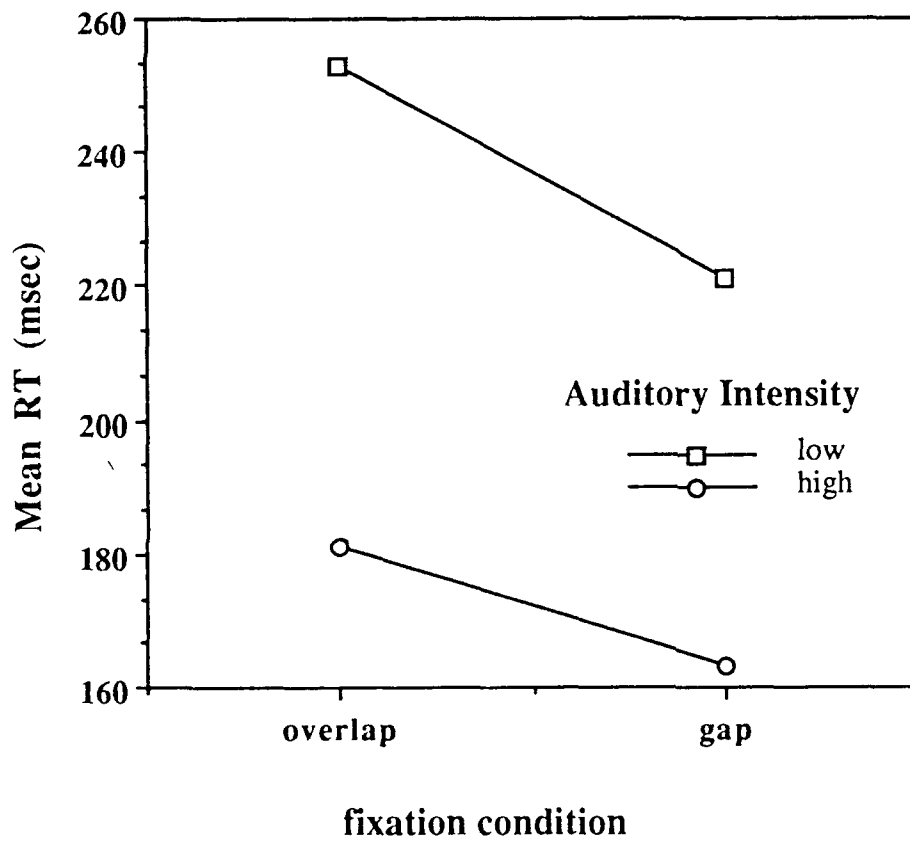
Two-stage Model of Bimodal Convergence and Express Saccades



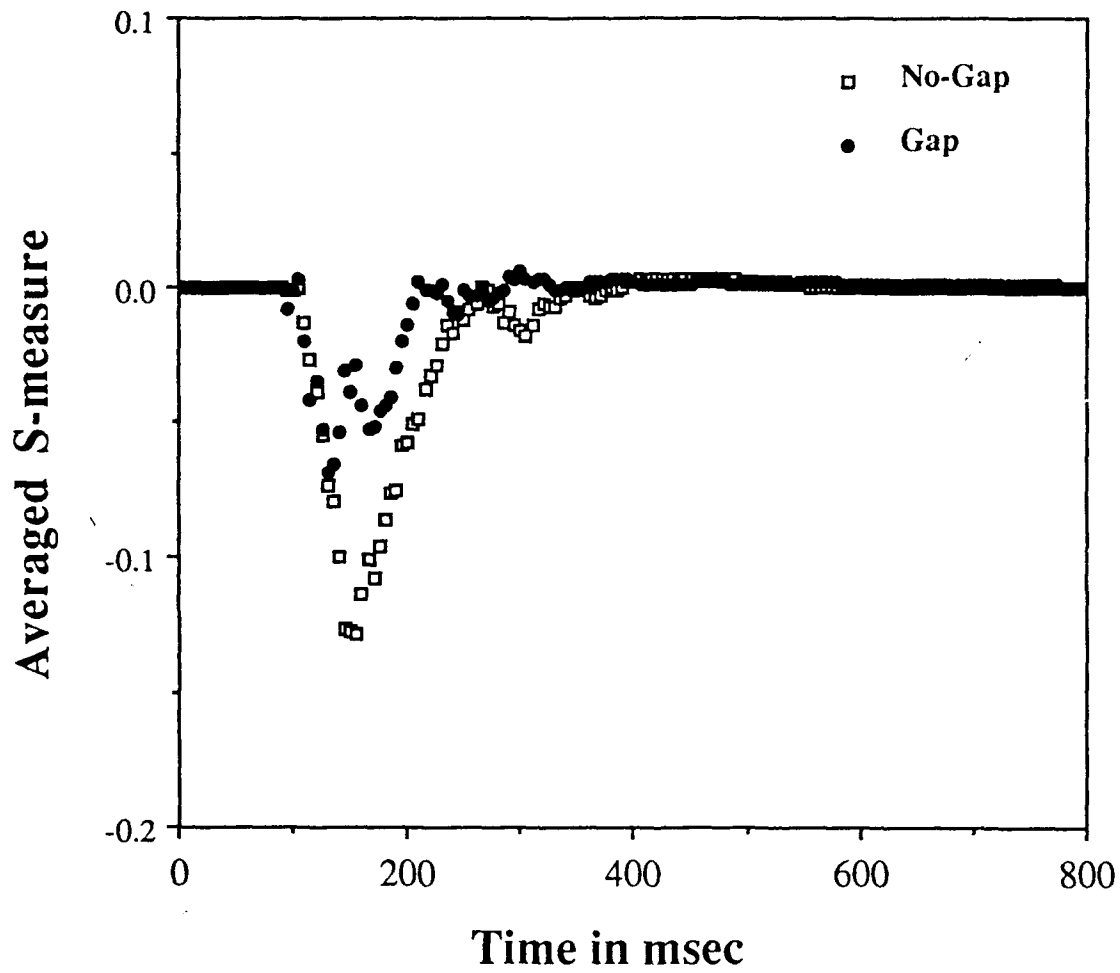
Visual Intensity by Fixation Additivity



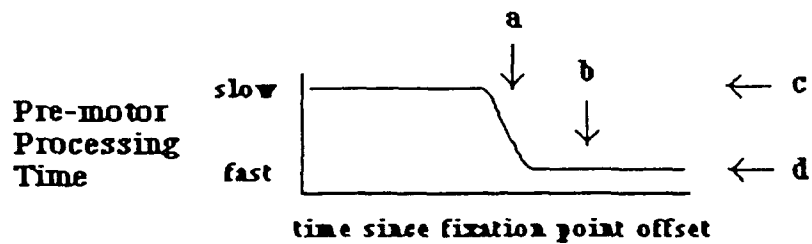
Auditory Intensity by Fixation Interaction



Evidence of Neural Summation in Gap and No-Gap Conditions



Time-course of Fixation Release



Possible explanation for interaction between target intensity and Fixation Release

- a: detection time associated with high intensity target
- b: detection time associated with low intensity target
- c: Pre-motor time associated with engaged fixation
- d: Pre-motor time associated with fixation release

SECTION 1.2

Bimodal Convergence in the Saccadic Control System

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Electrophysiological studies show that auditory and visual afferents converge onto premotor neurons within the oculomotor system of cats and non-human primates. Our analysis of saccadic latencies suggests similar patterns of convergence within the human oculomotor system. Saccadic and manual reaction times to bimodal targets were compared with predicted performance based on probability summation between independent visual and auditory channels. Only saccadic latencies showed facilitation from bimodal targets which exceeded probability summation. We suggest that this facilitation reflects neural summation of converging visual and auditory inputs within the superior colliculus.

Only 0.01% of a visual scene is processed with the exquisite spatial resolution conferred by the foveal cone mosaic. The dramatic reduction in acuity outside the fovea is partially compensated by rapid refixation eye movements (called saccades), which align the fovea with areas of interest. Recent electrophysiological demonstrations that visual and auditory inputs converge on individual neurons within the deeper layers of the superior colliculus¹⁻³ suggest summation of (bimodal) inputs prior to initiating saccades. If similar patterns of convergence operate in the human oculomotor system, saccades to combined visual and auditory targets should be faster than responses to unimodal targets. If this convergence is specific to the oculomotor system, intersensory summation may be restricted to saccadic responses. The present investigation reveals a form of intersensory summation that is *specific* for saccades, providing a behavioral correlate of bimodal convergence in oculomotor pathways.

We compared the magnitude of bimodal facilitation of saccades with two speeded manual tasks using similar stimulus conditions. *Directed-manual responses* required subjects to deflect a joystick toward the target location. For *simple-manual responses*, subjects depressed a microswitch in response to the target onset.

The visual targets were red light-emitting diodes (LEDs). The acoustic targets were 100 ms white noise bursts delivered through speakers located directly beneath the LEDs. Target eccentricity was 20°. The fixation point was a central LED. Warning tones (1.0 kHz, 300 ms duration) were presented through a central speaker 1.0 s before target onset. The apparatus was housed in a sound-attenuated enclosure. Eye position was digitized at 250 Hz using infrared reflection of the limbus. A bite-plate minimized head movements. Saccades and joystick responses were detected using a velocity criterion (≈ 50 deg. sec⁻¹).

To maximize the likelihood of central simultaneity of the inputs, preliminary testing identified stimulus intensities which produced equivalent auditory and visual RTs in each subject. The visual intensities ranged from 0.7 to 70.0 cd/m² while the auditory intensities ranged from 30 to 58 dB. Four naive observers participated in 15 experimental sessions of 60 trials each. Each observer generated at least 100 observations for each stimulus-response condition. Auditory, visual and bimodal targets occurred with equal frequency. Order and location were randomized.

Bimodal stimuli significantly reduced RTs for saccades and directed-manual responses, but not for simple-manual responses ($F_{4,12} = 7.26$, $p < .01$, see Table 1). However, faster responses to bimodal targets need not imply neural convergence (summation) between the two modalities^{4,5}. If each modality is processed by independent parallel channels, responses to bimodal targets could simply be determined by whichever modality is detected first (equivalent to the logical *OR* operation). Since the detection times for each modality are random variables, some latency reduction is expected on the basis of statistical or *probability summation*. Facilitation beyond that predicted by probability summation implies *neural summation* of the inputs, since under the same assumptions, neural summation is always faster than probability summation⁶. We therefore compared theoretical latency distributions derived from a simple model of probability summation between unimodal targets to the obtained bimodal distributions.

First, we assume that (I) the channels are stochastically independent and that (II) selective influence holds (e.g., the visual channel is not influenced by an auditory stimulus, see ref. 7). According to probability summation, bimodal RTs are determined by whichever modality is detected first. Thus, if we define T_A as the random variable representing auditory detection time and T_V as the random variable representing visual detection time, bimodal detection time can be expressed as:

$$T_{A\&V} = \min (T_A, T_V) \dots \dots \dots (1)$$

Considering $\min (T_A, T_V)$ in terms of the cumulative distribution function (cdf), the predicted bimodal latency distribution is expressed as

$$\begin{aligned} P^{\text{pred}} (T_{A\&V} \leq t) &= P (\min (T_A, T_V) \leq t) \\ &= P (T_A \leq t \text{ or } T_V \leq t) \\ &= P (T_A \leq t) + P (T_V \leq t) - P (T_A \leq t \text{ and } T_V \leq t) \\ &= P (T_A \leq t) + P (T_V \leq t) - P (T_A \leq t) \times P (T_V \leq t) \dots \dots \dots (2). \end{aligned}$$

The joint probability, $P (T_A \leq t \text{ and } T_V \leq t)$ is defined in the interval [0,1], yielding the following inequality:

$$\begin{aligned} P^{\text{pred}} (T_{A\&V} \leq t) &\leq P (T_A \leq t) + P (T_V \leq t), \text{ or} \\ P^{\text{pred}} (T_{A\&V} \leq t) - P (T_A \leq t) + P (T_V \leq t) &\leq 0 \dots \dots \dots (3) \end{aligned}$$

Miller^{8,9} proposed that violations of this inequality could be used to evaluate probability summation. However, $P(T_{A\&V} \leq t) \leq 1$, so Eq 3 can only be evaluated in the early portion of the cumulative latency distribution, where $P(T_A \leq t) + P(T_V \leq t) \leq 1$ holds.

An alternative to Eq (2) uses survivor functions, the complement of the cdf. The survivor function specifies the probability that a response has *not* occurred as a function of time "t" after target presentation. The survivor function for auditory RTs can be defined as $S_A(t) = P(T_A > t) = 1 - P(T_A \leq t)$,

$S_V(t) = P(T_V > t) = 1 - P(T_V \leq t)$ for visual RTs and

$S_{A\&V}^{obt}(t) = P(T_{A\&V} > t) = 1 - P(T_{A\&V} \leq t)$ for bimodal targets. The survivor function for bimodal targets predicted by probability summation is thus:

$$\begin{aligned} S_{A\&V}^{pred}(t) &= P(\min(T_A, T_V) > t) \\ &= P(T_A > t \text{ and } T_V > t) \\ &= P(T_A > t) \times P(T_V > t) \dots \dots \dots (4) \end{aligned}$$

Under assumptions I and II, Equation (4) states that the probability of *not* detecting a bimodal target by time t equals the joint probability that neither unimodal target has been detected by that time.

To compare obtained bimodal performance with that predicted by probability summation, we compute the difference between the obtained bimodal survivor function and the predicted survivor function (Eq.4) :

$$S(\text{survivor}) \text{ measure} = S_{A\&V}^{obt}(t) - S_{A\&V}^{pred}(t) \dots \dots \dots (5)$$

Negative values of the S-measure indicate bimodal facilitation in excess of probability summation, suggesting neural summation.

Fig. 1a -d illustrates S-measures computed for each subject for the three tasks. Only saccades show evidence of neural summation; manual task performance is consistent with the probability summation prediction. Although unimodal RTs for simple manual responses were not well matched, subsequent work in this laboratory has shown robust neural summation for saccades even when the unimodal means differ by as much as 100 ms. The integral of the survivor function is the distribution mean¹⁰, so we can compute predicted means from the predicted bimodal survivor function (Eq 4). Analyses of variance comparing the obtained and predicted means for each task confirms the conclusion that only saccadic latencies were significantly faster than probability summation (saccades: $F_{1,3} = 30.25$, $p < .015$; directed-manual: $F_{1,3} = 2.4$, n.s.; simple manual: $F_{1,3} = 3.6$, n.s.; see Figure 2).

While the S-measure (Eq. 5) assumes independent sensory channels, a negative dependency (i.e., faster processing in one channel occurs jointly with slower processing in

the other and vice versa) between channels predicts faster bimodal RTs than when independence is assumed. Since the joint probability, $P(T_A \leq t \text{ and } T_V \leq t)$, approaches 0 with increasing negative dependency, Eq 3 (here referred to as "Miller's inequality") represents the upper limit of facilitation attainable by any model of probability summation, whether or not stochastic independence is assumed¹¹. Eq 3 is therefore a more conservative test of neural summation than Eq 5. To analyze the data using Eq 3, we substitute the obtained (obt) bimodal **cdf** for the predicted bimodal **cdf**, and evaluate

$P^{obt}(T_{A\&V} \leq t) - P(T_A \leq t) - P(T_V \leq t) \leq 0$ (termed the **M** {"Miller's inequality"} measure). This analysis confirmed the finding of neural summation for saccades (Fig. 3). The failure of the **S** measure to provide compelling evidence of neural summation for manual responses, combined with the demonstration of neural summation using the conservative **M** measure, provides robust support for the conclusion that neural summation was specific for saccades.

Convergent visual and auditory inputs onto common premotor neurons provides an architecture which maximizes the speed of sensory-motor processing. The present evidence suggests that this design may be relatively unique to the oculomotor system, perhaps reflecting the high priority of rapid ocular orienting relative to other motor responses.

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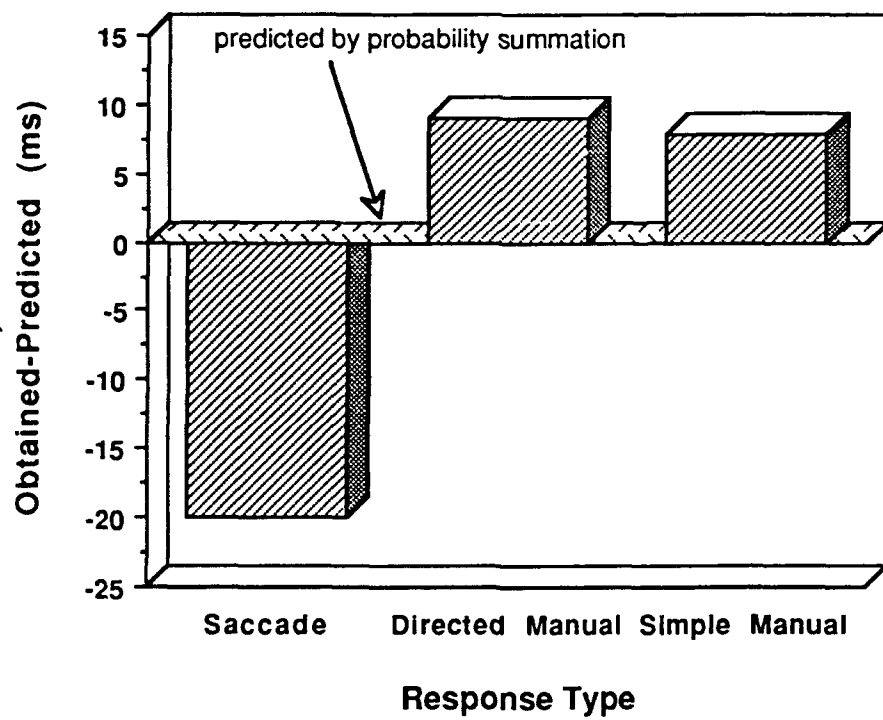
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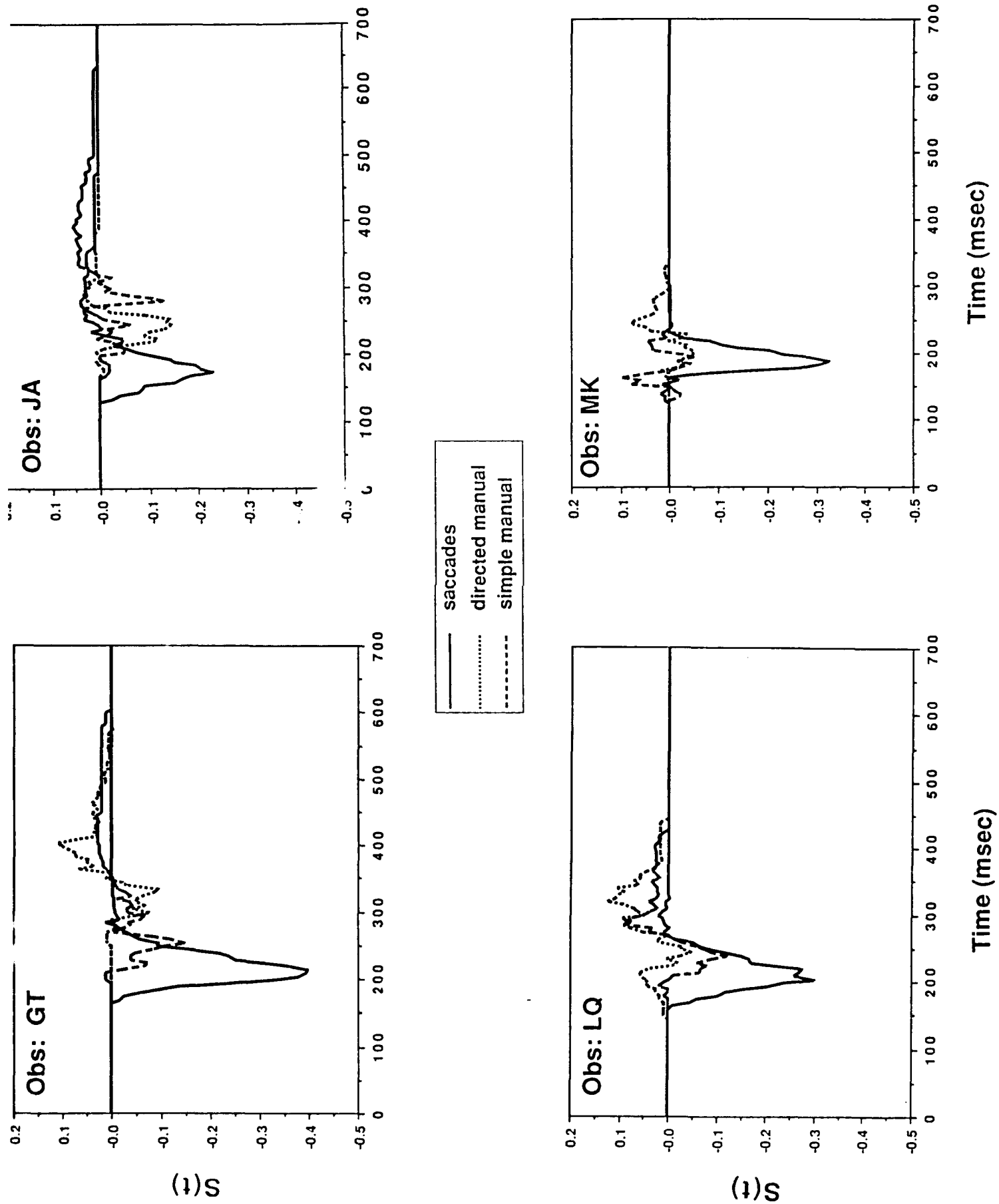
TABLE 1 Mean latency and standard deviations for each response condition with unimodal and bimodal targets.

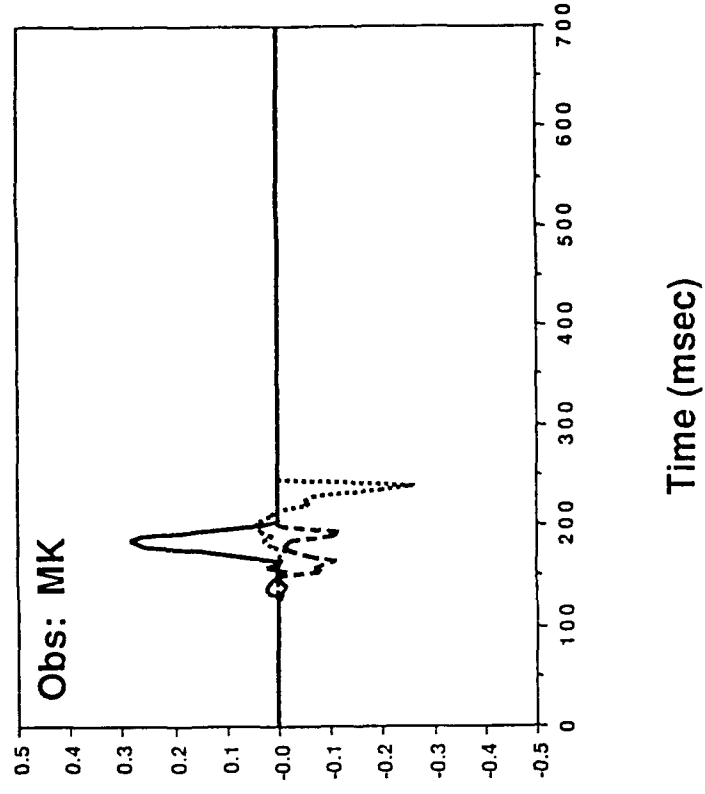
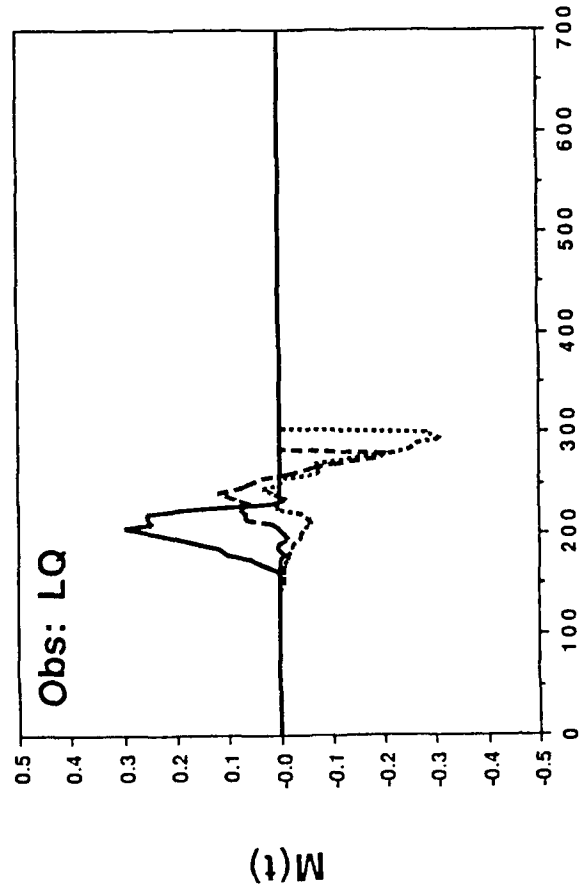
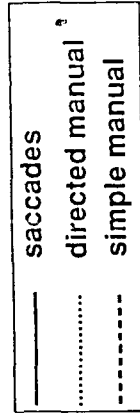
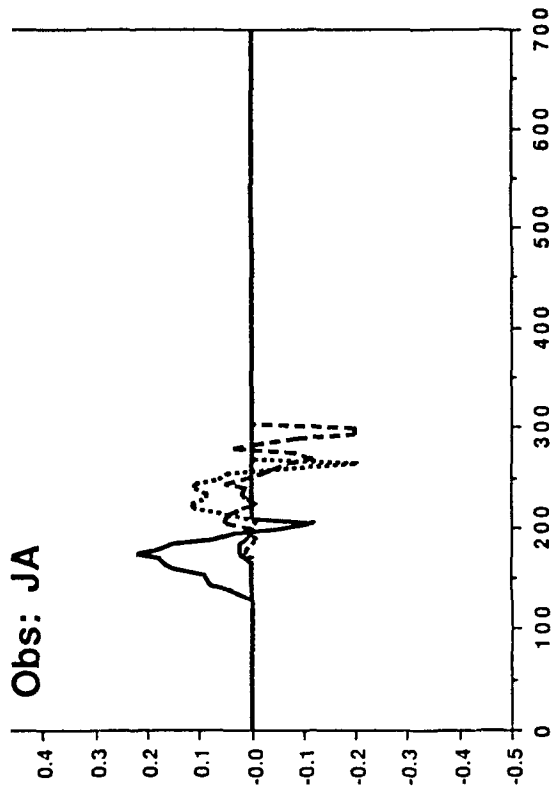
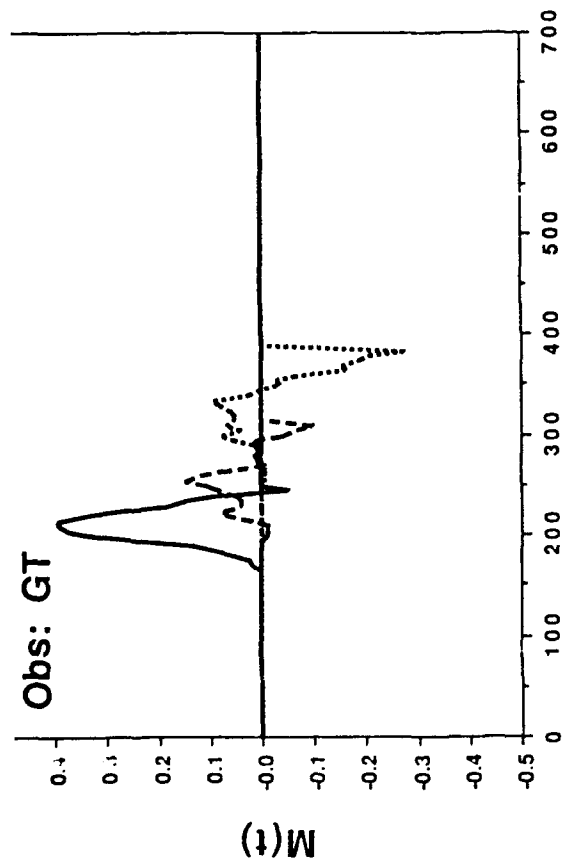
RESPONSE	STIMULUS CONDITION		
	VISUAL	AUDITORY	BIMODAL
SACCADE			
MEAN	233	218	191
S.D.	41.6	35.7	31.3
DIRECTED-MANUAL			
MEAN	335	313	299
S.D.	84.9	79.0	74.9
SIMPLE-MANUAL			
MEAN	303	255	248
S.D.	55.5	44.3	43.2

Figure Captions

- Figure 1. Differences (in ms.) between obtained bimodal RTs and predicted RTs based on probability summation for three different response conditions. Data are expressed as obtained-predicted, averaged across 4 observers.
- Figure 2. *S* measures ("survivor measures", Eq. 5) of bimodal performance for three different response conditions. Negative values represent facilitated performance beyond that predicted by probability summation. See text for details.
- Figure 3. *M* measures ("Miller's inequality, Eq. 3) of bimodal performance for three different response conditions. Positive values represent facilitated performance beyond that predicted by probability summation. See text for details.







SECTION 1.3

**Fixation Point Offsets Reduce the Latency of
Saccades to Acoustic Targets**

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ABSTRACT

If an observer's fixation point is extinguished just prior to the onset of a peripheral target, the latency to saccade to that target is reduced. We show that this "gap effect" is not specific to visual targets. Observers made saccades to a light flash or to a white noise burst. A warning tone was presented on every trial to control for the possible warning effect of the fixation point offset. For both target modalities saccade latencies were significantly reduced when the fixation point was extinguished 200 ms. prior to the target onset. Implications of this outcome for interpretations of the gap effect are considered. It is argued that the presence of a gap-effect for tones, in conjunction with previous findings, is consistent with the hypothesis that the gap effect is produced by a facilitation of premotor processes in the superior colliculus.

When human or monkey observers saccade to the onset of a visual target, their saccadic latency is reduced by the prior offset of the fixation point (e.g. Fischer, 1987; Fischer & Boch, 1983; Fischer & Ramsperger, 1984; Fischer & Ramsperger, 1986; Saslow, 1967). In human subjects, the magnitude of this reduction is about 50 ms. This reduction of saccadic latency - frequently referred to as the "gap effect" - is maximal if the fixation point is offset 200-300 ms. before the target's appearance. The gap effect occurs even if a warning signal is employed to control for the alerting effect produced by the fixation point offset (Reuter-Lorenz, Hughes, & Fendrich, 1991). Fischer and coworkers have suggested that this reduction of saccadic latency is the consequence of the appearance of a distinct subpopulation of "express saccades" with modal latencies of 120 ms. in humans (Fischer, 1987; Fischer & Boch, 1983; Fischer & Ramsperger, 1984). However, fixation point offsets decrease saccadic latencies even when the bimodal latency

distribution characteristic of express saccades has not been found (Kingstone & Klien, 1990; Reuter-Lorenz et al., 1991).

Several studies have indicated that the superior colliculus plays an important role in generating short-latency saccades produced in the gap paradigm. In monkeys, the gap effect is eliminated following ablation of the superior colliculus (Sandell, Schiller, & Maunsell, 1984; Schiller, Sandell & Maunsell, 1987). In addition, Rohrer & Sparks (1986) report that the interval between the visual and presaccadic activity bursts of cells within the deeper layers of the SC is reduced prior to such short latency saccades. One probable role of the colliculus is to initiate rapid reflexive saccades towards peripheral visual events (Sparks & Mays, 1980; Wurtz & Albano, 1980). These reflexive saccades may be inhibited during active fixation (Munoz & Guitton, 1989). Sparks and Mays (1983) have found that during fixation the threshold to elicit saccades by electrical stimulation of the colliculus is increased. The offset of a fixation point could enable short latency saccades by reducing this inhibition (Reuter-Lorenz, et al., 1991).

Saccades can be directed towards non-visual targets. There are cells in the deep layers of the superior colliculus which receive acoustic inputs and increase their rate of discharge prior to saccades to acoustic targets (Jay & Sparks, 1987, 1990). Thus, a disinhibition of collicular orienting mechanisms might well enable short latency saccades to acoustic stimuli. This suggests that if the gap effect is due to a disinhibition of collicular reflexes, a gap effect ought to be observable with auditory targets.

Other explanations of the gap effect also predict this effect with acoustic targets. Saslow (1967) suggested that the elimination of the fixation point may serve to reduce the probability of corrective microsaccades just prior to the onset of the target, thereby reducing the refractory periods microsaccades produce. Kalesnykas and Hallett (1987) have suggested that the offset of the fixation point may increase the likelihood of anticipatory saccades, with express saccades forming a population of direction-appropriate anticipations prepared before but executed after the target onset. Fischer and his colleagues (Fischer, 1987; Fischer & Breitmeyer, 1987; Mayfrank, Mobashery, Kimmig, & Fischer, 1986) have proposed that the offset of the fixation point serves to release a subject's attention, so that attention is more quickly engaged by the target. According to all of these views, the gap effect should be present irrespective of the modality of the saccadic target. On the other hand, one explanation of the gap effect does not predict saccades to acoustic targets, at least in its present form. Reulen (1984a,b) has proposed that the offset of the fixation point may serve to facilitate the visual processing of the target.

The occurrence of a gap effect to non-visual targets is therefore relevant to a number of explanations of this effect. However, the existence of a gap effect for saccades to such targets has never actually been demonstrated. Here we show that fixation point offsets do in fact reduce the latency of saccades to auditory targets. In addition we compare the magnitudes of the gap effect obtained with auditory and visual targets.

Method.

Subjects were seated 114 cm from a stimulus panel aligned on an arc with a radius of 114 cm. A central green light-emitting diode (LED) served as the fixation stimulus. Two red LEDs mounted on the panel 10° to the left and right of the fixation point provided the visual targets, and two small (4 cm.) speakers mounted directly below the red LEDs provided the auditory targets. The visual targets consisted of 300 ms. 0.7 cd/m² LED flashes while the auditory targets were 300 ms. 90dB white noise bursts. These intensity levels were chosen on the basis of preliminary testing which indicated they would produce similar saccadic response times. Warning tone bursts (at 2.8 KHz.) were provided by a small oscillator module mounted just above the fixation point. To minimize echoes, the apparatus was housed in a large enclosure (1.54 m. by 1.54 m by .9 m) which was lined with sound-absorbing foam (Sonex™).

Subjects sat with their head positioned by a bite plate just within the open front end of this enclosure. Subjects were run in a dark room after at least 5 minutes of dark adaptation, and could not see either the extinguished LEDs or speakers.

At the start of each trial, subjects fixated the green LED. An experimenter initiated the trial when an oscilloscope display of the subject's eye position indicated proper fixation. On each trial, the warning tone sounded for 100 ms. Two hundred ms. after the offset of this tone, the visual or auditory target was presented. Subjects were instructed to saccade to the target as rapidly as possible.

Testing was carried out in blocks of 72 trials. Within each block, there were 32 trials with visual targets and 32 with auditory target. Half of the targets were on the left and half on the right. In order to discourage anticipatory responses 8 trials in each block were catch trials in which there was no target. Half of the trials for each modality and half of the catch trials were "gap" trials. In gap trials, the fixation LED was turned off at the offset of the warning tone. The 200 ms. interval between the fixation point offset and target presentation constituted the gap. The remaining trials were "overlap" trials in which the fixation point remained on from the start of the trial until 1000 ms. following the target onset. The order of the various types of trials was completely randomized within each block.

An IBM PC-XT microcomputer controlled the display presentations via a 16 bit parallel output port and custom built interface unit. Eye motions were monitored with a scleral infrared reflection device (Narco Biosystems™ Model 200) sampled via a 12 bit A/D converter at 200 Hz. The Eye-trac 200 was calibrated at the start of each block of trials. On each trial, the subject's horizontal eye position was sampled for 1000 ms. starting at the onset of the warning tone. The eye records were stored on disc for subsequent analysis.

Saccades were detected by a computer program which used a velocity criterion (50°/sec.). In addition, accurate saccade detection was verified by visual inspection of a CRT display of each eye record. Trials in which computer detection errors occurred were corrected.

Six naive observers served as the subjects. Each observer received at least one block of practice trials prior to formal data collection. Formal data was collected over 7 blocks of trials, yielding a total of 112 observations per subject in each of the experimental conditions (gap-visual target, overlap-visual target, gap-auditory target, overlap-auditory target).

Results.

Any trial with an initial saccade in the wrong direction was discarded. In addition, for each subject in each condition, trials with saccadic latencies more than 2.5 standard deviations from the mean latency of that condition were excluded from the final analyses. Finally, saccades with latencies less than 100 msec. were taken to be anticipations and removed (Kalesnykas & Hallett, 1987). Altogether, these procedures eliminated 4.6 % of the data points, 4.1% of the no-gap trials and 5% of the gap trials.

Means were computed for each subject in the 4 experimental conditions. With visual targets, the mean saccadic latency across the 6 subjects was 287.8 ms. (s.d.=53) in the overlap condition and 244.8 ms. (s.d.=46) in the gap condition. With acoustic targets, the mean latency was 264.2 ms. (s.d.=52) in the overlap condition and 233.8 ms. (s.d.=37) in the gap condition. Thus, a mean gap effect of 43 ms. was obtained with visual targets and 30.4 ms. with auditory targets. These data are graphed in Figure 1.

Insert Figure 1 about here.

An ANOVA was run on the subject means in the four conditions. Although acoustic targets produced faster responses and a smaller gap effect than visual targets, only the main effect of the gap was significant ($F(1,5)=10.76$; $p<.03$). Paired comparisons using the Newman-Keuls procedure indicated that for both visual and

acoustic targets, latencies were significantly faster in the gap than in the overlap condition ($p < .05$). It should be noted, however, that the gap-modality interaction came quite close to significance ($F(1,5) = 5.4$; $p < .07$), suggesting the tendency for the gap effect to be smaller with acoustic than visual targets may be genuine.

Latency histograms for two representative observers are illustrated in Figures 2.

Figure 2(a-h) about here

Similar to the report of Reuter-Lorenz et al. (1991), the majority of the obtained latency distributions failed to show evidence of bimodality. In the distributions illustrated, a suggestion of bimodality is observable only in the auditory target data for subject A.Y. Generally, a gap effect was found because fixation offsets tended to shift or compress entire distributions toward shorter latencies.

The false alarm rate was .08 (4.5 per 56 catch trials), with half the subjects showing 2 or fewer catch trial saccades. The majority (19 of 24) of the saccades occurred in gap catch trials. The average latency of the catch trial saccades, measured from the time the target would have onset had one been presented, was 182 ms. (s.d.=66.5). There was no apparent relationship between the number of saccades a subject made during the catch trials and the magnitude of a subject's gap effect; averaged across modality, virtually identical gap effects of 28 and 27 ms. were obtained from

the subjects with the highest (9) and lowest (0) number of catch trial saccades.

Discussion.

These results indicate that the prior offset of a fixation point facilitates saccades to acoustic as well as to visual targets. This finding suggests that the gap effect cannot be attributed simply to enhanced visual processing (Reulen, 1984a,b). The fact that the gap effect is not modality specific is consistent with an interpretation of this effect in terms of a facilitation of motor or premotor processes. The deep layers of the superior colliculus appear a likely candidate for the locus of this premotor facilitation, since this structure receives convergent visual and auditory inputs and is involved in initiating saccades (e.g Jay & Sparks, 1990; Meredith & Stein, 1986).

There is an indication that the magnitude of the gap effect may be stronger for visual than for auditory targets. Assuming this interaction is real, we can only offer suggestions as to how such a difference might arise. The magnitude of the gap effect varies with gap duration (Saslow, 1967). The gap duration we employed is optimal for visual targets (Saslow, 1967), but might not be optimal for auditory targets. In addition, although visual and auditory inputs converge in the colliculus, the characteristics of saccades to auditory targets differ from visually triggered saccades. For example, auditory targets have a lower peak velocity and are more likely to be double saccades (Jay and Sparks, 1990). These

differences imply a difference in saccadic programming for targets of different modalities, which could also affect the magnitude of the gap effect.

As noted in the introduction, several alternative accounts of the gap effect are also consistent with the fact that it occurs with auditory targets. Saslow's (1967) attribution of this effect to an increased incidence of microsaccades during the gap would predict this outcome. However, Saslow's hypothesis fails to account for the absence of a gap effect with antisaccades (Reuter-Lorenz et al., 1991).

The attribution of the gap effect to an increased incidence of anticipatory saccades (Kalesnykas and Hallett, 1987) is also consistent with the present finding. As this hypothesis would predict, more saccades occurred in gap than overlap catch trials. Furthermore, the mean latency of these saccades (relative to the time a target would have appeared in a test trial) was short. However, the gap effect was present in subjects who made few or no saccades in catch trials, and across subjects the number of catch trial saccades was not related to the magnitude of the gap effect. We therefore acknowledge that anticipations may sometimes contribute to the gap effect, but believe they are unlikely to be the primary source of this effect. (A further discussion of the role of anticipations in the gap effect can be found in Reuter-Lorenz et al., 1991).

An attribution of the gap effect to the release of attention (Fischer, 1987; Fischer & Breitmeyer, 1987; Braun & Breitmeyer, 1988) is in agreement with the current outcome if one assumes that

once released attention will be engaged by acoustic as well as visual stimuli. Precuing experiments have failed, however, to demonstrate any effect of spatial attention on responses to auditory targets (Buchtel & Butter, 1988; Posner, Nissen & Ogden, 1978). Admittedly, this observation needs to be regarded with caution, since it has not been specifically demonstrated that spatial precues do not influence saccades to auditory targets. In addition, spatial precues interact with target luminance (Hawkins, Shafto & Richardson, 1988) while the gap effect is additive with luminance (Reuter-Lorenz et al., 1991), and the absence of a gap effect with either manual responses or antisaccades (Reuter-Lorenz et al., 1991) does not seem in accord with the attentional hypothesis. On the other hand, it has been proposed that the superior colliculus serves to control movements of attention (Posner & Petersen, 1990) and that movements of attention are tied to oculomotor programming (Rizzolatti, Riggio, Dascola, & Umiltà, 1987). To the extent that these proposals are correct, an interpretation of the gap effect based on movements of attention might prove compatible with an account based on collicular premotor processes.

An explanation of the gap effect in terms of the release of collicular orienting reflexes does account for its absence with antisaccades and manual responses, since the colliculus does not control manual responses and antisaccades are not directed toward a sensory target. The present finding of a gap effect with auditory targets strengthens the case for such an explanation.

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Figure Captions

Figure 1. Mean saccadic latencies for visual and acoustic targets in the gap and overlap conditions.

Figure 2. Distribution of saccadic latencies for 2 representative subjects in the 4 experimental conditions.

Acknowledgement

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FIG. 1

GAP EFFECT FOR VISUAL AND ACOUSTIC TARGETS

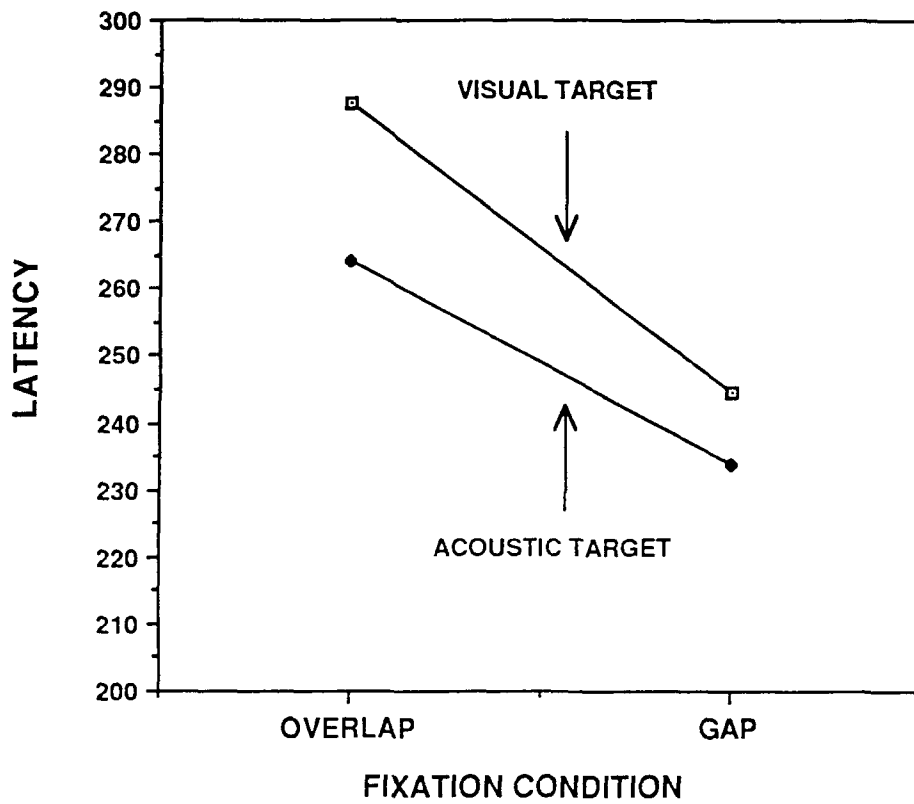


Fig. 2A

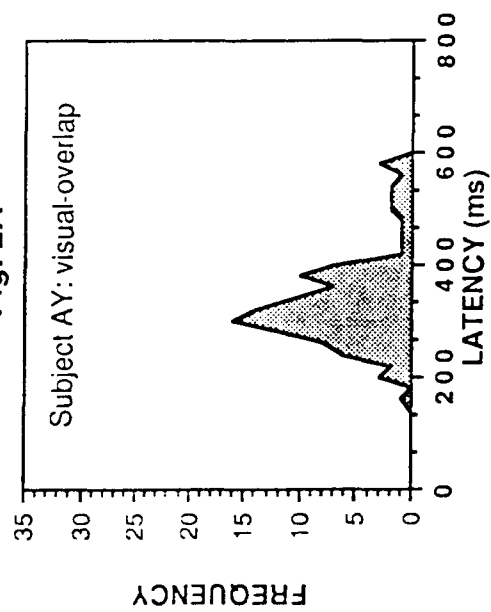


Fig. 2B

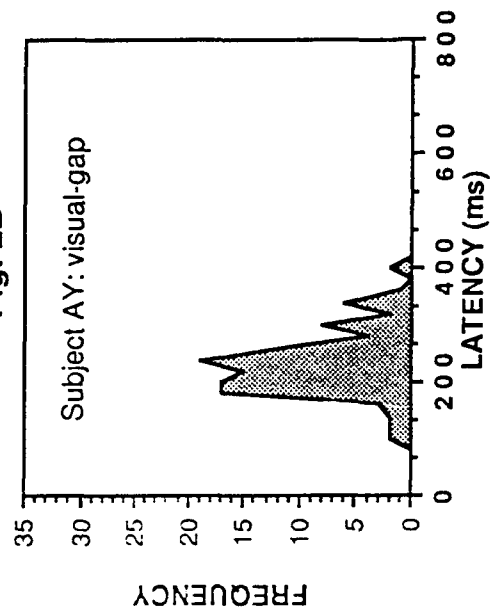


Fig. 2C

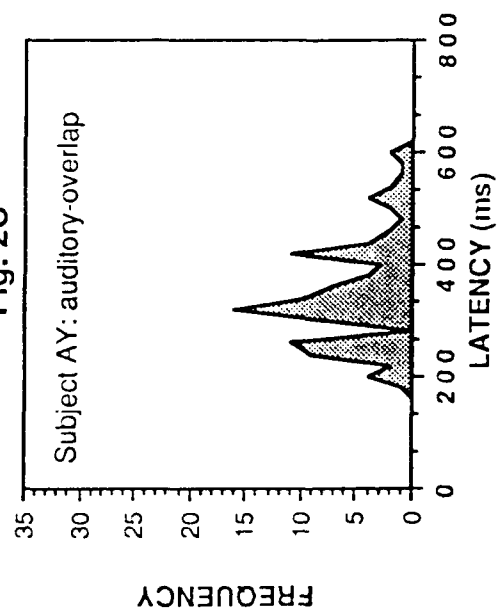


Fig. 2D

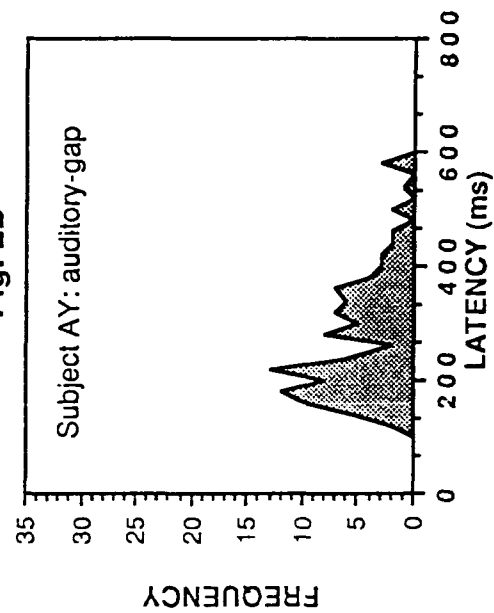


Fig. 2E

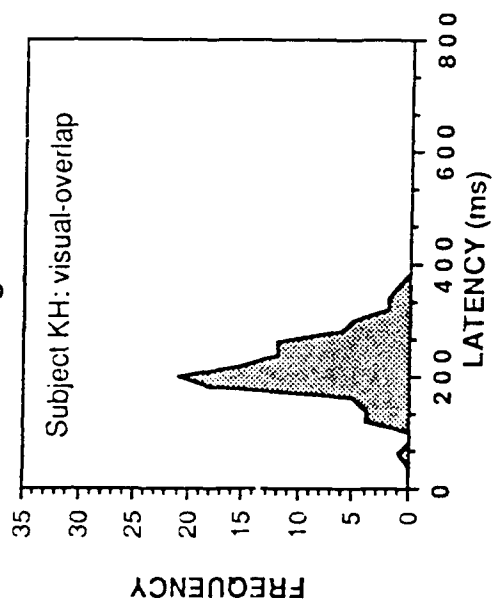


Fig. 2F

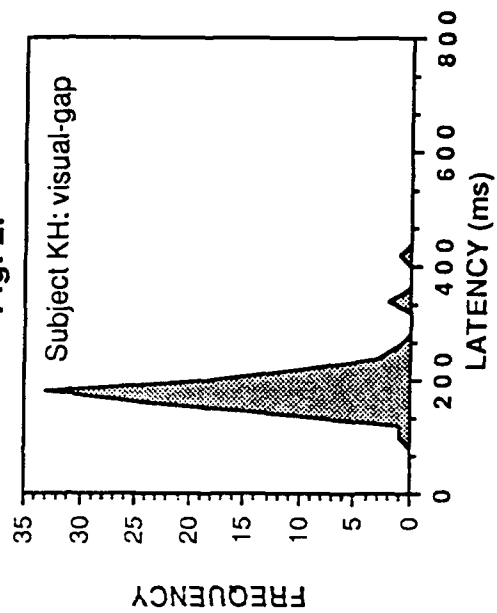


Fig. 2G

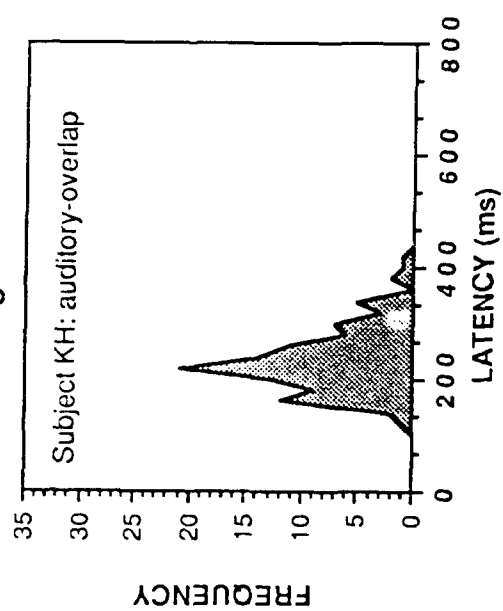
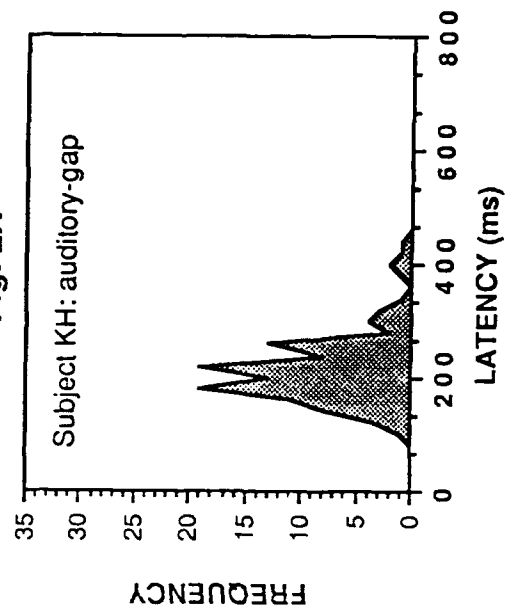


Fig. 2H



SECTION 2

Oculomotor Readiness and Covert Orienting:
Differences Between Central and Peripheral Precues

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ABSTRACT

The costs produced by invalid precues can depend on the spatial relationship between the cued location and the target location. If oculomotor programs mediate attention shifts, then the effect of varying the spatial relation between the cue and target should be the same for covert orienting (as indexed by manual responses) and saccadic responses. We found this to be true only for central precues, in which case both manual and saccadic costs were greater when cue and target occurred on opposite sides of the vertical meridian than when they occurred on the same side. For peripheral precues, no meridian effects were obtained in either response condition, but a significant dissociation in the patterns of saccadic and manual costs emerged. For manual responses costs were greater when the cue was eccentric relative to the target, whereas for saccades costs were greater when the target was eccentric to the cue. These results provide additional support for the idea that different orienting mechanisms are engaged by central and peripheral precues. They further suggest that the relationship between oculomotor and attentional orienting may depend on the nature of the precue, with the potential for interdependence being greater with central precues.

INTRODUCTION

Normally, our attention and gaze move together. However, it is possible to pay attention to one location while maintaining fixation at another. This ability has been demonstrated experimentally by cuing a spatial location prior to the onset of a target (Eriksen & Hoffman, 1973; Posner, 1980). Even if there are no overt eye movements, observers respond faster and more accurately when the target appears at a cued location than when it appears elsewhere (Hawkins, Hillyard, Luck, Mouloua, Downing & Woodward, 1990; Muller & Humphreys, 1991; Posner, 1980). Some investigators have characterized covert attention as a beam or spotlight that can be moved through space (Posner, Snyder & Davidson, 1980; Remington & Pierce, 1984), while others have suggested that it can be described as a zoom lens that goes from a broad to narrow focus (Eriksen & Yeh, 1985; Murphy & Eriksen, 1987). Both hypotheses entail the notion that attention is oriented or directed to a circumscribed region of space. The mechanisms underlying this ability are not well understood.

It has been proposed that movements of attention may be accomplished via the same motor programs that are used to make saccadic eye movements (see Klein, 1980). According to this hypothesis, attending to a location consists of generating an oculomotor program to move the eyes to that location. In the case of covert attention shifts, the saccade is not executed. While this view has intuitive appeal, initial tests provided no support for it (Klein, 1980; Posner, 1980).

Nevertheless, interest in this premotor hypothesis has recently been revived by Rizzolatti and colleagues (Rizzolatti, Riggio, Dascola, & Umiltà, 1987; see also Tassinari, Aglioti, Chelazzi, Marzi, & Berlucchi, 1987). Using a standard precuing task, they found that for invalid precues, response latencies were longer (i.e. costs were greater) when the cue and the target were on opposite sides of the vertical or

horizontal meridians than when cue and target were on the same side. While the need for interhemispheric interactions could explain the extra costs across the vertical meridian, this does not explain the costs associated with the horizontal meridian (e.g. Hughes & Zimba, 1985; 1987). Furthermore, since the arrays of possible target locations were never centered on the fovea, the extra costs associated with meridian crossings can not be explained by the need for the attentional focus to traverse the large cortical region representing the fovea (e.g. Downing & Pinker, 1985). As an alternative, the authors interpret the meridian effects in terms of saccadic programming. They reason that if a saccadic program is generated in response to a cue, invalid trials would require a modification of that program. Direction changes are required when the cue and target fall on opposite sides of the horizontal or vertical meridian, whereas only amplitude changes are required when the cue and target occur in different locations on the same side of the meridians. Evidence suggests that computations associated with saccade direction may take more time than programming amplitude (e.g. Komoda, Festinger, Phillips, Duckman & Young, 1973; Becker & Jergens, 1979; however see Abrams & Jonides, 1989). Therefore, Rizzolatti et al. (1987) proposed that the extra attentional costs associated with meridian crossings reflect the greater time required to change the direction parameter of the oculomotor program.

If attentional costs reflect the time course of saccadic reprogramming, then the pattern of costs for covert orienting indexed by manual response latencies should correspond closely to the pattern obtained with saccadic responses. For example, if meridian effects found with covert orienting reflect the relative rates at which saccadic amplitude and direction can be modified, similar meridian effects should also be obtained when subjects actually make saccadic responses.

To evaluate these predictions, two experiments were performed which compared the effects of spatial precues on simple manual responses and saccadic eye

movements in the same subjects. Since central and peripheral precues may involve different attentional mechanisms (Jonides, 1981; Shepherd & Muller, 1989), the first experiment used peripheral precues and the second used central cues.

EXPERIMENT 1

Method

Apparatus. An IBM PC-AT microcomputer with a Kinetic Vision Systems vector generator produced and controlled the stimulus displays. Data Translation DT2821 A/D converters were used to record response latencies. The stimuli were presented on a Hewlett Packard 1310A large screen X-Y monitor with a fast phosphor (P15) CRT. Display refresh and response sampling were synchronous at 250 Hz. Eye movements were monitored with a Purkinje-image eyetracker. Head motion was minimized with a bite-bar/head rest assembly.

Displays. Four outline boxes ($.66^\circ \times .66^\circ$) defined the target locations. These boxes were horizontally aligned on an oscilloscope screen, 2° and 6° to the left and right of a central fixation point. The precue consisted of a second outline box ($1^\circ \times 1^\circ$) briefly flashed around one of the target locations. The target was $.5^\circ \times .5^\circ$ "X" which was presented within one of these boxes.

Insert Figure 1

Procedure. At the start of each trial the array of four boxes and central fixation point appeared on the CRT screen. When subjects were fixating the central point, the cue appeared for 300 msec. The target was presented after a randomly selected ISI of 400 to 800 msec., providing that the subject's gaze remained within $.25^\circ$ of the fixation point. If this criterion was not met, the target presentation was delayed until it

was met or 1200 msec. had elapsed since onset of the cue. Trials which exceeded this maximum SOA were aborted and subsequently rerun. After the target appeared, it remained on the screen until the subject responded or 2 seconds had elapsed. In different trial blocks, subjects responded to the target onset by either pressing a response button with their right hand or making a saccade to the target location.

At the start of each experimental session, a fixation matrix was used to calibrate the eyetracker so that 1 ADC increment equalled 1 minute of arc. Small drifts in baseline eye position were corrected in the course of each session while the subject fixated the central point. Saccadic responses were detected with a position criterion. The onset of a saccade was taken to be the time at which the eye moved more than $.5^\circ$ in the direction of the target, if the eye continued to move at least $3/4$ of the distance to the target position without returning to the fixation point. Rare trials in which these criteria were not met, or an initial saccade was made in the wrong direction, were aborted and subsequently rerun.

Seventy-five percent of the trials were valid with the target appearing in the cued location. The remaining 25% were invalid with the cue and target appearing in different locations. All possible invalid cue-target pairings were presented with an equal probability. For each response type 768 trials were run, of which 192 were invalid. Subjects participated in eight experimental sessions, approximately 60 minutes long, over the course of 2-4 weeks. During each session, two blocks of 48 trials were run for each response type in a counter-balanced order. Upon the completion of each subset of 192 trials for each response condition, a preliminary analysis was performed to identify and eliminate outliers. At that time, trials with latencies falling more than 2 standard deviations above or below the mean for that subset were rerun along with any previously aborted trials.

Subjects. Five student volunteers with normal or corrected vision were paid for their participation.

Results

The group means obtained with valid and invalid cues for each response condition are presented in Table 1. The spatial position of the cue and target are indicated by a two digit code in which the first digit refers to cue position and the second digit indicates target position. Going from left to right, the numbers correspond to each of the four location boxes, with the far left position designated number 1 and the far right position designated 4 (refer to Figure 1).

Insert Table 1

We computed "costs" by subtracting the mean latency of valid trials from the mean of invalid trials for each target position. Although this difference includes both the benefits of valid cues and the costs of invalid cues relative to a neutral baseline, we cannot separately evaluate these components in the absence of a neutral condition. We refer to the valid-invalid difference as "costs" for the sake of brevity.

The mean latencies in Table 2 indicate that both saccadic and manual responses were faster on valid than on invalid trials. An analysis of variance (ANOVA) confirmed the significance of cue validity ($F(1,4)=111.3$; $p<.001$). However, no difference was found between the manual and saccadic response conditions. Costs were, in fact, identical at 40 msec for both conditions.

Insert Table 2

The effect of the spatial relationship between the cue and target was initially evaluated for each response condition by comparing the costs for the following four

cue-target pairs: (1) those separated by 4° and falling in the same hemifield or (2) those separated by 4° and falling opposite sides of the vertical meridian; and (3) those separated by 8° and falling on opposite sides of the vertical meridian and (4) those separated by 12° and falling on opposite sides of the vertical meridian. In a two-way ANOVA with response condition and cue-target pair as factors, only the effect of cue-target pair was reliable [$F(3,12)=4.67$; $p<.02$], indicating that regardless of response condition, costs were reliably influenced by the spatial relation of the cue and target. The comparison between the 4° same and opposite pairs is critical to evaluating the meridian effect, since the retinal separation between cue and target is the same for these pairs. However, a Newman-Keuls analysis indicated that this comparison was not significant. The 12° cue-target pair differed significantly from the three other pairs ($p<.05$), and these were the only significant differences. The relevant means are shown in Table 3.

 Insert Table 3

Further analysis, however, did reveal a striking dissociation between the manual and saccadic response conditions when the cue and target fell on the same side of the meridian. For manual responses, when the cued location was eccentric to the target location (pairs 1:2 and 4:3; see figure 1), costs were smaller than when the target was eccentric to the cued location (pairs 2:1 and 3:4). This pattern of decreasing costs with increasing cue eccentricity has been previously reported (Shulman et al., 1985, 1986). However, the *reverse* pattern was obtained for saccadic responses: greater costs were obtained when the target was eccentric to the cue. This dissociation was shown by all subjects and the interaction of response condition and cue eccentricity was statistically reliable [$F(1,4)=17.7$; $p<.02$]. Figure 2 presents the relevant costs.

Insert Figure 2

Discussion

The premotor hypothesis predicts that the costs observed for manual responses should parallel those obtained for saccades. Although the overall validity effect was equivalent in the two conditions, the relative eccentricity of the cue and target produced significantly different cost patterns. For manual responses, cue-target pairs 1-2 and 4-3 were associated with greater costs than pairs 2-1 and 3-4. Shulman, Sheehy, and Wilson (1986) provide evidence suggesting this effect is due to cue eccentricity *per se* rather than greater efficiency in shifting attention toward than away from the fovea. However, the opposite pattern emerged for saccades, with cue-target pairs 2-1 and 3-4 producing significantly larger costs than pairs 1-2 and 4-3. In this response condition subjects are likely to preprogram a saccade to the cued location. The obtained pattern of costs, therefore, suggests that subjects are more efficient at increasing than decreasing the amplitude of a prepared saccade. Using similar eccentricities and target displacements in a double step paradigm, Komoda et al. (1973) also found evidence that lengthening a saccade is performed more efficiently than shortening a saccade (however, see Findlay & Harris, 1984).

The essential point is that a significant divergence in the pattern of saccadic and manual costs was observed. This outcome is inconsistent with the premotor hypothesis of attentional orienting. In addition, contrary to the results of Rizzolatti et al. (1987), invalid cue-target pairs on the same side of the vertical meridian produced costs equivalent to pairs on opposite sides of the meridian. This was the case for both response conditions. It is important to note, however, that Rizzolatti et al. (1987) used central precues. While some investigators have reported meridian effects with

peripheral precues (e.g. Hughes & Zimba, 1985), other investigators have also failed to find them (Egley & Homa, 1991). Thus, the absence of a meridian effect in the present experiment could be due to the use of peripheral precues. Experiment 2 used central precues to evaluate this possibility.

EXPERIMENT 2

Method

Stimulus display. The stimulus display was identical to that used in the first experiment with the exception of the precue. Instead of the peripheral cue, a single or double arrow was positioned $.5^\circ$ directly above the fixation point for 300 msec. Arrow direction indicated the likely side of the target. Double arrows indicated an outer location and a single arrow indicated an inner location.

Apparatus and Procedure. The apparatus and procedure were identical to those used in Experiment 1.

Subjects. Six volunteers with normal or corrected vision were paid for their participation.

Results and Discussion

The effect of cue validity on response latency was highly significant ($F(1,5)=98.02$; $p<.0004$; see Table 2). Invalid trials produced an overall cost of 43 msec in both response conditions. As in Experiment 1, an ANOVA was used to evaluate the effects of the four types of invalid cue-target pairs (4° same hemifield, 4° opposite hemifield, 8° opposite hemifield and 12° opposite hemifield) on the two response conditions. This analysis indicated a highly significant effect of invalid cue-target pair [$F(3,15)=18.23$; $p<.0001$] and no effect of response condition. In contrast to Experiment 1, a Newman-Keuls test indicated that the costs associated with each of the opposite field conditions, including the 4° condition, was significantly greater than

costs in the 4° same field condition (see Table 4). The costs for the three opposite field conditions tended to increase with increasing retinal distance between cue and target, but these differences were not significant. A final ANOVA evaluated the effects of the relative eccentricity of the cue and target within each hemifield. Contrary to the first experiment, there was no difference between the 1-2/4-3 pairs and the 2-1/3-4 pairs for either response system (see Table 5). Thus in both response conditions the relative eccentricity of the cue had no effect on the magnitude of costs.

 Insert Tables 4 and 5

The above analyses indicate that with a central cue, in accord with Rizzolatti et al., costs are significantly greater when cue and target occur on opposite sides of the vertical meridian than when they occur on the same side. This was the case for both manual and saccadic responses.

GENERAL DISCUSSION

The present experiments suggest that the relationship between oculomotor programming and attentional orienting depends on the nature of the precue. With peripheral cues the vertical meridian had no effect on costs for either response system (cf. Egly & Homa, 1991) and the relative eccentricity of the cue and target had opposite effects on saccadic and manual responses. This dissociation is inconsistent with the premotor hypothesis, and suggests that attentional orienting and oculomotor programming may be independent processes. A different picture emerges for central cues. Both saccadic and manual responses are associated with greater costs when the cue and target occur on opposite sides of the vertical meridian than when they

occur on the same side. The absence of any dissociations between the response conditions and the presence of a meridian effect in both conditions are consistent with the premotor hypothesis. While our results do not specifically demonstrate a dependence of the attention system on oculomotor programs, they allow for a stronger coupling of these systems in response to central than peripheral precues.

Figure 3 represents three possible ways that precuing effects might operate with saccades and manual responses. These hypothetical schemes all assume that for saccadic responses, reprogramming time contributes in part to the latency difference between valid and invalid trials (e.g. Abrams & Jonides, 1989). The schemes also include a preliminary detection process which precedes attention reorienting or saccadic reprogramming. Considerable debate surrounds the idea of a preliminary detection stage that can initiate reorienting but cannot support a manual detection response (for discussions see Egly & Homa, 1991; Hawkins, Shatto & Richardson, 1988; Hughes, 1984; Rizzolatti et al., 1987). In the case of saccadic reprogramming, however, preliminary detection seems reasonable since modifying the program clearly requires prior specification of the target coordinates. Furthermore in a recent analysis of choice manual response times, Egly and Homa (1991) account for valid-invalid latency differences by a two-stage process that includes a delay in target detection time in addition to a subsequent movement or reorienting time. According to this view, preliminary detection time is more efficient on valid than on invalid trials. The present analyses make the simplifying assumption that any delay in preliminary detection time on invalid trials contributes equally to manual and saccadic responses¹. Thus all three hypothetical schemes attribute any differences between manual and saccadic conditions to reorienting time, reprogramming time or both.

¹ We acknowledge that the following analysis depends on the veracity of this assumption. We note that for simple detection responses in the absence of precues, Hughes and Kelsey (1984) found that saccadic latencies were less dependent on target luminance than manual latencies. However, these differences were only evident at near threshold levels. In the present study target luminances were well above threshold.

Insert Figure 3

Hypothesis A proposes that attention must be oriented toward the target location before a response can be initiated (Posner, 1980). Thus, reorienting time contributes to costs produced by invalid precues for both manual and saccadic responses. However, saccadic costs also include reprogramming time, so this scheme predicts that saccadic costs should be greater than manual costs. In the present experiments saccadic and manual costs were equivalent with both central and peripheral precues. Thus, there is no simple way in which hypothesis A can account for present data.

According to hypothesis B, saccadic reprogramming and attentional reorienting are the same process. This is the scheme proposed by the premotor hypothesis. This hypothesis predicts that in addition to equivalent overall costs, the *patterns* of costs produced by the varying the spatial relation between the cue and target should be the same for manual and saccadic responses. Therefore, hypothesis B is consistent with the results obtained with central precues but cannot account for the data obtained with peripheral cues.

According to hypothesis C, attentional orienting and saccadic programming are separate processes that occur in parallel. This scheme can accommodate the different patterns of saccadic and manual costs that emerge with peripheral precues, but permits equivalent overall costs for the two responses. This view suggests that with peripheral precues, attentional orienting and oculomotor programming may be independent processes. Attentional reorienting is not required for oculomotor responses².

While hypothesis C is also consistent with the effects of central precues, the meridian effects observed for manual and saccadic responses would then have to be attributed to different processes. Hypothesis B allows for the possibility that these effects have a common basis. However, the results obtained with central precues are also consistent with the idea that attentional orienting and saccadic programming are separate, but depend on a prior process which is the source of the meridian effects. The present data do not permit us to decide among these alternatives, but at least one recent finding argues against hypothesis B. Rafal, Calabresi, Brennan, & Scialtoja (1989) report that inhibition of return occurs in response to central precues only if observers are explicitly instructed to prepare a saccade. Covert orienting to symbolic cues is not sufficient to produce inhibition of return. This outcome suggests that saccades are not typically prepared during covert orienting to central cues.

In sum, hypotheses B and C can account for the data obtained with central precues, but only hypothesis C is able to account for the effect of peripheral precues. It is clear that neither experiment can be explained simply by a scheme such as A, in which saccadic programming depends on prior attentional orienting.

Shepherd and Muller (1989) also report evidence suggesting that the premotor hypothesis may be more compatible with the effects of central than peripheral precues. Using a manual response task, they compared the effects of each of these cue types on the magnitude of attentional costs and benefits at various cue-target SOA's. Peripheral cues were associated with a narrow focus that was maintained across all SOA's. In contrast, central cues produced an initially broad distribution of attention on the cued side which became more narrowly focused on the cued location with increasing SOA's. They suggest that the central precue results are consistent with the premotor

² We note that, with respect to hypothesis C, if we assume that manual responses do not require attentional reorienting but maintain the assumption of a common detection stage, we would expect manual costs to be less than saccadic costs. This prediction is clearly discomforted by the present data.

hypothesis in that the initially broad focus could reflect the programming of saccade direction prior to the specification of saccadic amplitude (Rizzolatti et al., 1987).

Sudden peripheral events seem to elicit a unified orienting response. The suggestion that peripheral precues may activate independent oculomotor and attentional orienting systems may, therefore, seem counter-intuitive. However, the finding that peripheral stimuli can elicit reflexive saccades and capture attention automatically (Yantis & Jonides, 1984; Muller & Rabbitt, 1989) need not imply that these responses are mediated by a common system. A peripheral cue directly specifies a spatial location, producing multiple sensory representations of that location within the visual system. A representation at the level of the superior colliculus could be used to program a saccade while a parietal representation could mediate attentional responses.

The closer coupling of saccadic and manual responses suggested by the central precues effects may be understood as follows. The computations associated with central precues are likely to be more complex than with peripheral cues, since the expected target location must be derived from an interpretation of the precue. It seems reasonable that instead of duplicating these computations the attentional and saccadic subsystems make use of a common representation. This could produce a stronger association between these subsystems. In addition, effective voluntary control of orienting may require that the orienting subsystems are directed toward a common goal. This could be achieved by having one subsystem assume control over the other, or by having all subsystems follow a common executive (see also Shepherd, Findlay & Hockey, 1986).

The present findings suggest that the nature of saccadic programs, as well as attentional responses, may be influenced by the type of precue. In a recent study of saccade preparation, Abrams and Jonides (1988) found a similar effect. They report that for voluntary saccades, amplitude and direction may be computed separately

whereas more reflexive saccades appeared to be programmed holistically. The present finding of a meridian effect only for saccades to central cues may, therefore, reflect the independent specification of direction and amplitude for voluntary, cortically mediated saccades, whereas saccades prepared to peripheral precues may reflect more holistic programs mediated by the colliculus (cf. Abrams & Jonides, 1988).

The manner in which saccade direction and amplitude are computed is a matter of on-going debate (Abrams & Jonides, 1988; Becker, 1989). The work of Abrams and Jonides suggests no difference in the time to program the initial direction or amplitude of a saccade. The time it takes to *modify* these parameters may differ, however, and it is on this possibility that the premotor account of the meridian effects depends. The results from Experiment 2 are consistent with the possibility that amplitude can be modified more rapidly than saccadic direction.

In summary, the premotor hypothesis cannot readily account for the effects obtained with peripheral precues. A model in which attentional orienting and saccadic programming are separate processes that proceed in parallel is more consistent with the effects of these cues. On the other hand, the results obtained with central cues are consistent with the premotor hypothesis, although they do not demonstrate that attentional orienting depends on oculomotor programs. However, the results do suggest a closer coupling of attention and oculomotor processes in response to central than peripheral precues, and indicate that the vertical meridian affects the time course of overt and covert orienting only with central cues.

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Target Locations

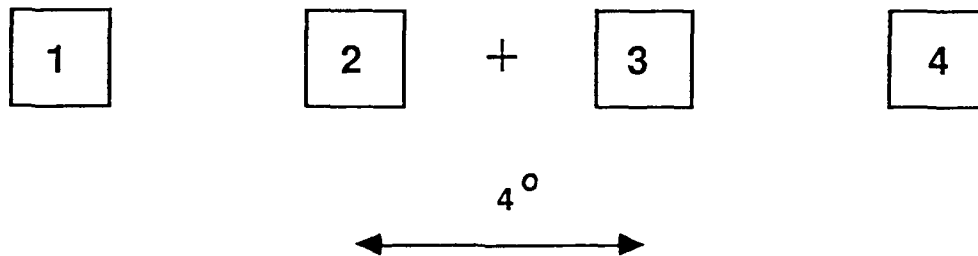


Fig. 1. Numbers in the boxes are the numeric designations used in tables and the text, and did not actually appear.

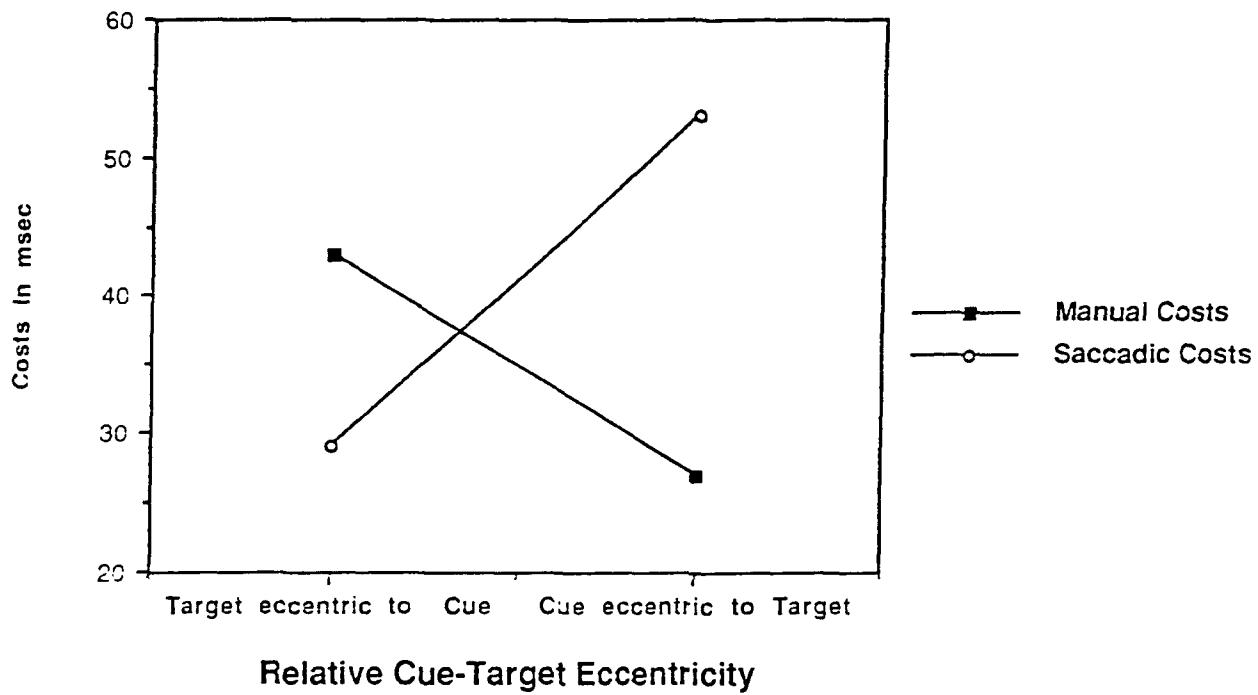
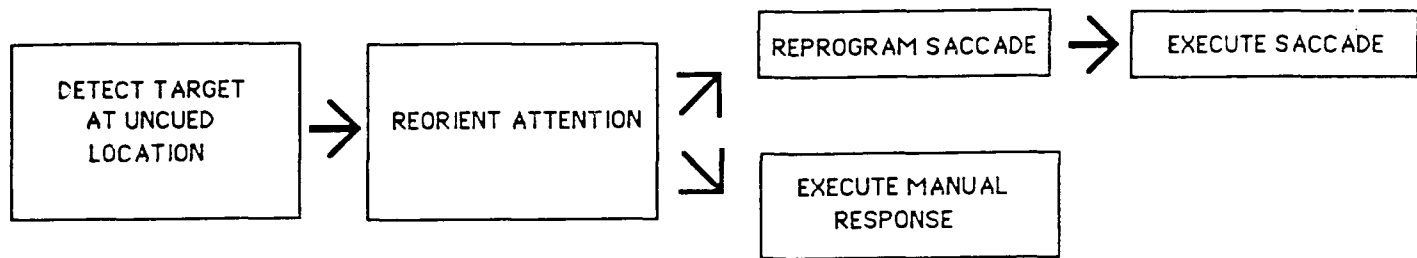


Figure 2: Costs as a function of relative cue-target eccentricity for Experiment 1

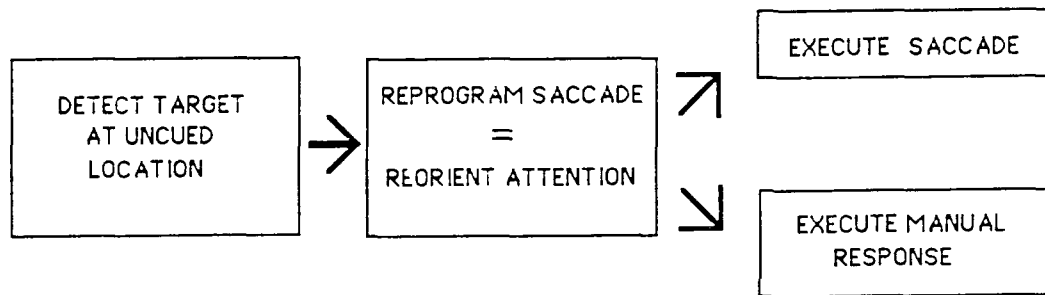
Figure Caption

Figure 3. Schematic diagrams of three possible accounts of precuing effects for manual and saccadic responses.

A.



B.



C.

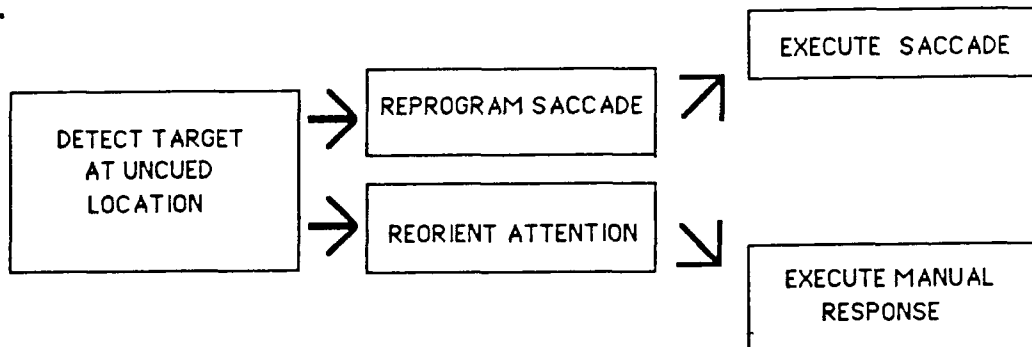


TABLE 1: Average Latency for Manual and Saccadic Responses for
All Cue-Target Pairs

Cue:Target Pair	Experiment 1: Peripheral Cue		Experiment 2: Central Cue	
	Manual	Saccadic	Manual	Saccadic
1:1	280	292	270	263
2:1	339	322	304	284
3:1	334	321	327	320
4:1	343	328	326	325
2:2	272	294	264	278
1:2	301	340	293	307
3:2	311	324	310	320
4:2	294	330	324	324
3:3	274	282	265	281
1:3	305	331	312	335
2:3	312	319	309	315
4:3	300	343	283	301
4:4	285	283	275	272
1:4	335	338	331	337
2:4	330	331	330	333
3:4	312	312	286	300

TABLE 2: Average Latencies and Standard Deviations for Valid and Invalid Cues for Manual and Saccadic Responses in Experiments 1 and 2.

	Experiment 1		Experiment 2	
	MANUAL	SACCADIC	MANUAL	SACCADIC
Valid	278 (16.5)	288 (42.4)	269 (27.0)	274 (21.4)
Invalid	318 (23.9)	328 (51.2)	311 (31.8)	317 (32.7)
NET COST	40	40	43	43

TABLE 3: Average costs and standard deviations of cue-target pairs for each response condition in Experiment 1.

	4° same	4° opposite	8° opposite	12° opposite
Manual Response	35.2 (9.3)	38.4 (6.3)	37.9 (12.2)	56.6 (11.9)
Saccadic Response	41.3 (10.2)	33.4 (14.8)	40.3 (14.6)	45.2 (11.8)

TABLE 4: Average costs and standard deviations of cue-target pairs for each response condition in Experiment 2.

	4° same	4° opposite	8° opposite	12° opposite
Manual Response	22.8 (11.8)	44.9 (9.5)	54.4 (12.9)	58.1 (16.5)
Saccadic Response	23.2 (14.9)	37.9 (19.5)	54.3 (22.1)	60.7 (21.8)

TABLE 5: Average costs and standard deviations for within field trials as a function of relative cue-target eccentricity and response condition for Experiment 2.

	Cue eccentric to target (pairs 1:2, 4:3)	Target eccentric to cue (pairs 2:1; 3:4)
Manual Responses	31.1 (16.2)	30.5 (14.6)
Saccadic Responses	25.6 (12.8)	25.5 (15.4)

SECTION 3

GENETIC INFLUENCES ON THE GROSS MORPHOLOGY
OF HUMAN CEREBRAL CORTEX

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Abstract

Using a new computerized method of ascertaining regional cortical surface areas from 3D reconstructions of MR scans of the the human brain, we examined the similarities that may exist in the left and right hemispheres of monozygotic twins as compared to unrelated controls. Marked differences were observed between the hemispheres with the left hemisphere being far more specified than the right hemisphere.

The convolutions of the cerebral hemispheres show wide variation in size and shape. This variation presumably reflects regional differences in cell count and/or synaptic density. At present, little is known about genetic influences on normal size and shape of cortical areas, and whether normal variations in cortical morphometry might mirror normal individual differences in psychological processes.

One possible avenue for examining how genes influence cortical morphometry is to examine the brains of monozygotic twins using magnetic resonance imaging (MR). Evidence for less variance within twin pairs as compared to the variance among unrelated pairs would raise the possibility that neuroanatomical similarities might underlie the psychological and neurophysiological similarities known to exist in monozygotic twins (1-9). Our previous quantitative *in vivo* analysis of the corpus callosum in monozygotic twins demonstrated that mid-sagittal area and shape are more similar within twin pairs than within unrelated pairings (10). This result encouraged us to analyze cortical morphometry in monozygotic twins using computer-generated reconstructions of the cortical surface recently developed in our laboratory (figure 1) (11).

Five pairs of female monozygotic twins were studied. Monozygosity was determined by analyzing 9 red blood cell surface markers and by a standardized questionnaire (12,13). All subjects were righthanded (Edinburgh Laterality Index 74 - 100) (14). The brain of each subject was scanned using a Siemens 1.0 Tesla Magnetom MR system. Contiguous 3mm thick T1-weighted sections were imaged in the coronal plane. Individual gyri and additional regions of interest were labelled in accordance with the coronal atlases of Matsui and Hirano (1978) (15) and Krieg (1963) (16).

Percent surface area was computed by dividing absolute regional surface area (rSA) by lobar SA.

Two hypotheses were examined. First, we examined whether there was a difference in rSA among the five pairs of twins using a repeated measures ANOVA. Subsequently we examined whether the variability of any particular rSA was lower for related twins as compared to unrelated pairs. Bartlett's test for the homogeneity of variances was used to test this hypothesis.

The ANOVA revealed that for each hemisphere there is no difference among unrelated pairs or between related twins. At the same time, the 27 ROI's differ significantly in rSA ($p = .0001$), which had been expected. Finally, the difference in measurements between related twins does not depend on the region; however, for the left hemisphere, pairs of unrelated twins differ more for some regions than for others ($p = .0001$). This last result suggested that reduced variability in rSA of related twins as compared to unrelated pairs might be found in certain left hemisphere ROI's. This prompted us to analyze the data on a per region basis.

Results of Bartlett's test to compare the variation of unrelated pairs vs. variation of related twins for each region are summarized in Table 1. Using this analysis, we found that unrelated pair variances exceeded related twin variances for 15 regions in the left hemisphere and 4 regions in the right hemisphere.

These data indicate that the development of left hemisphere structures is under considerable more genetic control than is the right hemisphere. Given the dominance of the left hemisphere in language and in problem-solving ability, it is interesting to speculate that some of the cortical areas showing less variance may reflect a structural basis of the

similarities in cognitive skills and personality that are commonly noted in twins (1-9, 17-23). In view of previous clinico-pathologic correlations (24), the finding of reduced variance in the pars opercularis of the left inferior frontal gyrus and in the left insula as well as the trend for the pars triangularis suggests that development of the speech area is under greater genetic control than homologous regions in the right hemisphere. Studies of split-brain patients as well as follow-studies of patients with middle cerebral artery stroke indicate that speech is the most highly lateralized of all human brain functions (25).

It is also of interest to consider why the ROI's encompassing the left cortical somatosensory and both visual systems, which are tied to body surface parameters, have reduced variance in twin pairs. Current theories of cortical development point to the possibility that afferent specification from neurons representing the body surface in the thalamus specify otherwise omnipotent cells underneath the cortical mantle (26). Since the topology of the body is highly similar in twins, it might follow the brain areas representing these sensory spheres of information would be more alike. In the present study this was the case for the left somatosensory representation. It is conceivable that in the domain of somatosensory processes that the phenomenon of handedness might exaggerate the determination of the right body, leaving the left half body surface area more unstructured and thus less specific in its specification of cortical areas. In the visual domain, important considerations for the final connectivity of visual cortex heavily relies on the inter-ocular distance that are crucial for establishing normal stereopsis (27). Again, the similar physiognomy of twins would be consistent with greater

similarity of such measures and argue under the afferent specification model for more similar visual association areas.

Neuropsychological analysis of patients with focal lesions have emphasized the importance of the structures of the left hemisphere for most measures of intelligence. For example, lesions to frontal lobe structures can seriously disrupt cognitive processes, such as modifying the capacity to switch categorical sets, verbal fluency, story comprehension, and problem solving (28). Interestingly, lesions to frontal areas in the monkey are now thought to disrupt working memory in addition to the well-known disruptions on delayed response and other tasks (29). In this light it has recently been shown that variations in working memory capacity in the human correlate with individual variations seen in reading comprehension (30). Finding reduced variance in frontal lobe structures in the left hemisphere is consistent with the view that more similar anatomical organization may reflect more similar psychological capacities.

This same line of reasoning is also supported by the careful examination of split-brain patients where it has been shown that the left hemisphere is far superior in problem solving (31). These same studies have also shown only the left hemisphere can make causal inference and apprehend complex linguistic constructions. In short the left hemisphere is clearly specialized for cognitive operations in the normal brain. Related studies have also shown that performance scores on measures of intelligence are lowered following brain bisection, presumably because the neural structures associated with these activities are no longer able to contribute their processing capacities to the left hemisphere.

In summary, the present findings are consistent with the view that the previously observed similarities in psychological and physiological functions in monozygotic twins may correlate with the reduced variance seen in major cortical structures, particularly those of the left hemisphere.

Table 1

	LEFT HEMISPHERE			RIGHT HEMISPHERE		
<u>Region of Interest</u>	<u>Pair Var.</u>	<u>F</u>	<u>p</u>	<u>Pair Var.</u>	<u>F</u>	<u>p</u>
<u>Frontal Lobe</u>						
Frontal Superior (Fs)	1.61	0.65	.63	1.99	0.36	.84
Frontal Middle (Fm)	25.91	10.55	<.0001	3.48	0.62	.65
Pars orbitalis (Fiorb)	1.64	0.67	.62	2.00	0.36	.84
Pars triangularis (Fit)	5.11	2.08	.11	3.95	0.71	.59
Pars opercularis (Fiop)	12.73	5.18	.003	13.45	2.41	.07
Precentral (Ca)	5.92	2.41	.07	9.68	1.74	.17
Orbitofrontal (Or)	7.05	2.87	.04	3.29	0.59	.67
Rectal (R)	0.54	0.22	.92	1.20	0.22	.93
<u>Temporal Lobe</u>						
Temporal Superior (Ts)	4.27	1.74	.17	6.41	1.15	.36
Temporal Middle (Tm)	3.58	1.46	.24	3.81	0.68	.61
Temporal Inferior (Ti)	8.27	3.37	.02	2.57	0.46	.76
Uncus (U)	2.96	1.20	.33	1.44	0.26	.90
Heschl's (Hg)	0.63	0.25	.90	1.54	0.28	.89
Fusiform (Fus)	4.82	1.96	.13	7.07	1.27	.31
Hippocampus (H)	1.89	0.77	.56	0.63	0.11	.98
Amygdala (A)	1.04	0.42	.79	0.22	0.04	1.00
<u>Parietal Lobe</u>						
Post Central (Cp)	24.41	9.94	<.0001	8.53	1.53	.22
Supramarginal (Sm)	12.29	5.00	.004	6.03	1.08	.39
Angular (Ang)	18.80	7.65	.006	23.94	4.30	.008
Superior Parietal (Ps)	22.77	9.27	<.0001	33.83	6.07	.001
Precuneus (Pc)	12.61	5.16	.003	5.17	0.93	.46
<u>Occipital Lobe</u>						
Lingual (Lg)	15.25	6.21	.001	1.76	0.32	.86
Cuneus (Cu)	12.61	5.13	.004	11.39	2.04	.12
Lateral Occipital (LO)	110.17	44.84	<.0001	32.65	5.86	.002
<u>Other</u>						
Cingulate (Ci)	41.39	16.85	<.0001	37.69	6.76	.0007
Basal Forebrain (BF)	0.19	0.08	.99	0.22	0.04	1.00
Insula	36.55	14.88	<.0001	5.42	0.97	.44

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Figure 1. Three dimensional reconstruction of a left hemisphere shell of the human brain using surface triangulation. This mathematical model consists of a multitude of small triangles which are connected in three-space so as to compose a smooth yet convoluted mesh based upon contours extracted from serial coronal MR scans. A summation of the areas of the triangles that compose the model provides a basis for an approximation of true cortical surface area (both intrasulcal and superficial). In this method a "skin" is draped over the space (mesh) between adjacent contours in a manner which does not overestimate overall surface area but does provide a smooth interpolation between the contours (32, 33).