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GENERATING AND MEASURING INHIBITION OF RETURN

by

Tracy L. Taylor

Submitted in partial fulfillment of the requirements
for the degree of Doctor of Philosophy

at

Dalhousie University
Halifax, Nova Scotia
October, 1997

DALHOUSIE UNIVERSITY

FACULTY OF GRADUATE STUDIES

The undersigned hereby certify that they have read and recommend to the Faculty of Graduate Studies for acceptance a thesis entitled "Generating and Measuring Inhibition of Return"

by Tracy L. Taylor

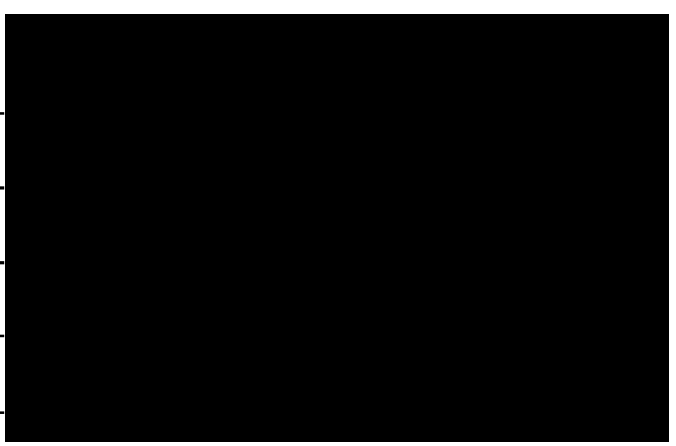
in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

Dated: October 20, 1997

External Examiner

Research Supervisor

Examining Committee



DALHOUSIE UNIVERSITY

DATE: *October 20, 1997*

AUTHOR: *Tracy Lynn Taylor*

TITLE: *Generating and Measuring Inhibition of Return*

DEPARTMENT OR SCHOOL: *Psychology*

DEGREE: *Ph.D.* CONVOCATION: *May* YEAR: *1998*

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ABSTRACT

Unpredictive visual transient cues have a biphasic effect on response times (RTs) to peripheral onset targets. At relatively short (e.g., 150 msec) cue-target stimulus onset asynchronies (SOAs), RTs to targets at cued versus uncued locations are facilitated, whereas at relatively long SOAs (e.g., beyond 300 msec), they are inhibited. The literature converges on the view that this latter, inhibitory, effect - referred to as Inhibition of Return (IOR; Posner & Cohen, 1984) - is a motor response bias that is generated by the activation of an oculomotor program to fixate the cue. This view is challenged by the present study, which systematically manipulated the stimulus/response conditions used to generate and measure IOR.

On each trial, subjects fixated the centre of three horizontally-arranged outline boxes and then were presented with a 300 msec first signal (S1) that was followed at a 1000 msec SOA by a second signal (S2). Each of these signals could be an onset in the periphery (exogenous) or else an arrow at fixation (endogenous). In different experiments, subjects made no response, a manual localization response, or a saccadic response to S1, and a manual localization or saccadic response to S2. The nature of S1 (exogenous, endogenous), S2 (exogenous, endogenous), and the relative locations signalled by S1 and S2 (different, same) were combined factorially for each of the S1-S2 response combinations that comprised Experiments 1-6, respectively: No Response-Manual, Manual-Manual, Saccadic-Manual, No Response-Saccadic, Manual-Saccadic, and Saccadic-Saccadic. IOR was measured as the difference in RT to S2 when S1 and S2 signalled the same versus different locations.

Two inhibitory processes were revealed. The first was a visuo-motor inhibition that operated when the saccadic system was not explicitly engaged by S1/S2 response demands. This effect appears to reflect inhibited exogenous attentional orienting to S2 due to prior covert attentional orienting to S1. The second was a motor inhibition that is revealed as a response bias that appears to be generated by the implicit or explicit activation of a motor response to S1. Despite the operation of two inhibitory processes, the present findings are consistent with the possibility that both visuo-motor and motor inhibition may be based on a common mechanism within the superior colliculus but that generating and accessing the neural representation of IOR depends on explicit engagement of the saccadic system.

LIST OF ABBREVIATIONS

ANOVA	analysis of variance
Ant.	anticipations
D	different spatial location
Dir. Err.	directional error
End	endogenous
Exo	exogenous
IOR	inhibition of return
LED	light-emitting diode
min	minutes
M-M	manual-manual
M-S	manual-saccadic
msec	milliseconds
N-M	no response-manual
N-S	no response-saccadic
RSI	response-stimulus asynchrony
RT	reaction time
S	same spatial location
S1	first signal
S2	second signal
SC	superior colliculus
S-M	saccadic-manual
SOA	stimulus onset asynchrony
S-S	saccadic-saccadic
%	percent

ACKNOWLEDGEMENTS

Words don't seem big enough to hold all the love and gratitude they are meant to convey. My supervisor, Ray Klein, has been a well-spring of enthusiasm, acceptance, warmth, and intellectual stimulation. Whenever I have fallen down - academically or personally - he has helped me up. I could never express to Ray all that he means to me. I trust he will understand when I say that he is my Donald Hebb. My committee members, Ray, Bruce Earhard, and Paddy McMullen, were wonderful in accommodating my compulsive adherence to self-imposed deadlines and improved the quality of this work through their thoughtful consideration of my writing and thinking. Bruce's allusion to Camus is the reason I have more than a one-sentence thesis (alas, but no Pulitzer Prize!), and Paddy's support through the past 5 years has meant more to me than she probably realizes. I would like to thank Tom Carr for making a whirlwind trip to Halifax during what was clearly a very busy time for him. He was, without exception, an incredible external examiner - critical yet non-confrontational. He made my defence more fun and exciting that I'd dared hope it would be and his critique of my work was both incisive and thought-provoking. In addition to my examining committee, I would like to thank Doug Munoz, Dennis Phillips, Vin LoLordo, and Richard Brown. At a time when I had forgotten where my research "fit in", Doug allowed me to visit his lab and rekindled my flagging interest in cognition generally and sparked my interest in cognitive neuroscience. When I had forgotten, Dennis reminded me of the joy in science; when I needed a friend, I could always count on him to be there. My tremendous admiration for Dennis is equalled only by my affection for him. Vin LoLordo has been a quiet source of inspiration to me - he has achieved an enviable balance in his life and,

through that, has emphasized to me that there is more to life than eye movements. To Richard Brown I owe a special thanks - for chiseling the chip on my shoulder and awakening me to the walls I had built around myself. He is the reason that I have the following friends to thank for their support...John Christie and Billy Schmidt deserve medals for their endless patience over the years and many thanks go to them and to Mark Ryer for their technical help. Billy, Kelly Forbes, Peter Istvan, Brad Frankland, Bruce McCandliss, and Lew Stelmach have extended their hands in friendship and helped me over some very rocky roads. David Rasmussen found me at the end of one of those roads, brushed me off, and kept me from rebuilding my walls. Bruno Losier has offered me his valued friendship and, in so doing, has taught me the value of a warm heart and an open mind. Mike King has also taught me that lesson, in addition to reviving my interest in once-loved pursuits (although, perhaps in that, Robert Browning also had a hand!) My dance partner and friend, Alan MacDonald, will understand my heart best if I thank him for always remembering my birthday. Michael MacGregor's unconditional love and friendship continue to be foundational for me. Although our lives have taken different paths, he is - and will always be - an important part of who I am. My life is richer for his love and also for the love of two Jeffs. Jeff "Jay" Jones has shared life's teeter-totter with me, been my strength, and given me balance. As for Jeff Hamm, there are no words to express my feelings - he has been my catcher in the rye...I am leaving Dalhousie a very different person than when I arrived - more confident, hopeful, open, warm, caring. There are so many people that have touched me in such profound ways that 'thank-you' sounds hollow. I will always have a place in my heart for everyone who has made my time here so remarkable and I will look back on these years with both a wistful smile and a tear.

GENERATING AND MEASURING INHIBITION OF RETURN

INHIBITION OF RETURN

Using a variant of a spatial orienting paradigm (cf. Posner, 1980), Posner and Cohen (1984) observed that peripheral luminance changes have a biphasic effect on manual reaction times (RTs) to detect subsequent luminance onset targets. As depicted schematically in Figure 1, Posner and Cohen (1984) presented subjects with three horizontally-aligned outline boxes centred on a cathode ray tube and separated by 8 degrees visual angle. At the beginning of a trial, one of the peripheral boxes was cued via a 150 msec brightening. This cue was unpredictive of target location. Indeed, regardless of the location of the peripheral brightening, targets appeared with 60% probability in the fixated centre location; with 10% probability at each of the two peripheral locations; and, were withheld with 20% probability (catch trials).

Combined with eye movement monitoring, these predetermined spatial probabilities encouraged subjects to maintain both their eyes and their attention at centre. Despite incentive to focus attention at the fixated location, however, RTs to peripheral targets were impacted according to their spatial congruity and temporal contiguity with the cue. In particular, at short (i.e., less than approximately 150 msec) cue-target stimulus onset asynchronies (SOAs), RTs were facilitated to targets that appeared at the same versus a different location as the cue; at long (i.e., in excess of approximately 500 msec) cue-target SOAs, this trend reversed and RTs were inhibited to targets that appeared at the same versus a different location as the cue[1]. An idealized biphasic effect is depicted in Figure 1 (panel B).

The facilitatory component of the observed biphasic effect of a visual

POSNER AND COHEN'S (1984) METHODS AND IDEALIZED RESULTS

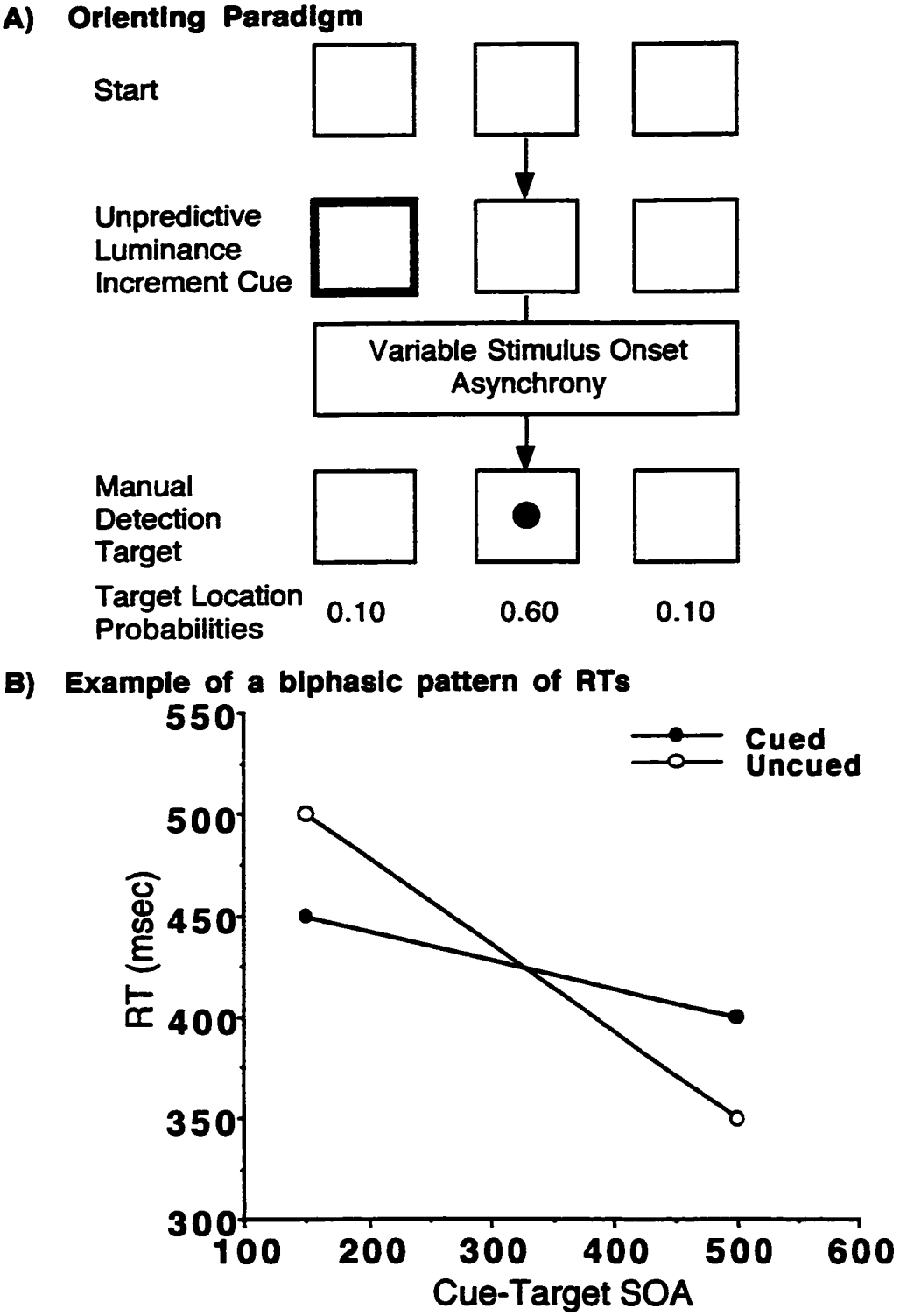

Figure 1. Panel A shows Posner & Cohen's (1984) use of the orienting paradigm. Panel B shows the characteristic (idealized) biphasic pattern of RTs, as a function of cue-target SOA and relative locations.

transient cue is due to covert orienting of visuo-spatial attention. Distinguished from overt orienting - which refers to the alignment of peripheral visual receptors with the source of visual input (e.g., through saccadic eye movements) - covert orienting refers to the allocation of attentional resources to a spatial location or object independently of changes in gaze direction. Such covert orienting can be accomplished by the operation of two distinct (e.g., Briand & Klein, 1987; Klein, 1990, 1994; Klein, Kingstone, & Pontefract, 1992 for a review; in contrast with Jonides, 1980, 1981; Muller & Rabbitt, 1989) but interacting (Theewes, 1991) control systems: One endogenous, the other exogenous. Endogenous covert orienting is accomplished via task demands, stimulus probabilities, or symbolic cues that encourage top-down allocation of attentional resources to a location (cf. Posner, 1980), object (cf. Duncan, 1984), or to the development of a non-spatial expectancy (cf. Kingstone & Klein, 1991; Klein & Hansen, 1990; Lambert & Hockey, 1986). Exogenous covert orienting, on the other hand, is achieved automatically, via bottom-up activation of transient visual channels (cf. Nakayama & Mackeben, 1989)[2] that code the location of an abrupt luminance change.

Whether allocated endogenously or exogenously, the locus of attention is inferred through chronometric analysis of performance. With respect to spatial orienting, this analysis presumes that attention has been oriented in accordance with a cue when performance (e.g., RT) is relatively improved (benefits) for responses to targets that appear at a cued location and relatively impaired (costs) for responses to targets that appear at an uncued location (cf. Posner, 1980; Posner & Snyder, 1975)[3].

Given this distinction between endogenous and exogenous control, the RT facilitation observed by Posner and Cohen (1984) at early cue-target SOAs

reflects exogenous capture of attention by the visual transient cue. That this early facilitation is replaced by a later inhibition has been said to depend on the withdrawal of attention back to fixation (although see Berger, 1992, as cited in Rafal & Henik, 1994; and Maylor & Hockey's, 1987, and Possamai's, 1986, demonstrations of IOR at fixation) following initial exogenous capture by the peripheral cue.

In Posner and Cohen's (1984) original study, subjects were encouraged to endogenously maintain attentional resources at the centre location due to the high probability of target occurrence at that location. However, subsequent studies have found a similar biphasic effect of luminance cues even when target probabilities are equated across location. These studies rely on subjects' endogenous motivation to maintain attention centrally in the absence of incentive to allocate resources elsewhere (cf. Possamai, 1986) and/or interpose a centrally-presented luminance transient in order to explicitly summon the return of exogenous attention to fixation (cf. Posner & Cohen, 1984). The modal paradigm in the literature uses this latter method to cue attention back to centre before presenting a target with equal probability at one of (usually) two peripheral locations (cf. Posner & Cohen, 1984).

Regardless of whether attention is drawn back to centre endogenously via task demands and/or exogenously via a luminance change at fixation, inhibitory after-effects of the prior cue are observed at relatively long cue-target intervals. This finding of an inhibitory after-effect following the withdrawal of attention from a peripheral location, however, occurs only when the initial orienting is accomplished without endogenous control: When Posner and Cohen (1984) attempted to replicate their pattern of biphasic results using an endogenous cue, they failed to find the signature cross-over interaction of SOA and cue-target spatial congruity.

In this attempted replication, Posner and Cohen (1984; see also Rafal, Calabresi, Brennan, & Sciolto, 1989) presented subjects with a central symbolic cue at fixation (a directional arrow). On half of the trials, the arrow cue was followed after 450 msec by a target whose location was predicted with 80% validity by the direction indicated by the cue. On the other half of the trials, the centre box brightened in order to summon attention exogenously, and target probabilities were re-established with 60% probability at centre and 20% at each of the two peripheral locations (thereby canceling the predictive validity of the cue). There was no evidence of inhibitory consequences: Facilitatory effects due to endogenous covert orienting were observed even out to SOAs of 1250 msec.

Taking into account the conditions under which an inhibitory effect is observed, Posner, Rafal, Choate, and Vaughan (1985) coined the term "inhibition of return" (IOR) to refer to this component of the biphasic pattern. This terminology derives from the belief that attention is drawn reflexively to the location of the luminance cue (resulting in initial RT facilitation) and that, upon reorienting to fixation under endogenous (target probabilities) and/or exogenous (a visual transient at fixation) control, attention is biased against returning to the previously-cued location (resulting in subsequent RT inhibition).

Although this presumed attentional locus of the inhibitory after-effects has been questioned (e.g., Klein & Taylor, 1994; Rafal et al., 1989), the term IOR has been applied ubiquitously to any pattern of performance data which shows relative deficits at cued versus uncued locations. It is this pattern of inhibited performance at a previously-cued location that is the focus of the current studies. To maintain consistency with the literature, the term IOR is used throughout the following paper; however, this usage is not intended as

a theoretical endorsement of Posner et al.'s (1985) characterization of the inhibitory effect.

Despite the pretheoretical assumptions that are inherent in the IOR terminology and the possible over-reliance on Occam's razor in referring generally to performance deficits at a cued location, in characterizing IOR, Posner et al. (1985) highlight a distinction that is critical to an understanding of the inhibition: The distinction is between what is inhibited by IOR and how that inhibition is generated. As indicated above, in defining the inhibition as IOR, Posner et al. (1985) postulated that it is exogenous covert attention that is inhibited and that this inhibition is a consequence of initial exogenous covert orienting to a peripheral location. By examining what is inhibited and how that inhibition is generated, it will become clear that - contrary to Posner et al.'s (1985) supposition - the phenomenon hereafter referred to as IOR cannot be understood as a unitary attentional effect, either in its measurement or in its genesis.

IOR: WHAT IS INHIBITED?

Since the discovery of IOR, several different views of what is inhibited have been proposed. According to one account (e.g., Posner & Cohen, 1984, *p.* 541), IOR is akin to sensory masking or habituation, wherein stimulation provided by the cue interferes with subsequent target processing by the sensory mosaic. However, this explanation seems unlikely. Even aside from the fact that IOR occurs at cue-target SOAs outside the typical range for masking effects (e.g., Foley & Boynton, 1993) and is a longer-lasting effect (up to 3 sec; Tassinari, Biscaldi, Marzi, & Berlucchi, 1989), if sensory processes were inhibited due to cue presentation, target detection might be expected to suffer at early as well as late SOAs. As such, the fact of early facilitation of

target detection is inconsistent with a sensory masking or habituation effect - unless it is assumed that the early facilitation is due to spatial and/or temporal summation of cue and target luminance.

Noting that Posner and Cohen (1984) obtained facilitation only for SOAs at which target occurrence overlapped with or else was in close temporal proximity to cue presentation, Tassinari, Aglioti, Chelazzi, Peru, and Berlucchi (1994; see also Possamai, 1986) manipulated systematically the temporal relation between cue and target. Utilizing SOAs between 0 and 900 msec, in different experiments, they: 1) Presented cues and targets of short (16 msec) duration; 2) presented cues that, regardless of SOA, outlasted the 16 msec target by a fixed duration of 300 msec; and, 3) replicated Posner and Cohen's (1984) 150 msec cue duration in combination with a 16 msec target presentation. In all cases, the biphasic pattern of results (idealized in Figure 1) was obtained, thereby indicating that the early facilitatory effect likely does not depend on luminance summation of cue and target[4]. This conclusion is bolstered by noting that a biphasic pattern of results is also obtained with peripheral cues that dim rather than brighten (Posner & Cohen, 1984).

Of course, even without local summation of cue/target luminance at early SOAs, it could be argued that IOR reflects a sensory processing deficit and that the failure to observe this deficit at early SOAs is due to a different timecourse for facilitatory and inhibitory effects of the cue. In other words, the cue may result in a sensory deficit that is masked at early SOAs by facilitatory processes (e.g., improved perceptual processing due to the allocation of covert attention). However, this account seems unlikely: Contrary to a low-level visuo-sensory account of IOR effects are three lines of evidence that negate local cue-target interactions at the retina. These lines of evidence, outlined below, are corroborated to the extent that a unitary effect is

represented by the observation of inhibition for ipsilateral cues and targets within and across visual, auditory, and somatosensory modalities (Breau, Mondor, & Milliken, 1995; Reuter-Lorenz, Jha, & Rosenquist, 1996; Reuter-Lorenz & Rosenquist, 1996; Schmidt, 1996a; Spence & Driver, in press, 1997; Tassinari & Berlucchi, 1995; Tassinari & Campara, 1996). If evidence should bear out the supposition that visual, auditory, and somatosensory inhibitory effects stem from the same, supra-modal, mechanism (see Ward, 1994 for a supra-modal and modality specific control of attentional orienting), this would obviously present conclusive evidence against retinal interactions.

The possibility of a supra-modal mechanism notwithstanding, there are data within the visual domain that argue against IOR at the level of the retina. The first line of evidence stems from the observation that IOR shows inter-ocular transfer. Employing a dichoptic viewing methodology, Tassinari and Berlucchi (1993; see also Maylor, 1983, as cited in Maylor & Hockey, 1987) required subjects to view stimuli through goggles that had a red filter over one eye and a green filter over the other. Cues and targets were red or green and, on a trial, did not share the same colour. As such, the goggles filtered the cue to one eye and the target to the other. In spite of the fact that cues and targets were not processed by the same retina, IOR was observed. Moreover, when compared to equivalent conditions run under binocular viewing, the magnitude of the IOR effect was not reduced by the dichoptic viewing procedures (M=37 msec under binocular viewing and M=48 msec under dichoptic viewing; see Tassinari & Berlucchi, 1993, same point RT-opposite point RT in their Tables 1 and 2).

The second line of evidence arguing against slowed retinal processing is that IOR can be measured at locations remote from the cue. Maylor and Hockey (1987) presented subjects with a cue 12 degrees visual angle to the

direct left or right of fixation. This cue was followed after a variable SOA by a target that could occur in any one of 14 positions: Seven positions were on either side of fixation, with three above and three below the centre location that could also serve as cue; the vertical extent of possible target locations was 4.5 degrees visual angle above and below the horizontal meridian. IOR was distributed in a graded but non-linear fashion according to the vertical distance between cue and target within a hemifield (for evidence that the inhibitory gradients do not cross the horizontal or vertical meridia, see Berlucchi, Tassinari, Marzi, & DiStefano, 1989; Tassinari, Aglioti, Chelazzi, Marzi, & Berlucchi, 1987).

Finally, the third line of evidence against retinal cue-target interactions stems from the demonstration that IOR is not coded in retinotopic coordinates. Maylor and Hockey (1987) presented the task illustrated in Figure 2. Subjects began with their eyes at the upper fixation stimulus and a cue was presented at the upper left or upper right location. When the cue was extinguished, subjects saccaded to and then maintained fixation on the lower fixation stimulus. A target requiring a manual detection response appeared with equal probability at one of the four locations. The results indicated that it is the cued environmental location that suffers the repercussions of IOR and not the cued retinal location (see also Posner & Cohen, 1984). Moreover, when an object is presented at a cued environmental location and then moves from that position, IOR follows the object - even if the object rotates in a 180 degree arc around fixation in order to occupy the mirror-symmetric location (Gibson & Egeth, 1994a; Tipper, Driver, & Weaver, 1991; Tipper, Weaver, Jerreat, & Burak, 1994; although, see Muller, & von Muhlenen, 1996, and Weaver, Lupianez, & Watson's, 1997, response). Such environmental coding of locations or objects is antithetic to a retinal basis of IOR.

METHODS FOR ASSESSING RETINAL VERSUS ENVIRONMENTAL CODING OF IOR (MAYLOR, 1985)

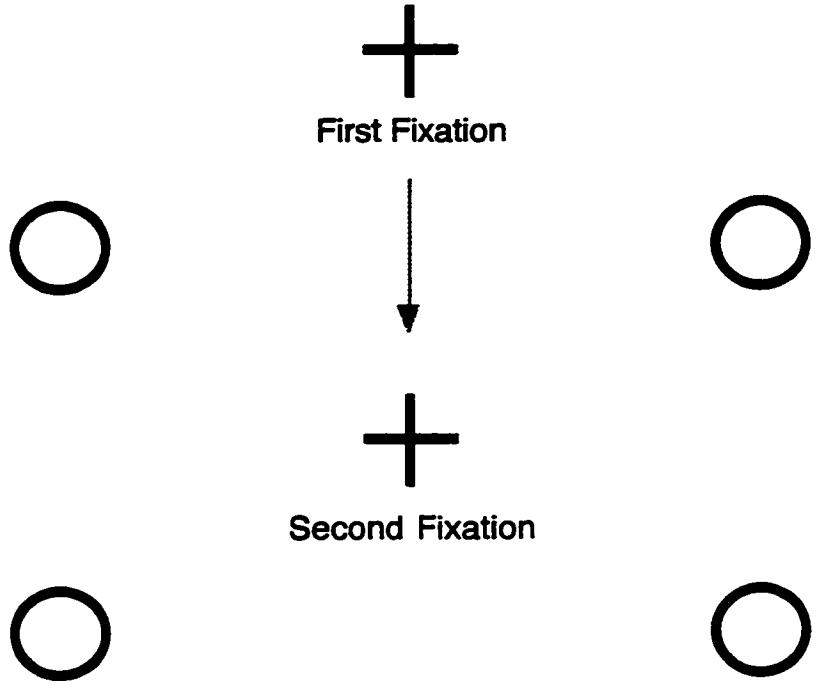

Figure 2. Methods used by Maylor and Hockey (1985) to determine whether IOR is mapped in retinotopic or environmental coordinates. Subjects began by fixating the location marked as the first fixation. One of the two locations represented by the upper circles could be cued with a luminance increment. Following the cue, subjects made a saccade to the location marked as the second fixation. A detection target appeared at any one of the four locations.

If IOR is not inhibition of low-level sensory registration of target information at the cued location, then what is inhibited as a result of prior cue presentation? Two alternatives dominate the literature. The first follows from Posner et al.'s (1985) functional interpretation of IOR and posits that it is covert attention that is inhibited from returning to the previously-cued location. As will be seen, the second follows from repeated demonstrations that IOR is not measured by tasks that are known to be vulnerable to attentional processing. Championed by Klein and Taylor (1994; see also, e.g., Posner & Cohen, 1984; Rafal et al., 1985; Rafal et al., 1989 for arguments supporting an oculomotor basis of the effect), this second view posits that IOR is a motor bias against making responses to a target that has appeared in a previously-cued location.

The view that IOR represents inhibition for returning attention to a previously-cued location implies a reduction in the speed and/or efficiency of perceptual processing at that location. This is because - in signal detection terminology - covert attentional allocation enhances perceptual sensitivity at the cued location (e.g., Downing, 1988) by bolstering the signal strength and/or it increases the speed of visual processing at the cued location (cf., Sternberg & Knoll, 1973). That attention affects the efficiency to process stimuli that occur at or near (cf. Downing & Pinker, 1985; Henderson, 1991; Henderson & Macquistan, 1993; LaBerge & Brown, 1986; Shulman, Sheehy & Wilson, 1986; Shulman, Wilson, & Sheehy, 1985; although see Egly & Homa, 1991 for dependence of gradients on task difficulty) the cued location is well-established (Bushnell, Goldberg, & Robinson, 1981; Corbetta, Miezin, Dobmeyer, Shulman, & Petersen, 1990; Hawkins, Hillyard, Luck, Mouloua, Downing, & Woodward, 1990; Hawkins, Shafto, & Richardson, 1988; Lyon, 1990; Mangun, Hanson & Hillyard, 1986; Mangun & Hillyard, 1991; Muller &

Findlay, 1987; although see Shiu & Pashler, 1994, 1995). To the extent that IOR represents the inhibited return of attention to a previously-cued location, perceptual processing at the cued location would therefore not be expected to show the usual benefits of enhanced perceptual processing and, instead, may be expected to show inhibited perceptual processing. Note that, in this case, perceptual inhibition is distinguished from postulated sensory inhibition on the grounds that it is the speed or accuracy with which the percept is formed from the sensory registry that is affected, not the sensory registration itself. Given that this is the case, if IOR is inhibition for returning attention to a previously-cued location, then perceptually-based dependent measures that impact the magnitude of attentional costs and benefits (and are therefore sensitive to the allocation of covert attention) should likewise reflect the operation of IOR.

Reuter-Lorenz et al. (1996) noted that the magnitude of attentional costs and benefits is: Greater for visual versus auditory targets; greater for low intensity versus high intensity targets; and, equivalent for manual detection and saccadic responses. As such, they argue that to the extent that IOR and attentional orienting share a common mechanism, the magnitude of IOR should be similarly influenced by target modality, target intensity, and response mode. To test this hypothesis, Reuter-Lorenz et al. (1996) presented subjects with a 200 msec peripheral visual cue to the left or right of fixation; this cue was followed at an SOA of 500 msec by a cue at fixation; and, at an SOA of 1000 or 1300 msec relative to the peripheral cue, a visual or auditory target was presented to the left or right. The results were consistent with patterns of attentional orienting effects. Specifically, the magnitude of IOR was: Greater for visual versus auditory targets; greater for low intensity versus high intensity targets; and, equivalent for manual detection versus

saccadic responses. However, this cannot be taken as evidence for an attentional basis of IOR.

Consider that, because attentional effects must be inferred from behaviour and cannot be assessed directly, tests of an attentional basis of IOR necessarily take the form of a modus tollens argument:

If A, then P.
Not P.
Therefore, not A.

More concretely, if A and P refer to an attentional basis of IOR and to perceptual effects, respectively, the argument is that: If IOR is attentional, then there will be perceptually-based effects; there are no perceptually-based effects; therefore, IOR is not attentional. Given this logical structure, the tactic of seeking perceptual effects of IOR in order to support an attentional basis is an illogical application in the form:

If A, then P.
P.
Therefore, A.

More concretely, the illogical structure is: If IOR is attentional, then there will be a perceptual effect; there is a perceptual effect; therefore, IOR is attentional. The conclusion is flawed because it presumes an attentional basis of IOR is the *only* factor that could produce perceptual effects. In other words, the fact that IOR may be affected by some of the same manipulations as affect attentional costs and benefits (Reuter-Lorenz et al., 1996; see also Handy, Jha, & Mangun, 1997), does not impel the conclusion that attention is inhibited by IOR. However, a conclusion against inhibited attention *is* impelled by a failure to demonstrate perceptual effects. This follows by noting that a logical

flaw occurs only in the case where P (a perceptual effect) is used to assert the veracity of A (an attentional basis); in the case of not P (no perceptual effect), the correct form of the modus tollens is adopted. In fact, evidence in the literature weighs heavily in favour of not P (that is, no perceptual effect). Studies that have used temporal order judgements, the frequency of illusory line motion, and non-spatial discrimination tasks - by virtue of failing to demonstrate an effect of IOR on either the speed or quality of perception - converge on the conclusion that IOR is not attentional.

Temporal order judgements are used to assess the locus of covert attention and are predicated on the perception of prior entry (i.e., early perceptual awareness of a stimulus) due to speeded transmission of stimuli at an attended location (cf. Hikosaka, Miyauchi, & Shimojo, 1993a; Stelmach & Herdman, 1991; Sternberg & Knoll, 1973). In a temporal order judgement task, two stimuli are presented, with a variable delay imposed between the onset of one and the onset of the other; both remain visible throughout the trial duration and the subject's task is to indicate which was onset first. The notion of prior entry maintains that if stimuli are presented simultaneously (i.e., with a 0 msec delay) - one at a cued location and one at an uncued location - subjects are likely to report the stimulus at the cued location as preceding that at the uncued location. If, on the other hand, there is a (non-zero) delay between the onset of the two stimuli, then attention serves to: 1) lengthen the perceived delay when the leading stimulus is at the cued location; and, 2) shorten the perceived delay when the leading stimulus is at the unattended location, affecting the judgement of the leading stimulus accordingly.

If IOR is inhibition for returning attention to a previously-cued location, then temporal order judgements should be impacted in the manner

outlined in Figure 3. At long cue-target SOAs, transmission from the cued location should be slowed relative to the uncued location: In the simplest example of two stimuli being onset simultaneously (as depicted in Figure 3), subjects should perceive the stimulus from the uncued location as being onset first; when a delay separates the onset of the two stimuli, there should be longer perceived delays (hence greater accuracy of temporal order judgement) when the stimulus in the uncued location leads and, shorter perceived delays (hence lower accuracy of temporal order judgment) when the stimulus at the cued location leads. This does not occur. At least when accuracy is employed as the dependent measure in a temporal order judgement task, subjects show no evidence of slowed transmission from the cued location (Maylor, 1985; Posner et al., 1985). However, it should be noted at this juncture that there may be some conditions under which temporal order judgements may reflect the operation of IOR if RT, rather than accuracy, is used as the dependent measure (Gibson & Egeth, 1994). This evidence will be considered in the General Discussion.

Like temporal order judgements, the frequency of illusory line motion (cf. Hikosaka, Miyauchi, & Shimojo, 1991, 1993a, 1993b, 1993c; Miyauchi, Hikosaka, & Shimojo, 1992) is predicated on the notion of prior entry (although see Downing & Treisman, 1995, 1997). Unlike a temporal order judgement task, however, which involves an actual temporal delay between the onset of events at disparate locations, the assessment of illusory line motion is made by presenting a line all at once and having subjects judge whether it appears to show motion. To the extent that attention speeds the transmission of neural signals, an illusion of motion away from an attended location would be predicted to result from the fact that the end of the line near the locus of attention is perceived sooner than parts of the line that are

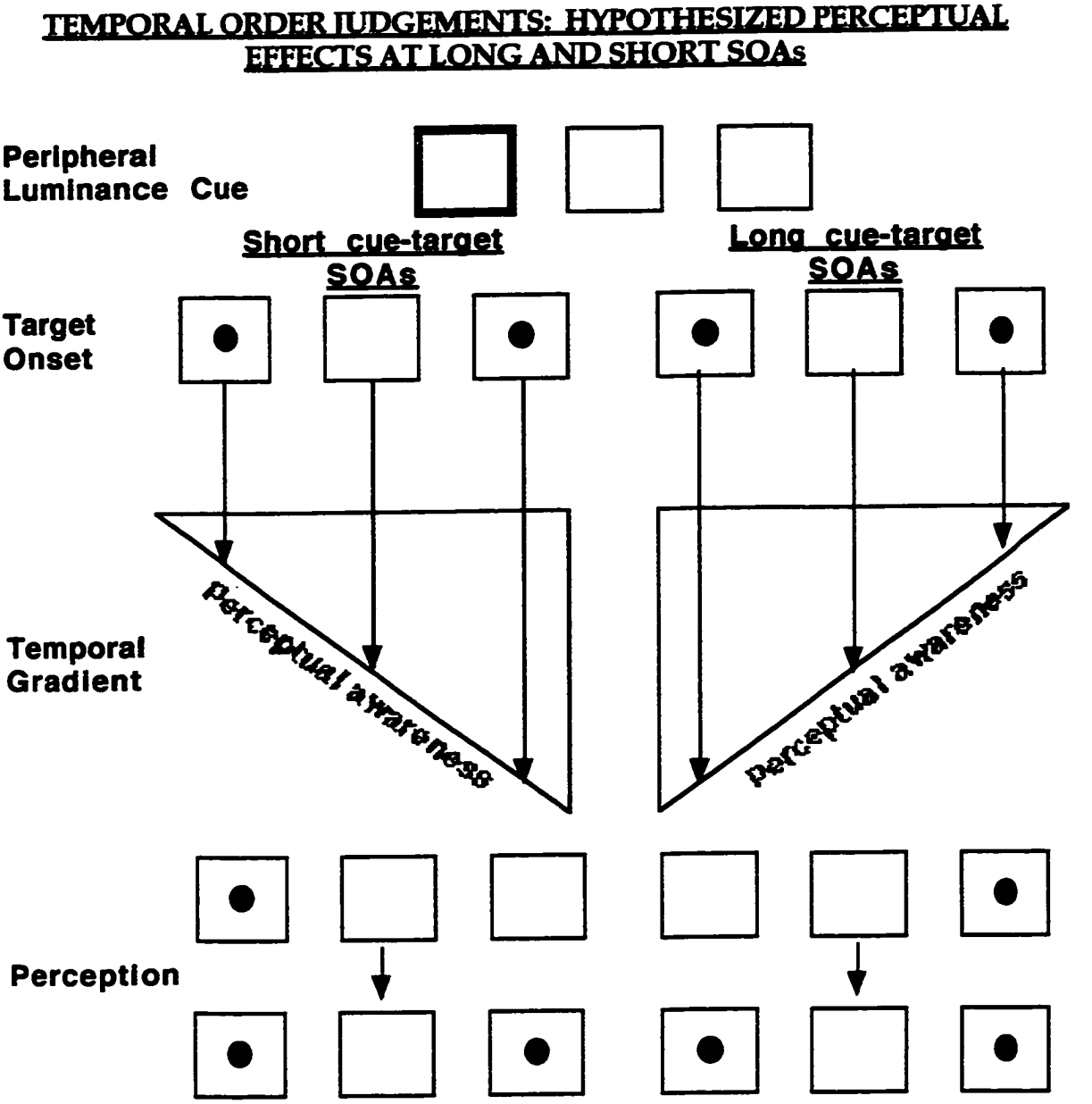

Figure 3. Temporal judgement: Hypothesized perception of simultaneously-onset targets, as a function of short versus long SOAs. At short SOAs, the cue sets up a processing gradient whereby locations near the target are processed to the level of perceptual awareness faster than are remote locations; at long SOAs, the gradient reverses and locations remote from the cued location are processed to perceptual awareness faster than are near locations.

more distant from that locus.

The rationale for determining whether the frequency of illusory line motion supports a perceptual basis of IOR is similar to the rationale underlying the attempt to find IOR with a temporal order judgement. In particular, consider the case of a cue that is presented to the upper left or upper right of fixation and followed at variable SOAs by a line that joins the cued and uncued locations. Using this procedure, Schmidt (1996b) reasoned that if IOR is inhibition of perceptual processing, then at short cue-target SOAs, an illusion of motion away from the cued location should predominate, whereas at long cue-target SOAs, an illusion of motion away from the uncued location should predominate. Despite being able to demonstrate the efficacy of his cueing procedures for inducing IOR in a manual detection task, Schmidt (1996b) found no evidence for inhibited perceptual processing at the cued location at any of the SOAs tested.

Results from the temporal order judgement and frequency of illusory line motion tasks demonstrate that IOR is not (consistently) observed for the for the speed of perceptual processing. Nevertheless, an attentional explanation of IOR can be rescued if it is assumed that the *quality* of a perceptual representation might be inhibited independently of the speed of forming that representation. In other words, even if IOR does not reflect slowed transmission of sensory signals from a previously-cued location, if perceptual awareness of the sensory activation is an insufficient basis upon which to generate the response, then IOR might be observed due to a poor quality percept from that location. Results from non-spatial discrimination tasks, however, demonstrate that this is not always the case. Indeed, although there may be circumstances under which IOR affects non-spatial discriminations (e.g., Danziger, Kingstone, & Snyder, 1997; Handy, Jha, &

Mangun, 1997; Lupianez, Milan, Tornay, & Tudela, 1997; Pratt, Kingstone, & Khoe, 1997), other findings demonstrate that whether subjects make a vernier (Tanaka & Shimojo, 1996), size (Pontefract & Klein, 1988), luminance (Tanaka & Shimojo, 1996), colour (Tanaka & Shimojo, 1996; although see Law, Pratt, & Abrams, 1995), orientation (Tanaka & Shimojo, 1996) or form (Terry, Valdes, & Neill, 1994) discrimination on the basis of a target presented at a previously-cued location, IOR is not observed (see the General Discussion for possible reasons for why this may be the case).

The failure to consistently observe IOR for target discriminations is not due to the choice response. It is well-established that IOR can be obtained for choice responses based on target localization and does not depend on simple manual detection. For instance, IOR is obtained for lateralized saccadic eye movements made to the target location (Abrams & Dobkin, 1994, 1995; Maylor, 1985; Posner & Cohen, 1984; Rafal, Egly, & Rhodes, 1994; Reuter-Lorenz, et al., 1996; Vaughan, 1984), for manual localization responses (i.e., left-right; e.g., Maylor, 1985; Tanaka & Shimojo, 1996), and for non-spatial discrimination tasks that require a localization response (e.g., saccade to a target square and away from a distractor diamond; Pratt, 1995).

This dissociation between non-spatial discrimination tasks and those based on target localization corresponds with the neurophysiological distinction between the ventral ("what") and dorsal ("where") pathways, as noted by Tanaka and Shimojo (1996). At a gross level, the ventral pathway leads to object recognition via the analysis of information through to the inferotemporal cortex; the dorsal pathway leads to object localization via the analysis of information through to the parietal cortex, via SC. The fact that IOR is measured by localization but not by non-spatial discrimination tasks therefore suggests that IOR might be mediated by the dorsal pathway. Because

the analysis of stimulus location guides directed motor responses, inhibition of activity associated with the dorsal representation of a spatial location would predict slower responses to that location.

This view of dorsal pathway involvement offers a possible neural correlate for Klein and Taylor's (1994) view of what is inhibited by IOR. In particular, Klein and Taylor (1994) note the preponderance of evidence showing that IOR occurs only for target localization and, on these grounds, argue that IOR is not inhibition of attention but, rather, is inhibition for making motor responses to a previously-cued location. They posit that IOR is represented in a spatial motor map that directs action: "In essence, there is a criterion shift for responding that something has happened at a particular location...this shift does *not* affect the processing efficiency of information coming from the attended location...the criterion is changed for 'responses to' stimuli from a particular location." (Klein & Taylor, 1994, *p*. 143). This motor biasing account of IOR is able to accommodate the findings of slower reaction times to make manual detection, manual localization, and saccadic responses, as well as for the failure to observe IOR in discrimination tasks that do not involve target localization. Moreover, it is able to predict the additional finding that IOR not only slows saccadic latencies, but also biases saccade *direction.*

Consider that, if IOR is a motor bias against responding to targets arising at the previously-cued location, this criterion change for responding in the cued direction should be reflected in a response measure based on directional preference. Using a temporal order judgement-type task, Posner et al. (1985) presented subjects with a 200 msec luminance increment cue at one of two peripheral locations. This cue was followed after a 500 msec delay by a brightening at fixation. Then, after another 750 or 1250 msec delay, a lead

target dot appeared at one of the two peripheral locations followed 10, 25, 45, or 200 msec later by the onset of a second target dot in the other peripheral location. Subjects were instructed to move their eyes in the 'most comfortable' direction following target onset.

The cue-(lead)target SOAs were within the range for obtaining IOR. As such, the dependent variable of interest was whether subjects would tend to demonstrate a motor bias by moving their eyes away from a first target that appears in the previously-cued location - at least when the interval separating the onset of the two targets was short. Although on the majority of trials, subject tended to move their eyes in the direction of the target that actually led, at the three shortest target-target intervals, this tendency was offset by a bias to move the eyes away from a first target that appeared at the cued peripheral location (i.e., subjects tended to move their eyes away from the cued location even when the lead dot appeared at that location). Notably, the analogous bias did not appear when subjects were, instead, asked to perform a temporal order judgement, thereby replicating Maylor's (1985) failure to find an effect of IOR on the speed of perceptual processing (see above). This finding of a directional bias in the absence of a perceptual processing effect cannot be easily accounted for by other than the motor bias account of IOR.

This ability of the motor bias view to account for a bias in saccade direction, increased saccadic and manual latencies, and an absence of effects due to the speed and/or quality of visual perception makes it the most plausible view of what is inhibited by IOR. In short, on the weight of evidence, IOR does not represent inhibition of low-level sensory processing or inhibition for allocating covert attention to a previously-cued location. Instead, IOR appears to reflect a motor bias against responding to targets that arise at a previously-cued location.

IOR: HOW IS THE INHIBITION GENERATED?

Whereas the question of what is inhibited by IOR focuses on manipulations of the target task, the question of how IOR is generated focuses on manipulations of pre-target events (most notably, cueing procedures). In so doing, manual and saccadic RTs have been used as the dependent variables, under the presumption that these measures are equivalently sensitive to inhibitory operations of the cue. Accepting that manual and saccadic responses are valid measures of a general motor bias against responding to target information arising at the previously-cued location, how is this bias established?

One possibility is that IOR depends on response inhibition (cf. Harvey, 1980). In the standard IOR paradigm, subjects are discouraged or else prohibited from making any overt response to the peripheral onset cue. This suggests that endogenous inhibition of a motor response to the cue may continue to operate at the time of target presentation. This view is consistent with anisotropies in the spatial distribution of the inhibitory effect and, in fact, such anisotropies have been cited as evidence for a motor response view of IOR.

These anisotropies take the form of meridian effects that are defined by a within-hemifield spatial gradient of IOR that is bounded by the major meridia. In other words, the gradient does not cross the vertical meridian when cues and targets are presented along the horizontal; and, does not cross the horizontal meridian when cues and targets are presented along the vertical (Tassinari, et al., 1987). Tassinari et al. (1987) intimated that these meridian effects arise from the vetoing of a motor directional command. In particular, they argue that selective attention to the cue elicits a state of motor readiness that includes "general facilitation of all motor outputs potentially

triggered or guided from the target area" (Tassinari et al., 1987, *p*. 68; for arguments that meridian effects derive from oculomotor, as opposed to general motor, preparation see Hughes & Zimba, 1987; Rizzolatti, Riggio, Dascola, & Umilta, 1987). Because of the requirement to suppress an overt response to the cue, this motor preparation - which includes a representation of cue direction - must be vetoed. This veto continues to be represented in the motor set at the time of target presentation. As a consequence, if the target occurs in the same direction as the cue, the veto and the target direction conflict, resulting in slowed RTs.

While the motor inhibition view of the genesis of IOR is able to account for field anisotropies, it is contradicted by a plethora of data indicating that IOR occurs in response-response as well as in cue-target paradigms. Whereas a cue-target paradigm expressly requires that the subject inhibit a response to a stimulus event (the cue), in a response-response paradigm (also referred to as target-target or continuous-responding paradigms), IOR is measured as slowed responses that are made to the same location as a preceding response. Indicating first and second responses, respectively, IOR has been observed for manual-manual (Maylor & Hockey, 1985, 1987; Posner, Cohen, Choate, Hockey, & Maylor, 1984; Terry et al., 1994), saccadic-manual (Posner et al., 1985; Rafal et al., 1989), and saccadic-saccadic (Vaughan, 1984) combinations. Despite the fact that a manual-saccadic combination has not been examined, these demonstrations force the conclusion that the genesis of IOR is not dependent on the inhibition of a (manual or saccadic) response. The implication regarding field anisotropies is that - contrary to Tassinari et al.'s (1989) view - these do not reflect how IOR is generated, but rather, they reflect what is inhibited: Responses to the location (or toward the direction) of the preceding cue. In this regard, the anisotropies may be a manual

analogue to the directional bias that Posner et al. (1985) observed for saccades.

Even though field anisotropies associated with the spatial distribution of IOR cannot speak to the generation of the inhibitory effect, Tassinari et al.'s (1989) conjecture that covert attention and motor control are intimately related is not without precedence (e.g., Klein, 1980; Posner, 1980; Posner & Cohen, 1980; Rizzolatti et al., 1987). In fact, given that IOR can be obtained in both cue-target and response-response paradigms, the interrelation of covert attention and motor control is a necessary precondition of any viable account of IOR. One account that is able to satisfy this condition - by allowing for an interrelation of covert attention and oculomotor control - is the attentional view that gave rise to the term, "IOR".

Recall that the attentional view maintains that allocation of covert exogenous - but not endogenous - attention to a first signal generates IOR (see page 6). This view makes no claims about responses to the first signal. IOR is seen to be a function of exogenous attention so that whether an exogenous signal is ignored or else serves as the basis of a manual or saccadic response is of no consequence - so long as attention is captured by the peripheral luminance increment, IOR is generated thereby.

The observation that saccades made in the absence of an exogenous signal (i.e., endogenously-generated saccades) are capable of generating IOR (e.g., Posner & Cohen, 1984; Rafal et al., 1989; Rafal et al., 1994) would seem to be at variance with the view that automatic allocation of attention in accordance with the first signal is a necessary condition for generating IOR. However, there is considerable evidence that both exogenously- and endogenously-driven saccades are intimately linked with covert attentional orienting. Even in the absence of incentive to do so (Henderson, 1990; Henderson, Pollatsek, & Rayner, 1991; Hoffman & Subramaniam, 1995;

Inhoff, Pollatsek, Posner, & Rayner, 1989; Kowler, Anderson, Dosher, & Blaser, 1994; Posner, 1980; Rayner, McConkie, & Ehrlich, 1978; Remington, 1980), covert attention is deployed in advance to the location of an executed saccade (e.g., Chelazzi, Biscaldi, Corbetta, Peru, Tassinari, & Berlucchi, 1995; Shepherd, Findlay, & Hockey, 1986).

By thus asserting that IOR is a consequence of the prior allocation of exogenous covert attention, the attentional view presumes an opponent process relation between the facilitatory and inhibitory components of IOR. It follows that the two effects must therefore be consistently correlated. However, this does not appear to be the case. Despite being a proponent of the attentional view, Maylor (1985) reported IOR for saccadic RTs, in the absence of early facilitation; Lambert and Hockey (1991) demonstrated that - particularly with a high-salience cue - facilitation is eliminated by practice in the absence of a concomitant diminution in the magnitude of IOR; and, these dissociations notwithstanding, direct attempts to define a role of covert attention in the genesis of IOR have likewise failed to provide strong supportive evidence (e.g., see the conflict between the double-cueing manipulations of Posner & Cohen, 1984; Maylor, 1985; although see also Tassinari & Berlucchi, 1993).

Whereas the attentional view maintains that saccades are effective in generating IOR because they result in the covert allocation of exogenous attention, in an innovative twist, Rafal et al. (1989) reversed the argument by postulating that exogenous cues are effective in generating IOR because they result in saccadic programming. Consistent with the dissociations observed between IOR and facilitation, this oculomotor view makes functional dependence between covert exogenous attention and saccadic eye movements superfluous with respect to the genesis of IOR. More explicitly, this view

asserts that IOR is not mediated via covert mechanisms but arises via direct activation of the oculomotor system; this activation may be independent of any effects that exogenous signals have on covert orienting.

Compelling evidence in favour of the oculomotor activation hypothesis was provided by Rafal et al. (1989). In a study that explored the conditions necessary to generate IOR, they varied both the nature of the first signal event (exogenous/endogenous) and eye movement instruction (eyes fixed, saccade execution, saccade preparation). Their methods, depicted in Figure 4, presented subjects with three horizontally-arranged stimulus boxes, separated by 10 degrees visual angle. At the beginning of the trial, one of the peripheral stimulus boxes was cued for 300 msec endogenously via the onset of a central arrow at fixation, or exogenously via a luminance increment. On half of the trials, a target followed this first cue at an SOA of 150 or 350 msec. The location of this target was predicted with 80% probability by the preceding cue. On the remaining half of the trials, following a 200 msec delay, the centre fixation box brightened for 300 msec. This centre cue cancelled the predictive validity of the preceding cue: Following a 950 or 1250 msec SOA relative to the first cue, the detection target appeared with equal probability to the left or right.

In the eyes fixed condition, subject maintained fixation throughout the trial duration and attended covertly in accordance with the cue; in the saccade execution condition, subjects saccaded to the first cue, and then back to centre upon onset of the cue at fixation; and, in the saccade preparation condition, subjects were to prepare a saccade in case the target appeared at an early interval (in which case, a saccadic response was required) but were to withhold execution of the saccade if a cue was presented at fixation. In the eyes fixed and saccade preparation conditions, subjects made a manual

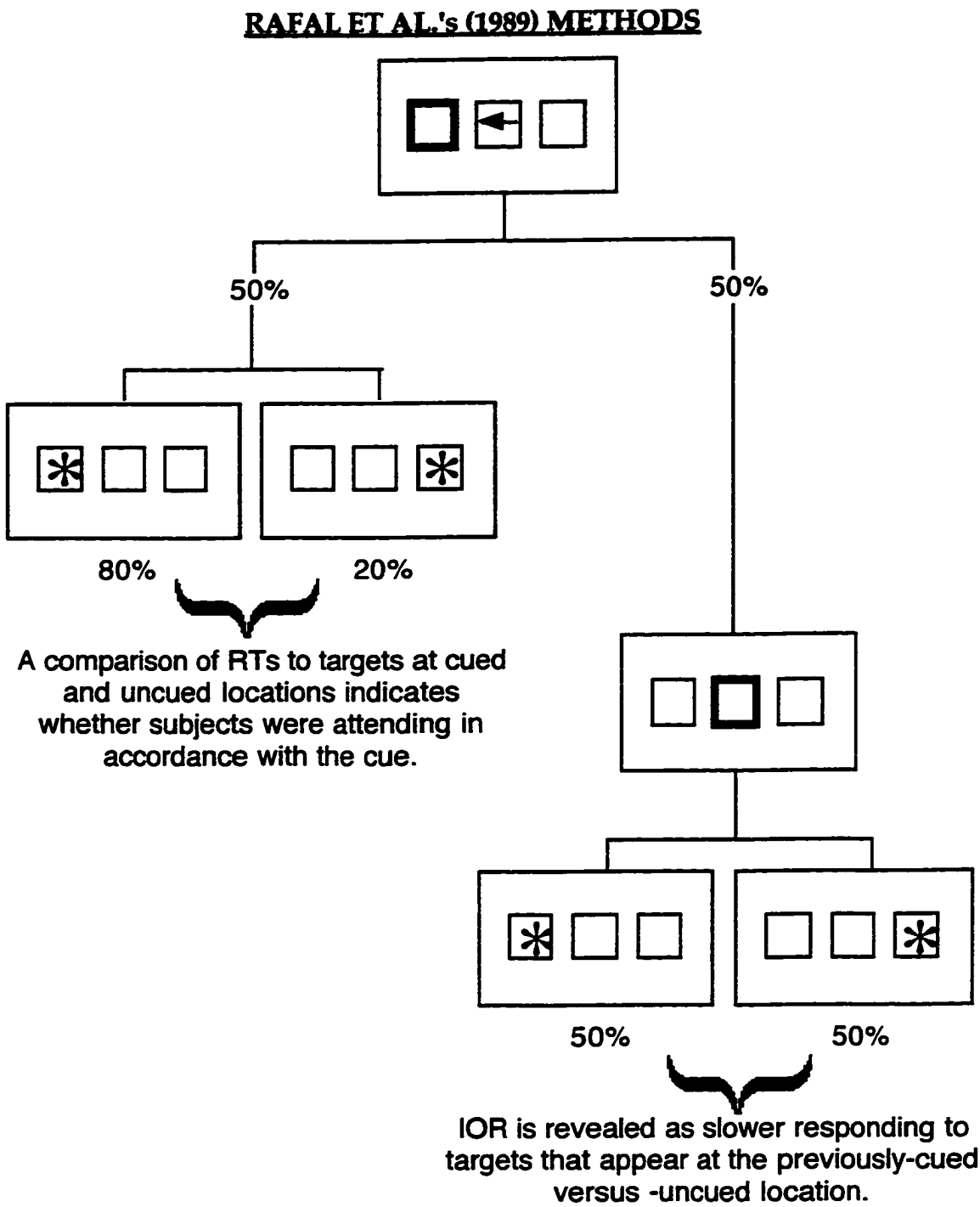

Figure 4. Example of the methods used by Rafal et al. (1989). A peripheral or else a central arrow cue (both are shown above) was presented on a trial-by-trial basis. On 50% of the trials, the first cue was followed by a target whose location was predicted with 80% accuracy by the cue. On the remaining 50% of the trials, the first cue was followed by a second cue at fixation, after which the two peripheral locations were equally likely to contain the target.

detection response to the onset of a target that occurred after the first cue. In all conditions, subjects made a manual detection response to the onset of a target that occurred after a second cue. The cue conditions and response requirements are summarized in Table 1 (A).

As seen in Table 1 (A), on the critical double-cue trials, the eyes fixed/endogenous cue and saccade preparation/endogenous cue conditions were identical, except that in the latter a saccade was prepared and then cancelled. In spite of the similarity of the subjects' behaviour in these two conditions, the saccade preparation/endogenous cue condition produced IOR whereas the eyes fixed/endogenous cue condition did not. Exogenous cues activate the oculomotor system even in the absence of an explicit requirement to prepare/execute a response; endogenous cues do not. Given this, the results shown in Table 1 (B) can be best summarized as follows: Any condition which activates an oculomotor program - whether via automatic activation of the oculomotor system by the cue (as in the eyes fixed/exogenous cue condition), via explicit instructions to prepare a saccade to an exogenous or endogenous cue, or via the execution of a saccade to an exogenous or endogenous cue - generates IOR. This finding that saccadic programming is the critical basis of IOR is consistent with three converging lines of evidence that places the inhibition at the level of the superior colliculus (SC).

IOR AND THE SC

The SC and the frontal eye fields (FEF) comprise two parallel pathways responsible for producing saccadic eye movements (Albano, Mishkin, Westbrook, & Wurtz, 1982; Albano & Wurtz, 1982; Schiller, 1977; Schiller, True, & Conway, 1980). Part of the phylogenetically-older retinotectal

RAFAL ET AL.'s (1989) RESPONSE REQUIREMENTS AND RESULTS

A) Response requirements as a function of cue and eye movement conditions (Rafal et al., 1989). Note that the first cue could be exogenous or endogenous.

Condition	Single Cue (50%		Double Cue (50%)	
	Cue	Target	Cue	Target
Eyes Fixed	Maintain Fixation	Manual Response	Maintain Fixation	Manual Response
Saccade Preparation	Prepare Saccade to Cue	Saccade	Cancel Saccade	Manual Response
Saccade Execution	Saccade to Cue	Manual Response	Saccade to Centre	Manual Response

B) IOR as a function of response and cue condition (Rafal et al., 1989): Black circles indicate significant IOR; the white circle indicates the absence of IOR.

	Exogenous First Cue	Endogenous First Cue
Eyes Fixed	●	○
Saccade Preparation	●	●
Saccade Execution	●	●

Table 1. Summary of Rafal et al.'s (1989) response conditions (A) and results (B).

pathway, the SC is responsible for reflexive orienting to visual targets (e.g., Mohler & Wurtz, 1977; Sparks, 1978). In contrast, the FEF is responsible primarily for goal-directed saccades (e.g., Schiller, Sandell, & Maunsell 1987) and exerts higher-level control over the reflexive machinery of the SC (e.g., Guitton, Buchtel, & Douglas, 1985) through both direct connections to the SC (e.g., Braun, Weber, Mergner, & Schulte-Monting, 1992; Sparks & Porter, 1983) and indirect connections via the caudate nucleus and substantia nigra pars reticulata (Hikosaka & Wurtz, 1983a, 1983b, 1983c, 1985a, 1985b).

Each SC receives predominantly monocular input from the contralateral visual field, with greater representation from the temporal hemifield (nasal hemiretina) than from the nasal hemifield (temporal hemiretina) of the open eye. As such, under monocular viewing conditions, to the extent that IOR is collicular, it should be greater for targets that appear in the temporal relative to the nasal hemifield. Rafal et al. (1989) tested this prediction under the exogenous, eyes fixed conditions depicted in Figure 4 and described previously.

At the two earliest cue-target intervals, RTs were faster at the cued relative to the uncued location; at the two longest cue-target intervals, there was IOR. The magnitude of the facilitatory effect did not differ by field (see also Shulman, 1984). Consistent with known connectivity of the retinotectal pathway, however, IOR - although significant in both fields - was greater for targets that appeared in the temporal relative to the nasal hemifield. Interestingly, the finding that initial orienting of attention to the cued location did not show a field bias whereas IOR did show such a bias supports the dissociability of the facilitatory and inhibitory consequences of the first cue. Moreover, given that a temporal field bias is suggestive of retinotectal involvement, the finding that initial orienting to a predictive cue does not

show a field bias argues against a common pathway with SC-generated saccadic eye movements. This is the case even though the first cue contained both an endogenous and an exogenous component (by virtue of being a predictive luminance increment).

The second line of evidence that converges with SC involvement in the genesis of IOR comes from neuropsychological studies of individuals afflicted with Progressive Supranuclear Palsy (PSP). Like Parkinson's disease, PSP is associated with degeneration of the basal ganglia and consequent motor impairment. However, ophthalmoparesis due to degeneration of the SC and peritectal region is pathognomic to PSP. These individuals demonstrate a marked impairment in making voluntary saccades, particularly in the vertical plane. Posner et al. (1985) argued that to the extent IOR is associated with the oculomotor system: 1) PSP patients should reveal deficits in IOR that are greater in the vertical than in the horizontal plane; 2) such deficits should be tied to collicular damage and should therefore not be a consequence of motor impairment associated with basal ganglia degeneration in Parkinson's disease or related to lesions of frontal or parietal cortex.

Posner et al., (1985) compared the performance of PSP patients, Parkinsonian, frontal, parietal, and normal controls in a study that presented a 150 msec brightening at one of two peripheral locations. Following 50 or 150 msec, early detection targets appeared with 80% probability at the location of the first cue. In the absence of an early target, the centre location brightened and a late target appeared with equal probability at the two peripheral locations, either 600 or 1000 msec following the first cue. Consistent with their predictions, the authors reported IOR for all groups in the horizontal plane; no difference in IOR for the vertical versus horizontal plane in Parkinson patients; and, no IOR in the vertical plane in PSP patients.

A collicular basis of IOR is likewise supported by a third line of evidence that results from Abrams and Dobkin's (1994) application of additive factors logic (cf. Sternberg, 1969) to the study of IOR and the gap effect. The gap paradigm presents subjects with a central fixation stimulus that can be offset at various intervals relative to the onset of a peripheral saccade target. When the fixation stimulus is offset prior to, or else coincident with, the onset of the saccade target, RTs are reduced relative to when the fixation stimulus remains visible (e.g., Fischer & Ramsperger, 1984; Kingstone & Klein, 1993; Reulen, 1984; Reuter-Lorenz, Hughes, & Fendrich, 1991; Saslow, 1967). Several lines of evidence converge on SC mechanisms as the basis for the facilitation of saccadic RT that occurs upon the offset of a fixated stimulus (i.e., the gap effect): 1) The threshold current required to elicit a saccade via the SC is increased during active fixation (Sparks & Mays, 1983); 2) fixation cells in the intermediate rostral SC inhibit the remaining intermediate SC during active fixation (Munoz & Wurtz, 1992, 1993a, 1993b); and, 3) activity in these fixation cells is correlated negatively with the latency to make saccades in the gap paradigm, such that the maximum decrease in discharge rates corresponds with the fastest saccades (Dorris & Munoz, in press). According to additive factors logic, to the extent that IOR and the gap effect depend on common neural processes/mechanisms, they should interact.

Abrams and Dobkin (1994) presented subjects with a fixation dot in the centre of an otherwise blank field. A 300 msec peripheral onset cue was presented 7 degrees to the left or right of fixation. Following a 200 msec delay, the fixation stimulus brightened for 300 msec. On overlap trials, the fixation cue was followed by a 360 msec delay during which the fixation stimulus remained visible; on 0 msec gap trials, the fixation cue was followed by a 360 msec presentation of the fixation stimulus at the end of which the fixation

point was extinguished; on 200 msec gap trials, the fixation cue was followed by a 160 msec presentation of the fixation stimulus, and then by a 200 msec blank screen. A saccade target then appeared with equal probability at one of the two peripheral locations. Consistent with a common, collicular, mechanism, the authors report a reliable IOR effect, a reliable gap effect, and a significant interaction of IOR with gap condition.

Taken together, the weight of evidence falls in favour of the oculomotor view of the genesis of IOR: Whether an oculomotor program is elicited automatically via the onset of an exogenous signal or else endogenously via the preparation to make a saccade, inhibition is established for subsequent saccadic or manual (cf. Rafal et al., 1989) responses. On the basis of converging evidence of a temporal field bias for IOR, deficient IOR along the vertical meridian for PSP patients, and the interaction of IOR with the gap effect, it appears that the oculomotor machinery of the SC plays a key role.

Is an SC-based oculomotor basis of IOR easily reconciled with the suggestion that IOR may be for motor and visuo-motor biases? Tanaka and Shimojo (1996) indicated that dependence of IOR on target localization is consistent with a role of the dorsal pathway in the measurement of IOR. On the output end, the SC is part of this pathway. As such, the SC presents a possible neural basis for linking the methods by which IOR is generated to the means by which it can be measured. However, a coherent picture of the inhibitory effect must account for the ability of IOR to be generated by endogenously- as well as exogenously-activated oculomotor programs and for the ability of this activation to produce an inhibitory effect that is measured as a bias against responding to targets that appear at the cued location.

SC MAPPING OF IOR?: ACTIVATION AND ACCESS

Neurophysiological data (see Mohler & Wurtz, 1976) indicate that visual response output from the retinotopically-organized (Colby, 1991) superficial layers of the SC converges with two-dimensional motor output from the deep layers (Hepp, VanOpstal, Straumann, Hess, & Henn, 1993) to create an intermediate layer mapping of motor error (Segraves & Park, 1993; Sparks & Porter, 1983; see also Krommenhoek, VanOpstal, Gielen, & VanGisbergen, 1993, Massone, 1994 for neural network models) that drives brainstem oculomotor centres[5]. Vector coding of saccade metrics in the intermediate layer of the SC can be influenced by topographic connectivity that exists between FEF and SC. For example (see Schall, in press, for an overview), the lowest thresholds for antidromic stimulation of FEF sites occur at SC sites that code the same vector; simultaneous stimulation of FEF and SC sites results in an elicited saccade that is the sum of the vectors from each stimulation site weighted by the applied current; and, in the absence of competing activation, electrically-evoked saccades from the FEF excite SC saccade cells of the intermediate layers that code the same vector (Schlag-Rey, Schlag, & Dassonville, 1992). Given this connectivity, a role of the SC in the genesis of IOR is not at variance with the ability of endogenously-generated saccades to either generate or measure inhibition.

In particular, because IOR is an environmentally-coded effect that influences motor responses based on target localization, to the extent that it may be SC-based, the representation of the inhibition would be expected at the level of the intermediate layer. If this reasoning is correct, then any response that determines activation within this map should be capable of producing IOR; any response that depends on the level of activation within that map should be capable of measuring IOR. Topographical connectivity of the FEF

and SC indicates that both endogenous and exogenous saccadic programming involves access to this intermediate layer SC map so both should be able to generate (i.e., lay down a neural representation of) and measure (i.e., be sensitive to the effects of) the inhibition.

This speculation regarding an SC basis of IOR is seductive because of its internal consistency: IOR appears to be generated by the activation of a saccadic vector in the intermediate layers of the colliculus and it is in precisely such a visuo-motor map that a neural representation of inhibition (e.g., decreased neural activity/responsiveness) could interfere with directed responses to a previously-stimulated location. However, there are holes in the data set from which this speculation derives. As will be seen, it is not clear whether the bias against responding to a previously-cued location is a motor and/or a visuo-motor effect; whether IOR is generated by activation of an oculomotor program or due to activation of any ocular or non-ocular directed response; and, perhaps most crucially, whether what is inhibited might depend critically on how IOR is generated.

GENERATING AND MEASURING IOR: TOWARD A COMPLETE ASSESSMENT

As indicated previously, the motor bias account of IOR derives considerable support from the body of literature manipulating the manner by which IOR is measured. However, all of the studies reviewed above demonstrated IOR using measures of RT or directional preference in response to *peripheral* onset targets. Even accepting the likely supposition that IOR is a bias for responding to stimuli from a previously-cued location, it is unclear whether this bias is strictly motor or whether it stems from a visuo-motor interaction that is supported by the presentation of a peripheral target.

Particularly if a role of the intermediate SC is assumed, the representation of inhibition in an integrated visuo-motor map could rest on inhibitory input from the motor layers themselves and/or from the integration of signals deriving from both the motor and visual layers. Given that a peripheral target represents visual stimulation at the location to which a response must be directed, the use of such targets confounds motor and visuo-motor processes.

By examining saccadic latencies in response to exogenous versus endogenous motor command signals, Abrams and Dobkin (1995) were able to distinguish visuo-motor and motor bases of IOR[6]. Abrams and Dobkin (1995) presented subjects with a 300 msec onset cue 7 degrees to the left or right of fixation; no response was required. After a 200 msec delay, a cue was onset at fixation for 300 msec. After a 160 msec delay, a saccade signal was presented, indicating left and right locations with equal probability. In separate blocks, this saccade signal was exogenous (the onset of a peripheral luminance target) or endogenous (a directional arrow presented at fixation). In the case of an exogenous command, the directed motor response was contingent on processing of the exogenous visual signal from a potentially-inhibited location (hence was visuo-motor); in the case of the endogenous command, the directed motor response was identical but occurred without requisite visual processing from a potentially-inhibited location (hence, was strictly motor with respect to the peripheral location).

Abrams and Dobkin (1995) found a significant 24 msec IOR effect in the exogenous command condition and a significant 9 msec IOR effect in the endogenous command condition. The fact of significant IOR for both command signals argues that there was an overall motor bias against responding to the cued location, irrespective of whether visual information

was presented at that location. However, the 15 msec difference in the magnitude of IOR in the two signal conditions indicated that the inhibitory effect observed with peripheral onset targets was not accounted for entirely by motor inhibition: There was an additional visual inhibitory influence associated with peripheral visual processing and/or integration of visual information with the motor response to the cued location[7].

Apart from qualifying the motor bias view of IOR, Abrams and Dobkin's (1995) study introduces the notion that what is inhibited by IOR may depend not only on how IOR is measured (i.e., perceptual, discrimination, localization tasks) but also on how the measured response is elicited (i.e., exogenously or endogenously). Their results indicate that - at least for saccades - IOR for making a speeded response toward a peripheral location represents inhibition for making a motor response to *either* an exogenous or an endogenous signal and that, in the case of the former, there is an additional inhibition associated with having to make the motor response on the basis of peripherally-presented information.

What is not clear on the basis of Abrams and Dobkin's (1995) results is whether IOR for a speeded manual response might also demonstrate a visuo-motor and/or motor basis, depending on whether the response is commanded exogenously or endogenously. If the same pattern did not emerge for manual responses as was observed for saccadic responses, this would challenge the assumption that manual and saccadic RTs are different rulers for measuring the same inhibitory effects. One goal of the current research is to test this assumption by measuring possible IOR effects under exogenous and endogenous command, using both manual and saccadic RT as dependent measures. Given that what is inhibited in saccadic RT depends on *how* the response is elicited, it seems a reasonable conjecture that what is

inhibited might also depend on *what* response is elicited. Are visuo-motor and motor inhibition represented by IOR for manual responses elicited under exogenous and endogenous control, respectively? Or might the differences between exogenous and endogenous control be specific to oculomotor responses (perhaps due to the accuracy of a saccadic response depending on the adequacy of - and not just on the fact of - target localization)?

Questions regarding the role that directed motor responses may play in the genesis of IOR can also be asked. Consider that, in a cue-target paradigm, comparison of covert orienting in response to exogenous and endogenous cues reveals that IOR does not occur following the latter. This is a defining feature of the inhibitory effect. Recall that, in establishing the importance of saccadic programming for the generation of IOR, Rafal et al. (1989) relied on the critical finding that IOR occurred following the cancellation of a prepared saccade to an endogenous cue but not following covert orienting to an endogenous cue (refer back to Table 1(B)). Because the only difference between these two conditions (i.e., eyes fixed/endogenous cue versus saccade preparation/endogenous cue) was whether subjects explicitly prepared an eye movement, Rafal et al. (1989) argued that it was saccadic preparation per se that generated IOR. This view meshed nicely with the additional observations that: 1) Exogenous cues produced IOR whether or not subjects explicitly generated a saccadic program (i.e., eyes fixed/exogenous cue versus saccade preparation/exogenous cue); and, 2) both exogenous and endogenous cues generated inhibition when they signalled the execution of an overt movement of the eyes.

Rafal et al.'s (1989) findings would suggest that the difference in the capacity for exogenous and endogenous signals to generate IOR rests with their links to the oculomotor system. In particular, it is tempting to conclude

that exogenous cues enjoy a privileged status in their ability to activate an oculomotor program and that endogenous cues can do so only under the demands of an instructional set (i.e., when subjects are explicitly required to use the cues to program and/or execute a saccadic response). However, given that endogenous and exogenous cues differ not only in the nature of the signal itself but are also associated with different forms of covert attentional orienting, it is possible that endogenous covert orienting interferes with the ability to observe IOR to endogenous signals.

Indeed, this potential problem is exacerbated in other studies (e.g., Posner & Cohen, 1984) for which the comparison of exogenous and endogenous cueing has been confounded by the use of probability manipulations in relation to the latter but not the former. While this is a motivated feature of any attempt to encourage subjects to attend in response to the endogenous cues, the probability manipulation clouds the comparison of exogenous/endogenous commands and makes the a priori assumption that covert attentional orienting is critical to the comparison.

Consider the possibility that an ignored uninformative exogenous cue may generate IOR for responses made to a peripheral onset target because both the cue and target stimuli are defined by a luminance increment (cf. Yantis & Jones, 1991) and not because the cue is peripheral stimulation per se. In other words, the knowledge that a response is required to a luminance onset target might establish a priority tag wherein the generation of a motor response occurs to *any* onset event. When stimulated by the cue, such motor activation could, in turn, lead to IOR. Accepting this as plausible, then in a cue-target paradigm that requires subjects to respond to endogenous signals as the target response might also set up an equivalent priority tag for endogenous signals that are used as the cue. To determine whether this

might be the case, the current study employs unpredictive exogenous and endogenous commands as first and second signals. Is it possible that withholding a response to an endogenous signal that is associated with a saccadic or manual motor command (as opposed to an endogenous covert orienting command) might be capable of generating IOR - perhaps via automatic motor activation?

With respect to this issue of activation of the motor response system, Rafal et al.'s (1989) study suggested that it is *oculomotor* rather than general motor response activation that generates IOR. In a control study that resembled the endogenous cue/saccade preparation condition (outlined in Table 1 and tested using the methods shown in Figure 4), Rafal et al. (1989) had subjects prepare, and sometimes cancel, a directed manual response with the left hand. On 50% of trials, the arrow cue was followed by an onset target that appeared with 80% probability at the cued location and subjects were required to make a speeded localization response with the left hand. On the remaining 50% of trials, the centre box brightened, indicating that subjects were to cancel the prepared left-hand response and that a target demanding a detection response with the right hand was equally likely at the two peripheral target locations. IOR was assessed following the cancellation of the prepared response.

Unlike when a prepared saccadic response was cancelled, the cancellation of a manual response did not produce IOR. While this is strong evidence that oculomotor activation is necessary for the generation of IOR and that manual response preparation is not sufficient, it does not allow for the possibility that manual response *execution* is sufficient to generate IOR. As such, the interest in the ability of exogenous and endogenous command signals to generate IOR will be extended to include conditions in which

subjects not only withhold a response to the first signal, but also conditions in which they execute a manual response. A condition will also be included wherein subjects execute a saccadic response to exogenous or endogenous first signals; this will allow examination of the effects of specifically oculomotor response execution.

Beyond questions asked of the particular means by which IOR is measured and the methods by which it is generated, there is a final critical question as to whether these interact. In other words, does the manner by which IOR is generated determine what is inhibited? The possibility that such interactions may be fundamental to a characterization of IOR is suggested by a study performed by Rafal et al. (1994) in which they examined latencies of pro- and anti-saccades under conditions in which IOR was generated by: 1) An ignored exogenous cue; 2) a saccade to an exogenous cue; 3) a saccade to an endogenous cue. Where a pro-saccade is an eye movement to the location of an onset target and an anti-saccade is an eye movement of equal amplitude but *opposite* direction as an onset target, the rationale for comparing the two is that both require the detection of a peripheral stimulus but the motoric response to the peripheral stimulus is opposite in the two conditions.

When subjects withheld a response to an exogenous cue, saccadic latencies were longer for targets arising at the cued location for both pro- and anti-saccades. This suggests that the inhibition is for making a response on the basis of target information presented at the cued location: RTs were slowed whether the response was in the direction of the target (pro-saccade) or in the opposite direction of the target (anti-saccade). The same was true when subjects made a saccade to the exogenous signal and then returned fixation to centre before target onset. On the other hand, when subjects

saccaded to an endogenous signal, returned gaze to fixation, and then made a pro- or anti-saccade in response to an onset target, saccadic latencies were longer for targets arising at the cued location but only for pro-saccades.

Why did saccades to an endogenous first signal fail to produce IOR for anti-saccades? Rafal et al. (1994) suggested that the endogenous saccade to the first signal may produce: 1) An inhibition associated with processing a peripheral visual target (i.e., this could represent visuo-motor inhibition wherein subjects are slow to make a response *based* on target information presented at the cued location, not necessarily slow to direct a response *to* that location) which would affect both pro- and anti-saccades; and, 2) an inhibition associated with a motor bias favouring responses away from the cued location/towards the uncued location, which would counter the visual/visuo-motor processing inhibition in the anti-saccade but not in the pro-saccade condition. When peripheral visual processing was circumvented by having subjects saccade to an endogenous signal that was followed by another endogenous saccade signal (i.e., as opposed to an onset signal that directed an anti-saccade to the mirror-symmetric location), IOR was observed. This corroborates the view that endogenous saccades may generate inhibition based on guiding a response via visual stimulation at the cued location *and* inhibition for actually responding to the cued location. In summary, generation of IOR by an exogenous signal produces inhibition associated with making responses based on peripheral visual information presented at a previously-cued location (i.e., visual/visuo-motor inhibition); generation of IOR by an endogenous saccade produces inhibition associated with both making responses based on visual information presented at a previously-cued location (i.e., visual/visuo-motor inhibition) and making responses to (i.e., motor inhibition) a previously-cued location.

The exact nature of Rafal et al.'s (1994) results notwithstanding, the fact that what is inhibited may be determined not only by the response that establishes IOR but also by the signal that commands the response (exogenous, endogenous) suggests a rich tapestry of possible interactions. Although critical research questions have been raised in the preceding discussion, to help visualize questions that remain regarding the generation, measurement, and possible interaction of these factors under conditions of exogenous and endogenous control, cells in which IOR has been obtained in the literature are indicated in Table 2. The design of the current study is intended to fill in all the cells of this matrix in the hope that the results will corroborate, clarify, and/or qualify existing views on the generation and measurement of IOR. Note that the studies contributing to Table 2 have been discussed or alluded to in the preceding review; nevertheless, key studies/findings will be (re-)examined when the relevant cells are introduced for each of the six experiments that comprise the present study.

GENERAL EXPERIMENTAL APPROACH

The six experiments that comprise the current study represent the factorial combination of the response used to generate IOR (no response, manual, saccadic) and the response used to measure RT inhibition (manual, saccadic). Each trial will have two signals (S1 and S2); the responses required to each of these will be constant within, but will vary across, experiment. Where the first response refers to S1 and the second to S2, the combinations for Experiments 1-6, respectively, are: No Response-Manual, Manual-Manual, Saccadic-Manual, No Response-Saccadic, Manual-Saccadic, Saccadic-Saccadic.

Whether to S1 or S2, manual responses require lateralized bimanual

SUMMARY OF THE CELLS THAT HAVE BEEN TESTED FOR IOR

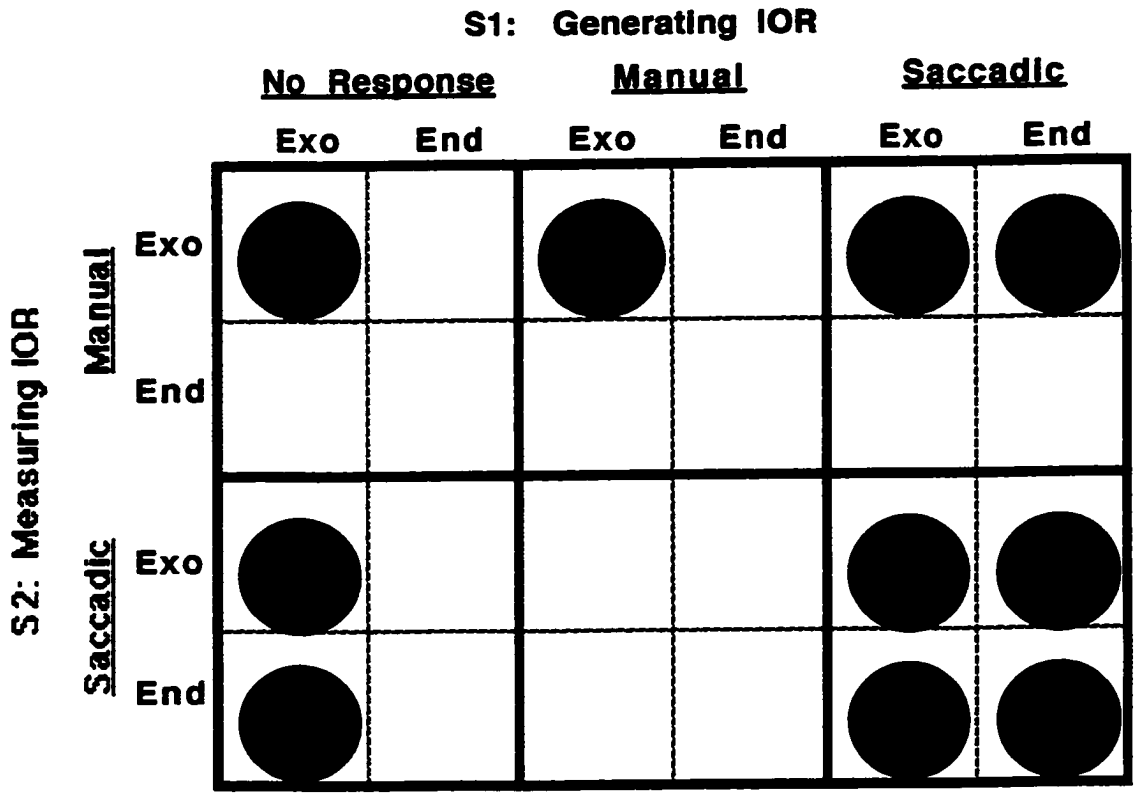

Table 2. Circles appear in all the cells that have been tested in the literature for IOR. Black colouring indicates that IOR was observed.

localization. The decision to use a manual localization rather than a manual detection response is based on the desire to equate - as far as possible - the response characteristics of manual and saccadic responses and the structure of the trial blocks (i.e., the use of detection responses requires variable target onset and/or catch trials). Of course, to be most similar to a saccadic response, a non-self-centring joystick might be most efficacious; however, a manual localization response is more appropriate given the desire to contact existing literature. The combination of responses used to generate and measure IOR for each of the six experiments is represented in Table 3.

Also represented in Table 3 is the factorial combination of command (exogenous, endogenous) and signal (S1, S2) that will be examined for each response combination. The use of exogenous and endogenous signals for the generation and measurement of IOR results in four combinations of S1-S2 commands: Exogenous-exogenous, exogenous-endogenous, endogenous-exogenous, endogenous-endogenous. These four combinations will be mixed within blocks.

To maintain consistency with the literature, the exogenous signal will be a brightening of a peripheral box if it is S1 and the onset of a peripheral target if it is S2. The endogenous signal will be the appearance of a directional arrow at fixation, whether it is S1 or S2. In terms of the response requirements, the only difference between the exogenous and endogenous commands is that the former requires the detection of a peripheral stimulus in order to make a directed response whereas the latter does not. In no case will S1 be predictive of the upcoming location of S2.

The use of peripheral onsets as the exogenous commands and central arrows as endogenous commands - particularly with respect to S2 - is motivated in large measure by the desire to parcel out possible motor and

OVERVIEW OF THE CURRENT EXPERIMENTS

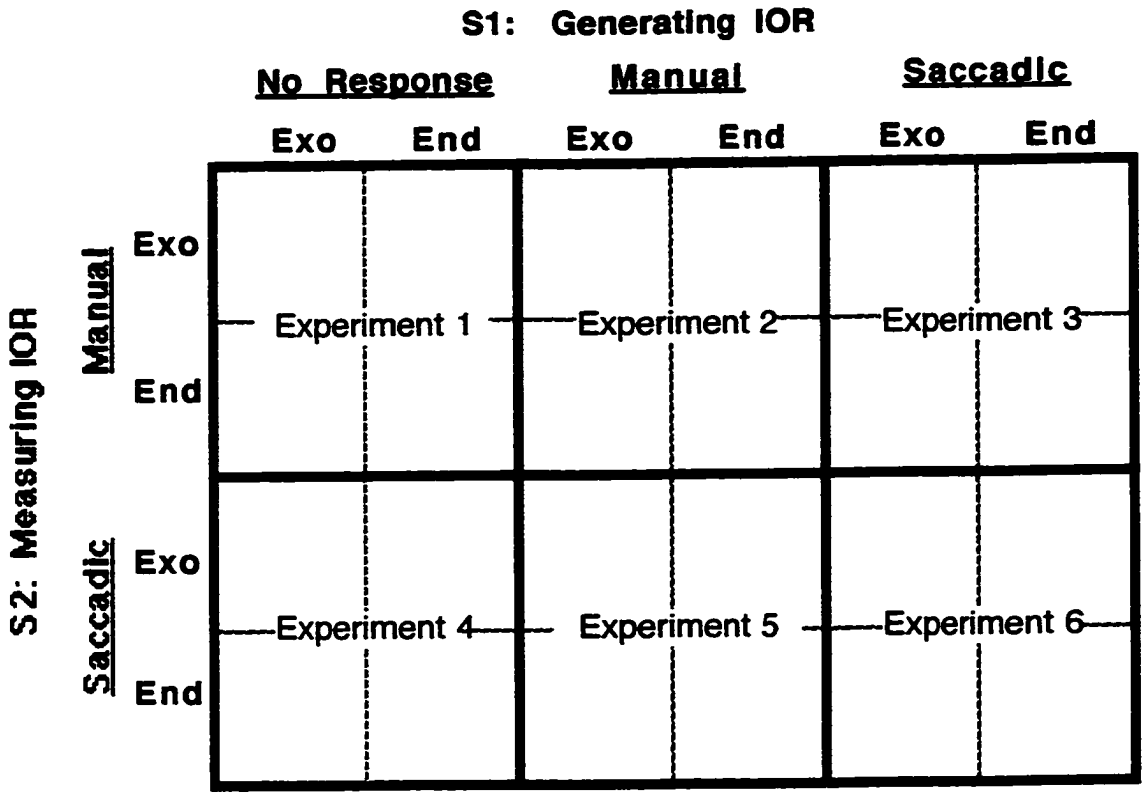

Table 3. Overview of the response combinations and signals (exogenous, endogenous S1 and S2) comprising Experiments 1-6.

visuo-motor response biases. The literature converges on the notion that IOR represents some form of a motor response bias. To the extent that this evaluation is correct, exogenous S2s are capable of measuring potential inhibition of visuo-motor integration and motor responding whereas endogenous S2s are capable of measuring only potential inhibition of motor responding (cf. Abrams & Dobkin, 1995).

Why is this the case? First, exogenous and endogenous S2s signal identical motor responses and are therefore both capable of measuring any inhibition that may occur for the motor response itself (i.e., a motor response bias). Second, exogenous S2s require that a motor response be directed by visual information appearing at a peripheral - and therefore potentially inhibited - location, whereas endogenous S2s direct a motor response based on a symbolic visual signal presented at a central location (which is neutral with respect to the locus of potential location-based inhibitory effects). Given that the comparison used for assessing IOR is the speed to respond to a previously-signalled versus -unsignalled location, if IOR reflects location-based inhibition for visuo-motor processing/integration, then only exogenous signals should be sensitive to the operation of those effects at a peripheral location. In contrast, because endogenous signals are presented at fixation, any location-based inhibition associated with visuo-motor processing/integration would not be attached differentially to arrows that signal the same versus different location relative to S1. In other words, even if there were inhibition for visuo-motor processing/integration based on a central visual signal, this effect would not be reflected in the difference score (i.e., RT when S1 and S2 signal same location versus different location).

It should be emphasized at this point that the distinction between visuo-motor versus motor inhibition offers a short-hand for summarizing

the conditions under which IOR may be observed: IOR may be reflected in slowed motor responses to peripheral visual signals and/or slowed motor responses to any signal regardless of whether it is peripheral or central. The distinction between visuo-motor and motor inhibition does not presume to characterize the underlying mechanisms of any observed effects; the terminology is that used by Abrams and Dobkin (1995) and intended as a heuristic for summarizing the *stimulus* conditions under which IOR is observed for a particular response measure. With this caution in mind, a comparison of IOR indexed by endogenous versus exogenous S2s provides a convenient means for summarizing the stimulus conditions that are sensitive to the inhibitory effects of IOR. More specifically, if equivalent IOR effects appear in both measures, the effects will be classified as reflecting a general motor inhibition; if IOR occurs for exogenous but not for endogenous S2s, the effects will be classified as visuo-motor; and, if IOR is of greater magnitude for exogenous than for endogenous S2s, then the effects will be classified as being both visuo-motor and motor inhibition.

With respect to the specific methods to be used in the current study, on each trial, subjects will be presented with three horizontally-aligned outline boxes. Five hundred msec following trial initiation, S1 will be presented. This will either be a 300 msec brightening of one of the peripheral boxes or else a 300 msec presentation of a directional arrow at fixation. On dual response trials, subjects will make a speeded manual or saccadic response to S1, as appropriate. After a 200 msec delay relative to the offset of S1, the centre box will brighten for 300 msec. Although an explicit cue to centre is not required in order to obtain IOR (cf. Possamai, 1986), the majority of studies in the literature have used a central brightening. Moreover, a central brightening is a convenient signal for conditions in which subjects are

required to make a saccade to S1, as the centre brightening will summon the return fixation to the original point of fixation (cf. Rafal et al., 1989). Following another 200 msec delay, S2 will appear. This will either be a target dot in one of the peripheral locations or else a directional arrow presented at centre.

The key to filling out the design depicted in Table 2 is examining the difference in RT when S1 and S2 signal the same versus different locations (i.e., a measure of IOR) under each combination of S1 (exogenous, endogenous) and S2 (exogenous, endogenous) command. Under what conditions is IOR established? Under what conditions is it measured? How do these interact?

PREDICTIONS STEMMING FROM CURRENT VIEWS OF IOR

On the strength of preceding arguments regarding what is inhibited by IOR and how IOR is generated, it is possible to outline predictions in the context of the current experimental design. First, consider the issue of what is inhibited by IOR. The weight of evidence in the literature suggests that IOR represents a motor inhibition for responding to a previously-cued location. As already noted with respect to manual RTs, only peripheral visual targets have been employed. As such, the literature predicts that a motor response bias will be reflected in the speed to make manual responses to exogenous signals; however, there are no grounds for predicting the same effect for manual responses made to endogenous signals. In contrast, on the weight of evidence that IOR measured by saccadic RTs does not require that the saccade be made to peripheral visual information (cf. Abrams & Dobkin, 1995; Rafal et al., 1994), IOR is expected to occur for saccades made to both exogenous and endogenous commands. These predictions regarding the measurement of

IOR are depicted in Table 4.

Consider now the conditions under which IOR is expected to be generated. The bulk of evidence favours the view that IOR is generated by activation of the oculomotor system. More particularly, IOR is generated by signals that elicit an oculomotor program. Given that exogenous signals appear to be closely linked with saccadic programming (e.g., Posner, 1980; Rafal et al., 1989; Remington, 1980) the occurrence of an exogenous signal should therefore be capable of generating IOR, irrespective of the response requirements associated with that signal. However, there is no clear evidence upon which to ground predictions for endogenous signals. Certainly, when an endogenous signal is used to command a saccadic response, IOR should be generated (i.e., because the execution of the endogenously-commanded saccade requires that there be a saccadic program). In the absence of any evidence that endogenous cues can establish priority tags or otherwise activate saccadic programming, there is no reason to assume that endogenous signals which require no response or else that require a manual response should likewise be capable of generating IOR. The predictions regarding those conditions believed to be capable of generating IOR are shown in Table 5.

The combined predictions for the generation and measurement of IOR are presented in Table 6. Superimposed on this table is a reiteration of those cells for which IOR has been observed in the literature. Not surprisingly, the data upon which views of the generation and measurement of IOR have been based are consistent with a conjunctive association: IOR has been observed in cells for which S1 stimulus/response conditions are capable of generating a saccadic program *and* S2 stimulus/response conditions are capable of measuring the presumed visuo-motor and/or motor bias against responding to the cued location. With respect to the current study, a conjunctive view

MEASURING IOR: PREDICTIONS FROM THE LITERATURE

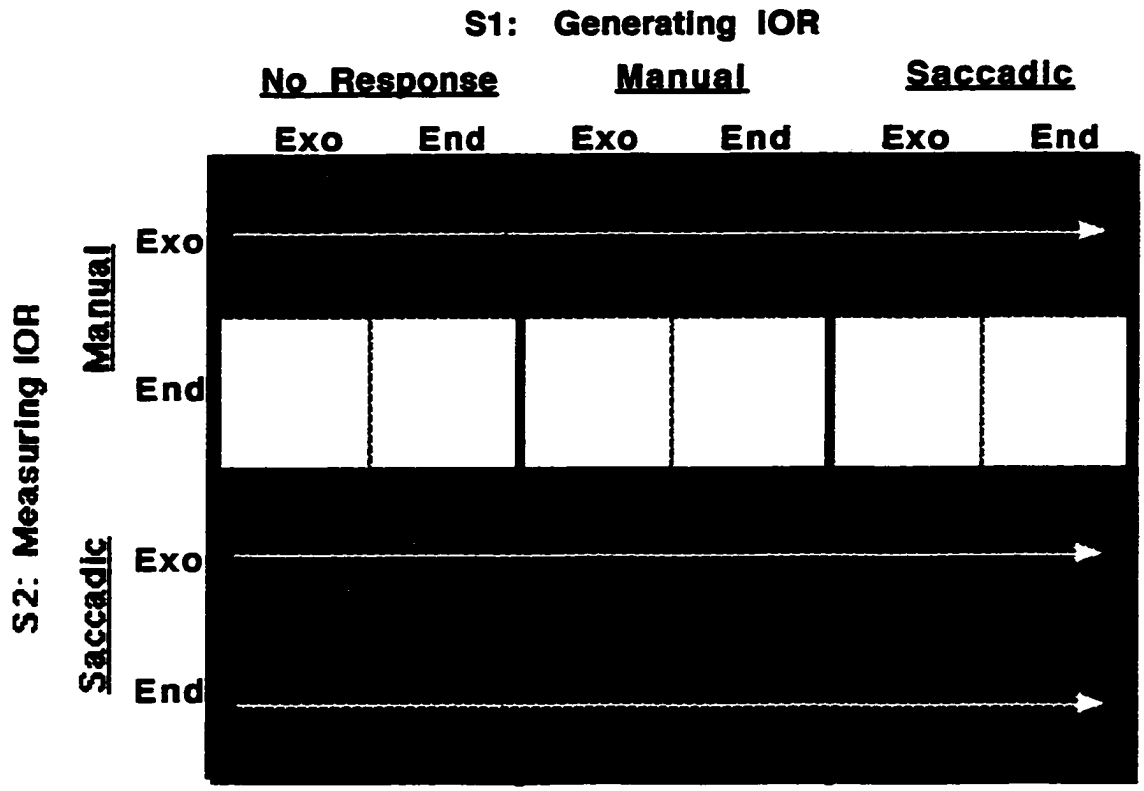

Table 4. S2 conditions predicted to measure IOR (shown with hatching and horizontal arrows). See text for details.

GENERATING IOR: PREDICTIONS FROM THE LITERATURE

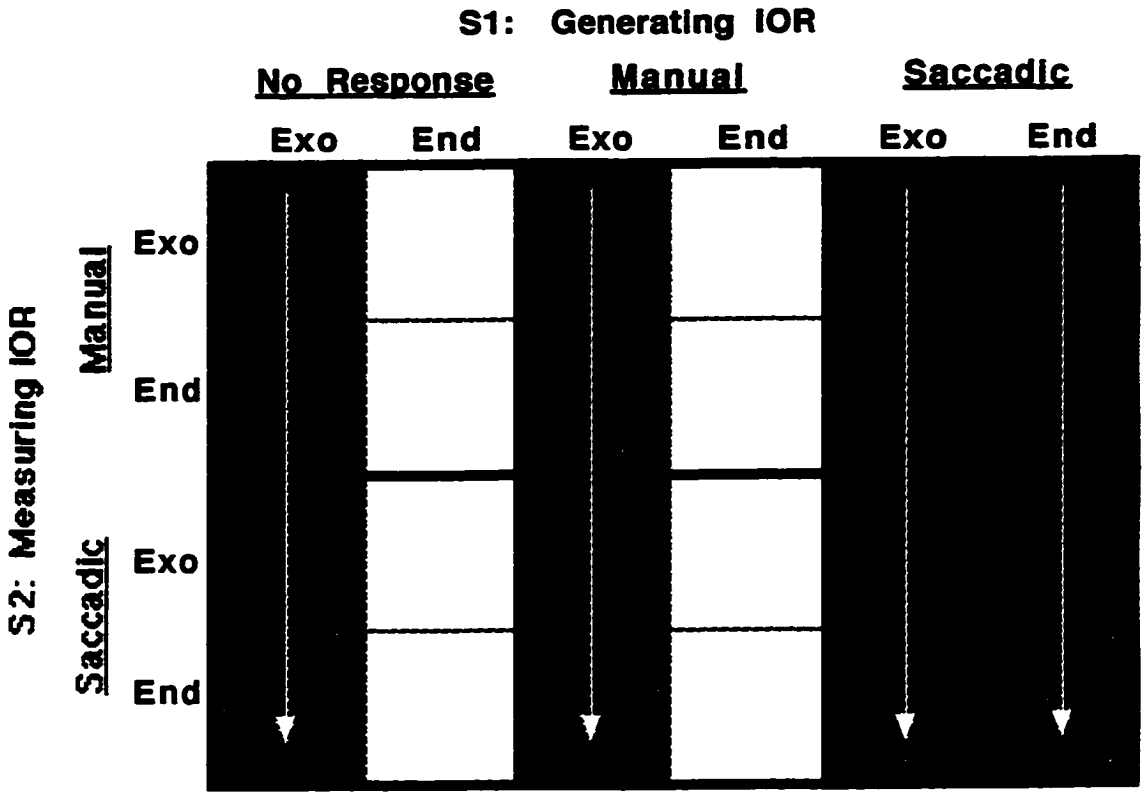

Table 5. S1 conditions predicted to generate IOR (shown with hatching and vertical arrows). See text for details.

GENERATING AND MEASURING IOR: COMBINED PREDICTIONS FROM THE LITERATURE

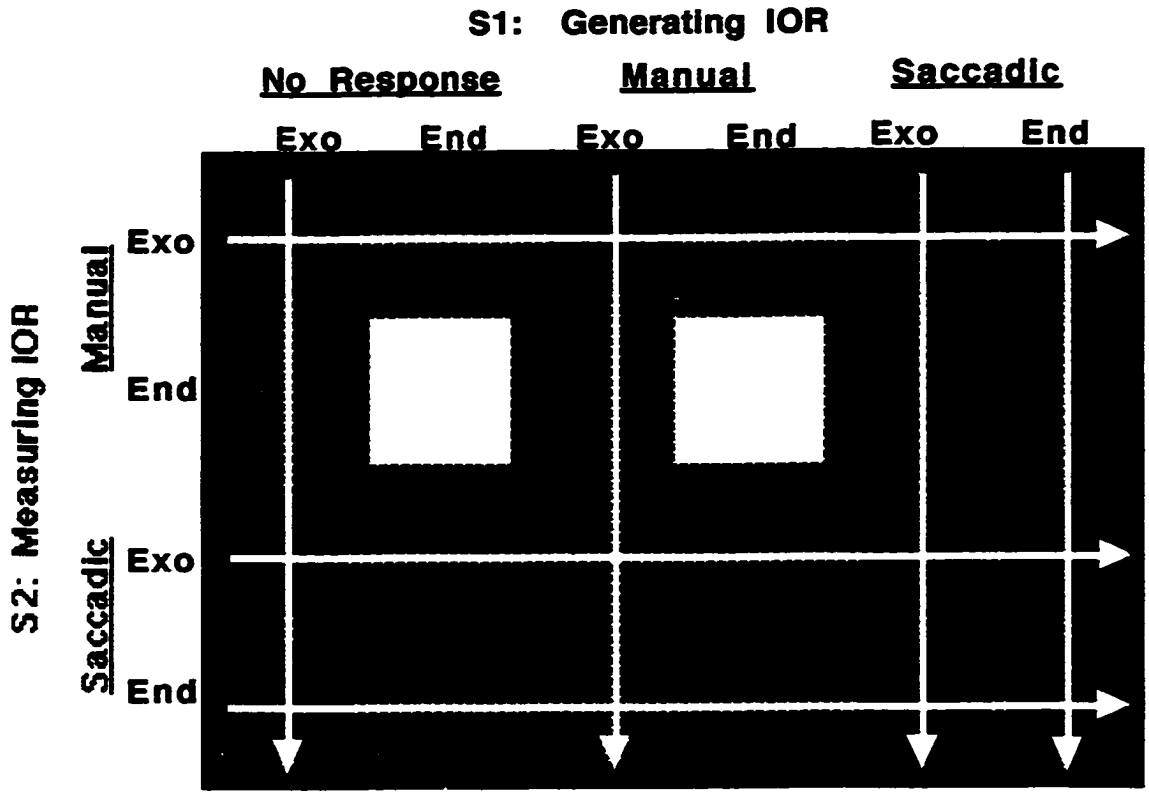

Table 6. S1 and S2 conditions predicted to measure and generate IOR (shown with hatching, horizontal, and vertical arrows). A conjunctive view predicts IOR at the junction of vertical and horizontal arrows; a disjunctive view predicts IOR wherever there is hatching (i.e., along either horizontal or vertical arrows). For comparative purposes, those cells which have been tested for IOR in the literature are indicated with circles; the black colouring indicates that significant IOR was found.

would therefore posit that IOR will occur only in those cells for which predictions for the generation of IOR converge with predictions for the measurement of IOR (i.e., in the cells of Table 6 for which the arrows intersect).

METHODS

SUBJECTS

Eighteen Dalhousie University undergraduate, graduate, and professional students (3 males, 15 females, mean age 24 years) participated in six experimental sessions, each of 45-60 min duration. Subjects were paid $6 per session, with an additional $9 bonus paid upon completion of all six. Subjects taking Introductory Psychology were allowed the option of earning one credit point per session towards their final class grade in lieu of the $6 remuneration. The maximum number of credit points earned by a single subject was 3 points; remaining sessions were paid at the $6 per session rate. By self-report, all subjects had normal or corrected-to-normal vision. All except 2 subjects were right-handed.

STIMULI AND APPARATUS

Stimulus presentation and data collection were controlled by a Macintosh LC630 computer running custom software. Subjects were tested individually in a darkened, sound attenuated room. Each subject was seated 46 cm from the computer screen, with this distance maintained by a head-stabilizing chin and forehead rest. Horizontal movement of the left eye was sampled every 2 msec by an Eyetrac (model 210) infrared reflection device that was mounted on eyeglass frames and supported by head straps. Subjects performed eye monitor calibration and initiated each trial by depressing the

mouse button. When required to make manual localization responses, subjects did so by depressing the left and right arrow keys on an Apple Extended Keyboard II.

Stimuli were presented on an Apple Color Plus 14-inch Display monitor, using an animated 256-colour palette. The initial stimulus display consisted of three, horizontally-arranged outline boxes, each subtending 1.6 degrees visual angle on a side and drawn with lines of 1-point thickness. These boxes were separated by 7.9 degrees visual angle, with the middle box centred on the computer screen. These boxes were light grey (hue: 11548; saturation: 1465; brightness: 45311; red: 45253; green: 45311; blue: 44298) presented on a uniform black background (hue, saturation, brightness, red, green, blue: 0). Imperative stimuli and the centre cue were presented in white (hue, saturation: 0; brightness, red, green, blue: 65535).

Stimuli that appeared subjectively as the brightening of a stimulus box (i.e., the exogenous S1 signals and cue to centre) were accomplished by plotting a white, 2-point outline box whose outside perimeter corresponded with, and was occluded by, the 1-point perimeter of the grey stimulus box. The net result was a 1-point white box plotted within the borders of the grey stimulus box. The subjective impression was that the stimulus box "flashed" or brightened. Arrow signals were drawn with 1-point standard Macintosh tools. They subtended 1.2 degrees visual angle horizontally, 0.4 degrees visual angle vertically at the widest point of the arrow, and were centred horizontally and vertically within the middle stimulus box. Peripheral dots were white filled circles with a diameter of 0.3 degrees visual angle, centred in one of the peripheral stimulus boxes.

DESIGN AND PROCEDURE

Where the first word designates the required response to S1 and the second word designates the required response to S2, the trial blocks which comprised the 6 experimental sessions were: No Response-Manual, Manual-Manual, Saccadic-Manual, No Response-Saccadic, Manual-Manual, Saccadic-Saccadic. With 4 exceptions that resulted from schedule conflicts, subjects ran different blocks on different days; in the case of the 4 exceptions, subjects ran two blocks on the same day, with at least 1 hour separation between blocks. The order of blocks was counterbalanced across subjects, according to a digram-balanced Latin square design (Wagenaar, 1969) that ensured no block immediately followed another in sequence more than once. This design had 6 block sequences (for blocks numbered arbitrarily 1-6, the orders were 123456, 241635, 536142, 315264, 462513, 654321); each sequence was used for three of the 18 subjects.

Each block consisted of a random presentation of 192 experimental trials that resulted from 24 repetitions of the 2x2x2 within-subjects factorial combination of S1 (exogenous, endogenous), S2 (exogenous, endogenous), and location signalled by S2 relative to S1 (different, same). Note that the location signalled by S1 was varied independently of that signalled by S1, with equal numbers of signals directing left and right. In other words, there were an equal number of trials with left-right, left-left, right-left, and right-right combinations of S1 and S2, respectively.

Prior to the experimental trials, 32 trials were drawn randomly from the block and presented as practice trials to the subjects. These were returned to the trial pool and the order of stimulus presentation was newly randomized before beginning the experimental block proper. Subjects were told that there were practice trials at the beginning of the block, but these trials

were not flagged for the subject or demarcated from the remainder of the trial block. During these initial trials, the experimenter stayed in the room with the subject to ensure that the subject could master the response demands and to answer any questions. Practice data was neither saved nor analyzed.

Each block of trials was preceded by eye monitor calibration. This required subjects to fixate each of three points that appeared singly (after a press of the mouse button) at the coordinates that corresponded to the centre of each stimulus box. Once a satisfactory calibration was obtained, subjects self-initiated a trial by depressing the mouse button when they were fixated in the middle of the centre stimulus box. For blocks requiring a manual response for either S1 or S2, subjects positioned their hands with the left arrow key under the index finger of the left hand, the right arrow key under the index finger of the right hand, and the mouse button under the thumb of the right hand; for blocks requiring only saccades, subjects positioned the right hand over the mouse and depressed the button with the fingertips.

The sequence of trial events is shown in Figure 5. The procedure associated with each step will be considered in turn.

Fixation Interval

Following trial initiation, the stimulus display remained unchanged for a fixation duration of 500 msec. Eye movement instability greater than 2 degrees visual angle during this interval aborted the trial and subjects were presented with an error message printed in yellow at the top left-hand corner of the screen; the content of the error message was: "You have moved your eyes from centre. Recycling Trial. Hit mouse to continue."

First Stimulus (S1)

In the absence of fixation instability during the fixation interval, S1 was presented for 300 msec. On half the trials, this was a brightening (with equal

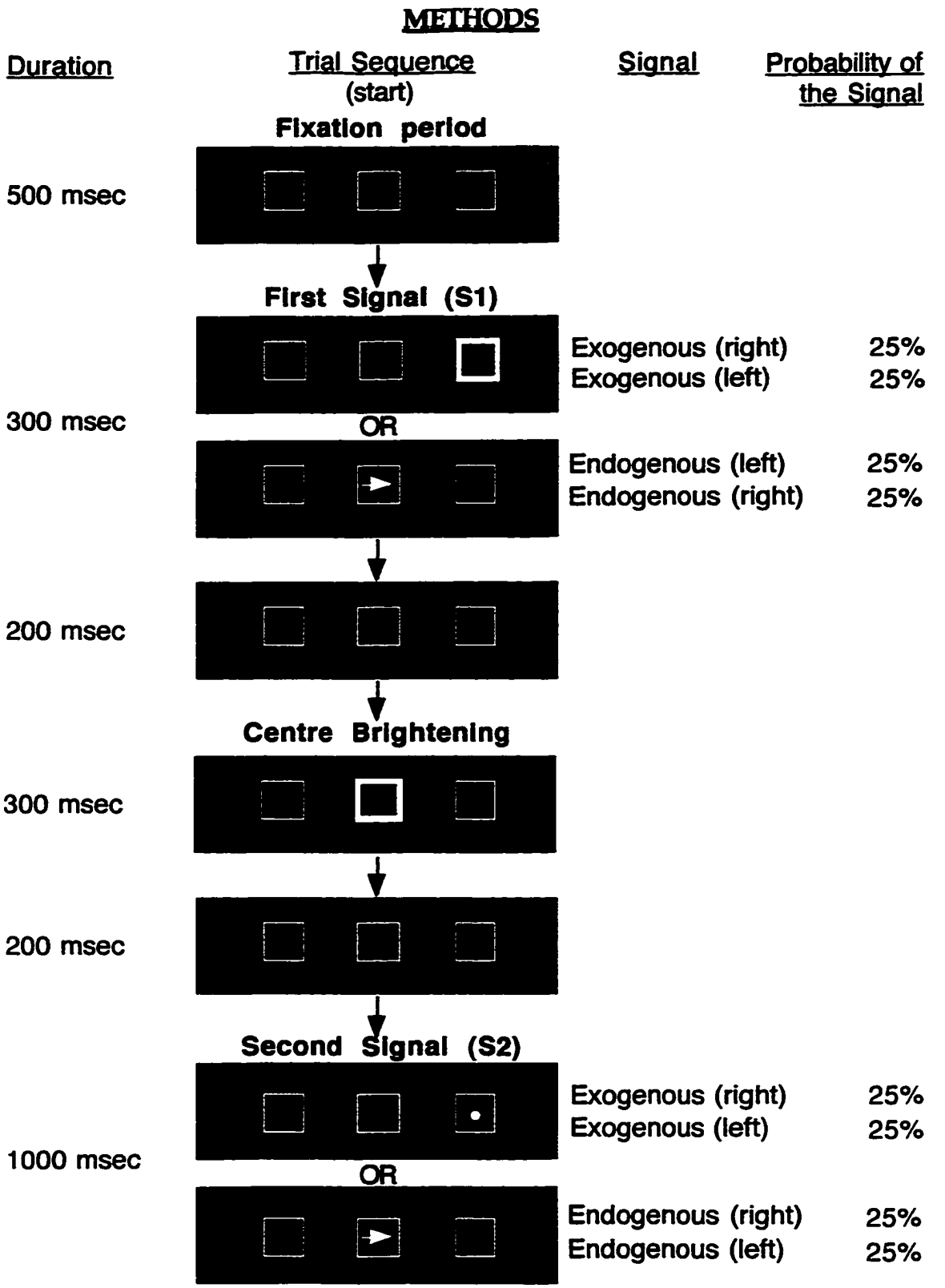

Figure 5. Methods used in the current study. Only rightward signals are shown. See text for details.

probability) of the left or right stimulus box; on the other half of the trials, this was the presentation in the centre box (with equal probability) of a left- or rightward pointing arrow.

For those conditions demanding a manual response to S1, subjects were required to make a speeded localization button-press response, in the absence of an eye movement. If the subject failed to respond within 500 msec, the message "You have taken too long to complete a response. Recycling the trial. Hit mouse to continue" was displayed; if the subject responded in less than 100 msec, the message "You have pressed one of the buttons too early. Recycling the trial. Hit mouse to continue" was displayed; and, if the subject responded with the wrong direction, the message "You have responded in the wrong direction. Recycling the trial. Hit mouse to continue" was displayed. For manual response conditions and also those that required no response to S1, if an eye movement greater than 2 degrees visual angle was detected at any time during S1 presentation or else in the 200 msec that followed, the subject received an error message. This message was the same as described above for eye instability during the fixation period.

For those conditions demanding a saccade, saccade onset was determined as the point at which a velocity criterion of approximately 30 degrees/sec was surpassed; once initiated, the saccade was presumed to terminate when the eye movement velocity fell below threshold for 8 consecutive samples. As with manual responses, subjects were alerted if they failed to respond within 500 msec, responded in under 100 msec, or responded in the wrong direction. Additionally, if subjects made a response on the keyboard, they were given the message "You have pressed one of the buttons too early. Recycling the trial. Hit mouse to continue."

The RT of correct responses made to S1 (i.e., those that were in the

correct direction and that occurred with latencies between 100 and 500 msec) were recorded; the number, latency, and types of errors were not.

Centre Brightening

Five-hundred msec following S1 onset, the centre stimulus box was brightened for 300 msec. For those conditions that required a manual or else no response to S1, fixation instability during the brightening of the centre box or else during the subsequent 200 msec was noted (as described above). For those conditions that required a saccade, the centre brightening was the signal to return the eyes to the centre of the display. The same velocity criterion as described above was used to determine the initiation and termination of the saccade; saccadic latency was calculated but not recorded. If subjects were required to make a saccade back to centre but failed to do so in 500 msec or else initiated the saccade in under 100 msec, they received the appropriate error message, as described above.

Trials on which errors occurred between trial initiation and the onset of S2 were aborted and recycled among those remaining in the block. Following an error, a press of the mouse button cleared the screen and resulted in the presentation of a new trial.

Second Stimulus (S2)

Responses to S2 were vulnerable to the same errors as described for S1. As well, saccadic latencies were determined in the same manner as described above. The only differences between S1 and S2 were: 1) the time limit required to make a response to S2 was increased to 1000 msec (rather than 500 msec); and, 2) errors in responding to S2 were recorded but the offending trials were neither flagged for the subject nor recycled.

Intertrial Interval

There was an imposed intertrial interval of 3000 msec, during which

the screen was a uniform black (i.e., no visible stimuli were displayed). At the end of this interval, the initial 3-box stimulus display reappeared and subjects were free to initiate the next trial at their discretion.

GENERAL STRATEGY FOR ANALYZING AND REPORTING DATA

The error and RT data for all six combinations of response to S1 (no response, manual, saccadic) and to S2 (manual, saccadic) are shown in Table 7. As is immediately clear upon inspection of this table, considerably more errors were observed when the response to S2 was saccadic versus manual. Moreover, there were differences in overall occurrence of each type of error. Separation of the data according to type of error, however, proves to offer little by way of theoretical understanding of the present results; instead, the errors reflect - in some measure - the limitations of data acquisition. In particular, the preponderance of misses when the response to S2 was saccadic reflect few instances of subjects' failure to respond to the signal. Rather, these errors reflect a failure to detect an executed saccade. In most cases, this was due to postural changes or slippage of the eye movement monitors from the time of calibration. The same difficulties in saccade detection arose when S1 responses were saccadic; however, in this case, subjects were alerted to their errors so that adjustments could be made to the equipment. In the absence of error feedback following S2, these changes from calibration were not detected as readily when the second, rather than the first, response was saccadic.

Similarly, the percentage of directional errors observed when S2 required a saccadic versus a manual response also does not reflect adequately the nature of the error. Whereas a directional error for a manual response reflected an explicitly-executed response, in the case of saccadic responses, directional errors could occur due to an intentional movement of the eyes to

EXPERIMENTS 1-6: SUMMARY

S1	Exogenous				Endogenous			
S2	Exogenous		Endogenous		Exogenous		Endogenous	
Location	D	S	D	S	D	S	D	S
Experiment 1: No response-Manual								
RT to IS2	255	276	308	310	256	272	304	304
RT to IS1	0	0	0	0	0	0	0	0
%Dir. Err.	0.00	0.00	2.78	3.70	0.23	0.23	3.47	4.86
%Misses	0.46	0.69	1.39	1.39	1.16	0.23	0.69	1.16
%Ant.	0.46	0.00	0.00	0.00	0.23	0.23	0.23	0.69
Experiment 2: Manual-Manual								
RT to S2	224	247	282	288	232	265	288	270
RT to S1	275	273	272	276	283	285	287	283
%Dir. Err.	0.46	0.00	3.70	5.09	0.23	1.85	4.86	3.24
%Misses	0.93	1.85	2.78	2.55	0.93	1.39	2.55	3.47
%Ant.	0.93	0.69	1.62	0.00	0.46	0.23	0.23	0.23
Experiment 3: Saccade-Manual								
RT to S2	277	291	296	326	278	297	313	337
RT to S1	215	214	213	216	220	216	220	220
%Dir. Err.	0.23	0.23	1.85	5.56	0.23	0.23	1.16	2.78
%Misses	0.23	0.00	0.69	0.46	0.00	0.23	0.69	0.00
%Ant.	0.69	0.00	0.00	0.00	0.23	0.69	0.00	1.16
Experiment 4: No response-Saccadic								
RT to IS2	177	189	229	240	179	186	229	225
RT to IS1	0	0	0	0	0	0	0	0
%Dir. Err.	1.85	1.16	2.31	3.94	1.85	1.85	3.47	3.24
%Misses	3.01	5.56	5.79	6.71	4.17	3.01	4.17	5.79
%Ant.	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.23
Experiment 5: Manual-Saccadic								
RT to S2	174	187	206	234	174	191	218	240
RT to S1	268	266	266	265	283	288	292	285
%Dir. Err.	4.40	2.78	3.47	5.32	3.47	2.08	2.78	3.70
%Misses	5.32	4.86	7.87	9.72	5.79	6.48	9.49	11.34
%Ant.	0.69	0.00	0.00	0.00	0.00	0.23	0.00	1.16
Experiment 6: Saccadic-Saccadic								
RT to S2	162	183	216	235	172	196	222	243
RT to S1	209	206	205	202	210	215	216	218
%Dir. Err.	2.31	1.85	3.70	4.40	2.31	1.39	3.24	3.94
%Misses	3.24	0.69	5.79	1.85	1.62	0.93	3.47	1.39
%Ant.	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

Table 7. Mean RTs to S1 and S2, and percent directional error (Dir. Err.), percent misses, percent anticipations (Ant.) made to S2, as a function of condition (exo: exogenous, end: endogenous), and relative locations (same, different) of S1 and S2.

the wrong stimulus location, an anticipatory saccade in the wrong direction, or an eye movement that changed direction after saccade initiation. As such, the coding of a response in the wrong direction had different meaning for manual versus saccadic responses.

Because the error data do not offer insight into the mechanisms underlying response production in the present set of experiments, there is not a strong motivation to analyze the data separately according to type of error. Instead, the results for each S1-S2 response combination proceeds with an analysis of the error data, collapsed over type of error. This, in essence, offers a measure of whether data was lost differentially across cells of the experimental design and also circumvents the difficulties associated with cells that lack variance ($M = 0$).

Consideration of the overall error data is followed, where applicable, by analysis of the RTs to S1. Even though S2 was executed following S1, S2 and the location it signalled relative to S1 were dummy-coded and included in the analysis. In other words, the S1 data is separated according to the cell in which the S1 responses fell, even though these responses preceded the S2 response. Of course, the expectation is that, if anything, S1 responses should be sensitive only to S1 and not to any other factors in the design. The purpose of analyzing this data according to the full factorial design is to determine whether any RT differences to S1 might have accounted for any observed RT differences to S2.

Following analysis of the error data and, if applicable, the RTs to S1, RTs to S2 are analyzed. It should be noted that these RTs include only those trials for which fixation was maintained when appropriate and when no error was recorded for the response to S2.

The analyses of errors and RTs are within-subjects analyses of variance

(ANOVAs), with S1 (exogenous, endogenous), S2 (exogenous, endogenous), and relative locations signalled by S1 and S2 (different, same) as factors. Given the centrality of the three-way interaction to identifying those signal combinations that produce IOR, planned comparisons (using pooled error terms) on this interaction will be performed on the RT data to S2, regardless of omnibus significance (cf. Keppel, 1982). These comparisons will determine the occurrence of IOR for each of the exogenous-exogenous, endogenous-exogenous, exogenous-endogenous, and endogenous-endogenous combinations of S1 and S2. Also, to give a sense of the ubiquity of any findings that are revealed by the contrast analysis, the number of subjects showing an effect in the same direction as the overall contrast (i.e., facilitation versus inhibition) will be noted.

Note that, for convenience, the difference in RT to S2 when it signals the same versus a different location than S1 will be referred to as a measure of IOR. This terminology does not presuppose a significant inhibitory effect for same- versus different-location RTs. Indeed, whereas positive values for this difference measure indicate slower responding when the locations signalled by S1 and S2 are the same versus different (i.e., IOR), negative values indicate the opposite (i.e., facilitation).

In most response-response paradigms, the response-stimulus interval (RSI) controls stimulus onset, rather than SOA. Because that was not the case here (where S1-S2 SOA was fixed), to determine whether the magnitude of any IOR (or facilitatory) effects in the present experiments are related to the speed of responding to S1 in response-response conditions, simple regression analyses will be carried out on mean RT to S1 and mean IOR measured by S2 for each of the exogenous-exogenous, exogenous-endogenous, endogenous-exogenous, and endogenous-endogenous combinations of S1 and S2. These

analyses will highlight any tendency for IOR to S2 to be dependent on RTs to S1.

Correlation analysis will also be used to explore any systematic increase or decrease in the magnitude of obtained IOR effects that might occur as a function of the number of sessions preceding an experimental block (i.e., practice). Consider that the within-subjects design requires that all subjects perform in six experimental sessions. As such, for each response combination, subjects had 0-5 blocks of previous experience with the signal combinations used. As such, for each combination of S1 (exogenous, endogenous) and S2 (exogenous, endogenous), the magnitude of IOR will be examined as a function of whether the particular response combination represented the first to the sixth session (block) for each subject. All statistical tests will be evaluated at a .05 error level.

Following each experiment, the general data pattern will be summarized (see also Appendix A for a summary table of significant effects to emerge from analysis of individual experiments) with reference to patterns in the existing literature (where applicable). However, there will not be an attempt to define the mechanisms or measures of IOR on the basis of data from individual experiments. Instead, the issues of what is inhibited and how IOR is generated will be grappled with once the data patterns have been defined for each dependent measure.

MANUAL RESPONSES AS A MEASURE OF IOR

EXPERIMENT 1: NO RESPONSE-MANUAL

In Experiment 1, subjects were required to withhold responses to exogenous and endogenous S1s and to make speeded manual responses to

exogenous and endogenous S2s. As summarized in Table 2, the literature has examined the No Response-Manual combination only under the exogenous-exogenous combination of S1 and S2. This combination represents the typical paradigm for examining IOR - a peripheral luminance cue precedes a peripheral onset target - and has been repeatedly shown to produce IOR at relatively long S1-S2 SOAs (e.g., Maylor, 1985; Posner & Cohen, 1984; Rafal et al., 1989; Tassinari et al., 1987). There are no instances of similar exogenous cues being used to examine manual responses to endogenous S2s (i.e., the exogenous-endogenous combination). For this reason, IOR for manual RTs has been characterized as a motor bias against responding to peripherally-presented targets. To determine whether a peripherally-presented stimulus is required in order to obtain IOR for a manual response, it is critical that a motor response be elicited in the absence of peripheral stimulation. Experiment 1 will explore this possibility by examining IOR for manual responses that are directed by endogenous S2s.

Whereas endogenous signals have not been used in the literature as S2, there are cases in which endogenous signals have been employed as S1 (e.g., Posner & Cohen, 1984; Rafal et al., 1989); however, as indicated previously, this has only occurred under manipulations of target probability. Because higher target probabilities are intended to encourage the top-down allocation of attentional resources, endogenous signals that have predictive validity do not require "no response"; they require no *overt* response - attending in the cued direction without making an eye movement *is* a complex spatial response. Under the demands of covert orienting, IOR does not occur for manual responses that follow endogenous signals (at least, not when S2 is an exogenous peripheral onset; cf. Posner & Cohen, 1984; Rafal et al., 1989). The current experiment will determine whether an endogenous

signal that requires neither an overt *nor* a covert response will - perhaps by virtue of its association with a motor response (i.e., because of need to respond to endogenous S2s) - generate IOR for manual RTs to exogenous and endogenous S2s. This will determine whether the generation of IOR for manual responses is dependent on peripheral stimulation per se or whether it is dependent on having *any* spatial signal that is unconfounded with endogenous covert orienting.

Thus, the present experiment will attempt to replicate the exogenous-exogenous IOR effects that are observed consistently in the literature, explore the effects of an exogenous S1 on an endogenous S2, and will employ a non-informative (with respect to S2) endogenous S1 in order to examine the hitherto untested endogenous-exogenous and endogenous-endogenous cells of this design.

Experiment 1: Results

Errors

As seen in Table 7, when subjects ignored S1 and made a manual response to S2, the greatest loss of data due to a single type of error in a cell was just over 4%. When collapsed over type of error (directional errors, misses, anticipations), analysis revealed only a significant effect of S2 ($F(1,17)=13.57, MSe=4.98, p<.002$), with greater overall errors when S2 was endogenous (1.7%) versus exogenous (0.3%). No other main effects ($Fs<1$) were significant, nor were the interactions of S1 and S2 ($F<1$), of S1 and relative locations signalled by S1 and S2 ($F<1$), of S2 and relative locations signalled by S1 ($F(1,17)=2.04, MSe=2.37, p<.17$), or of S1, S2 and relative locations signalled by S1 and S2 ($F<1$).

RTs to S2

RTs for trials on which no errors were made in responding to S2 are

shown in Figure 6, according to S1 (exogenous, endogenous), S2 (exogenous, endogenous), and relative locations signalled by S1 and S2 (different, same). Analysis revealed no difference in manual RTs to S2 as a function of whether S1 was exogenous or endogenous ($F(1,17)=2.54$, $MSe=159.30$, $p<.13$). However, there was a significant main effect for S2 ($F(1,17)=88.65$, $MSe=694.79$, $p<.00$). This effect can be seen in Figure 6 as overall slower RTs made to endogenous signals ($M=306$ msec) than to exogenous signals ($M=265$ msec). As seen above, in addition to slower RTs to endogenous versus exogenous S2s, more errors occurred following the former than the latter, thus countering the suggestion of a speed-accuracy trade-off for S2 responses. Returning to consideration of the RT data, analysis also revealed a significant main effect for relative locations signalled by S1 and S2 ($F(1,17)=8.56$, $MSe=415.72$, $p<.01$). This main effect reflects overall IOR, with slower RTs when S2 and S1 signal the same ($M=291$ msec) versus different ($M=281$ msec) locations.

There were no significant two-way interactions between S1 and S2 ($F<1$), or between S1 and the relative locations signalled by S1 and S2 ($F<1$). There was, however, a significant two-way interaction between the S2 and the relative locations signalled by S1 and S2 ($F(1,17)=12.30$, $MSe=205.88$, $p<.00$). This interaction is seen in Figure 6 as no difference in the RTs when an endogenous S2 signals the same ($M=307$ msec) versus a different ($M=306$ msec) location than S1 (collapsed over the outline and filled squares of Figure 6) but slower RTs when an exogenous S2 signals the same ($M=274$ msec) versus a different ($M=256$ msec) location than S1 (collapsed over the outline and filled circles of Figure 6). More simply: IOR is measured by manual RTs to exogenous signals but not by manual RTs to endogenous signals.

The three-way interaction between the S1, S2, and relative locations signalled by S1 and S2 was not significant ($F<1$). Where the first indicant

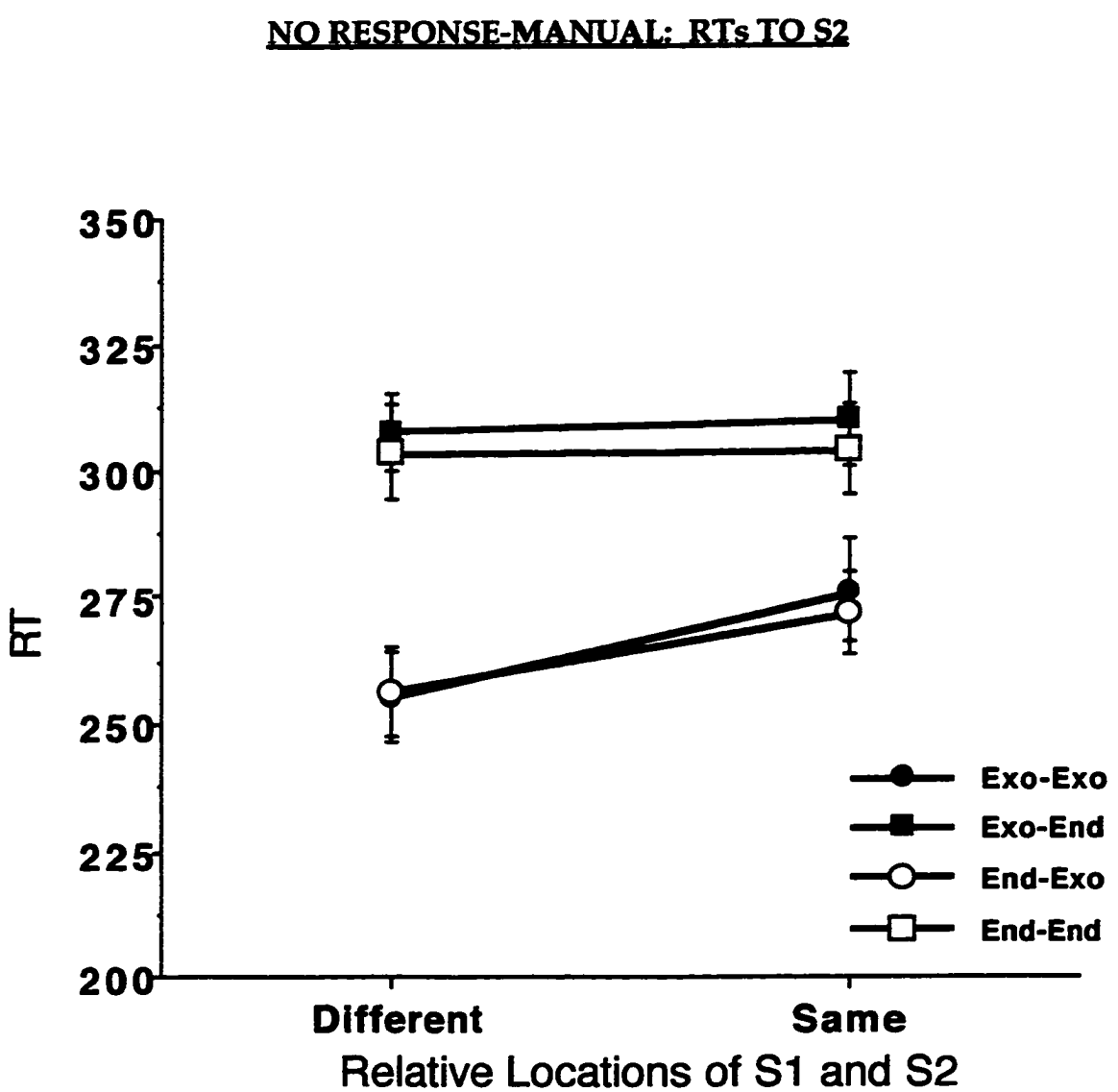

Figure 6. Experiment 1: Manual RT to S2, as a function of the relation between S1 and S2 locations (different, same) and signals (exo: exogenous, end: endogenous).

refers to S1, the second to S2, and the number of subjects showing an effect in the same direction as the overall mean for a cell is shown parenthetically, planned comparisons on this three-way interaction revealed a significant 21 msec IOR effect (n=18) for the exogenous-exogenous combination ($F(1,17)=14.45$, $MSe=272.02$, $p<.00$); a significant 16 msec IOR effect (n=17) for the endogenous-exogenous combination ($F(1,17)=8.21$, $MSe=272.02$, $p<.01$); a non-significant 3 msec IOR effect (n=11) for the exogenous-endogenous combination ($F<1$); and, a non-significant 1 msec IOR effect (n=7) for the endogenous-endogenous combination ($F<1$). These data are shown in Table 8.

IOR as a Function of Block Number

To determine whether there were any systematic effects of practice, correlations were examined between the magnitude of the IOR effect for each combination of S1 and S2, as a function of the block number in which subjects performed Experiment 1. Scatterplots of these data are shown in Figure 7. None of the correlations were significant. In particular, the correlations were: -0.26 for the exogenous-exogenous combination ($F(1,17)=1.14$, $MSe=601.31$, $p<.30$); -0.01 for the endogenous-exogenous combination ($F<1$); -0.16 for the exogenous-endogenous combination ($F<1$); and, -0.23 for the endogenous-endogenous combination ($F<1$).

Experiment 1: Summary

As seen in Table 8, Experiment 1 replicates the reported ability to obtain IOR for manual responses made to an exogenous S2 that follows an exogenous S1. In contrast to this significant IOR effect for the exogenous-exogenous combination, when the same exogenous S1 is used to generate IOR for an *endogenous* S2 (i.e., the exogenous-endogenous combination), IOR does not occur. Similarly, an endogenous S1 results in IOR for exogenous S2s

EXPERIMENT 1 VERSUS THE LITERATURE: SUMMARY OF IOR IN THE NO RESPONSE-MANUAL COMBINATION, AS A FUNCTION OF S1 AND S2

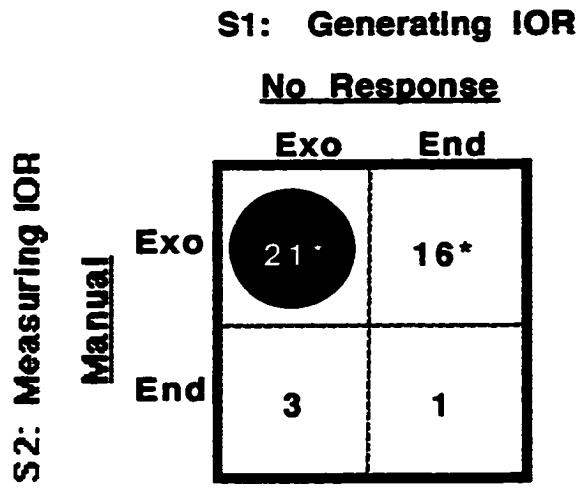

Table 8. Magnitude of IOR in Experiment 1. Significant effects are indicated with "". Positive values reflect IOR, negative values reflect facilitation. For comparison, cells that have been tested in the literature are indicated with circles (the black colouring indicates that IOR was observed).*

NO RESPONSE-MANUAL: CORRELATIONS OF IOR WITH EXPERIMENTAL BLOCK NUMBER

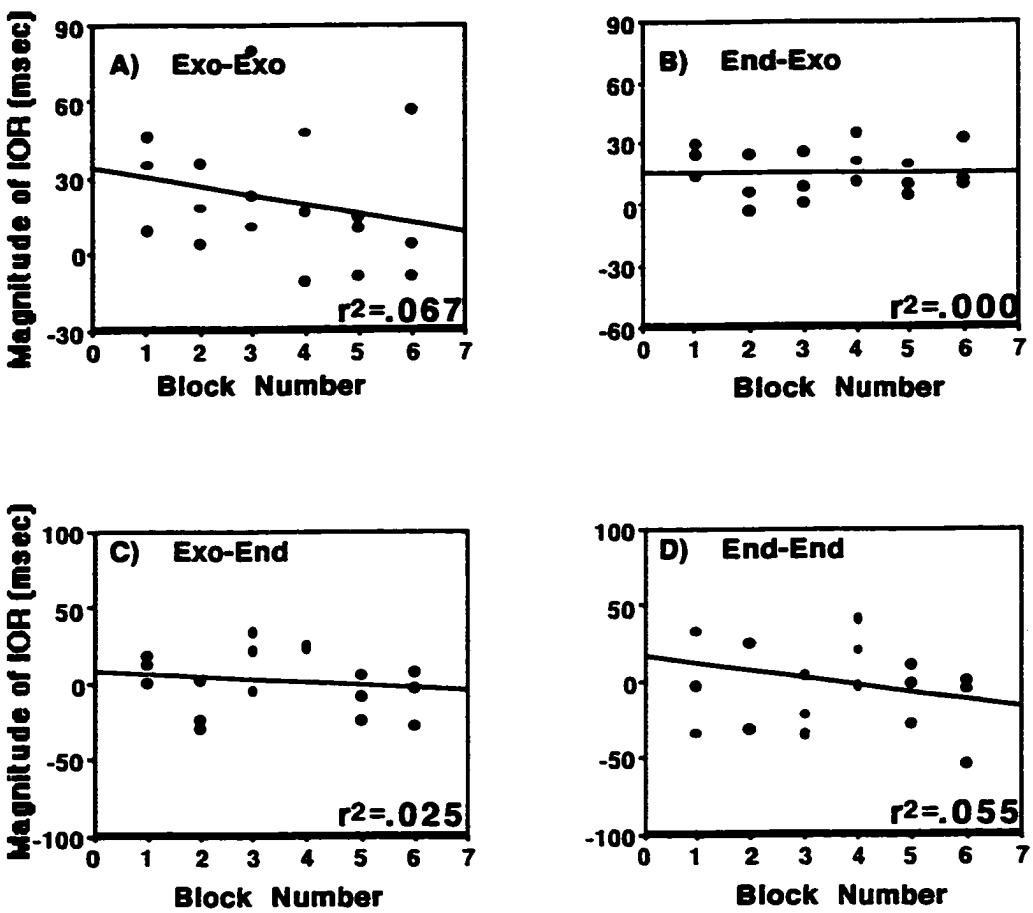

Figure 7: Experiment 1: Scatterplots of the magnitude of IOR versus experimental block number for the exogenous-exogenous (A), endogenous-exogenous (B), exogenous-endogenous (C), and endogenous-endogenous (D) combinations of S1-S2.

(i.e., the endogenous-exogenous combination) but not for endogenous S2s (i.e., the endogenous-endogenous combination).

To use the motor/visuo-motor distinction discussed earlier with reference to the nature of S2, IOR generated by both exogenous and endogenous S1s that require no response is revealed as a visuo-motor inhibition: Identical manual responses exhibit IOR when commanded by visual signals in the periphery (i.e., by exogenous S2s) but not when commanded by arrows presented centrally (i.e., by endogenous S2s). In other words, manual responses are inhibited only when they are directed by a visual signal that occurs in the same location as had been signalled by the preceding S1. The nature of the S1 signal itself is inconsequential to the generation of this inhibition. A possible mechanism for the generation of IOR by a centrally-presented arrow will be examined in the General Discussion.

Together, the results of Experiment 1 suggest that withholding a response to a signal - be it exogenous or endogenous - generates a visuo-motor inhibition for manual responses. Experiment 2 will explore whether the same inhibitory effect may operate when a manual response is, instead, required to the first event.

EXPERIMENT 2: MANUAL-MANUAL

In Experiment 2, subjects were required to make speeded manual localization responses to exogenous and endogenous S1 and S2s. As summarized in Table 2, the literature has examined Manual-Manual responses only for the exogenous-exogenous combination.

Tests reported in the literature of the exogenous-exogenous cell are based on response-response paradigms in which no distinction is made

between S1 and S2. For example, the exogenous-exogenous condition has been examined (e.g., Kwak & Egeth, 1992; Maylor & Hockey, 1985, 1987; Tanaka & Shimojo, 1995) by presenting subjects with a continuous series of onset targets, separated in time by a variable RSI. Each target in the series requires a manual detection response, and IOR is evaluated as slowed RT to a target on trial N when it occurs in the same position as the target on trial N-1. Representing IOR in this paradigm, negative sequential dependency in manual RT occurs as function of RSI and spatial congruity of successive targets. However, note that in such a paradigm, establishing IOR cannot be successfully distinguished from measuring it: Manual responses to all but the first and last targets contribute both to the generation and to the measurement of obtained inhibitory effects.

To the extent that the minimum interval between the S2 on trial N-1 and the S1 on trial N are separated by an interval that extends beyond the 3 sec window that is characteristic of IOR, Experiment 2 eliminates the confounding of IOR generation and measurement by virtue of the discrete-trials procedure for which there is a functional delineation of S1 and S2: Manual responses to exogenous and endogenous S1s generate IOR; manual responses to exogenous and endogenous S2s measure the IOR. Indeed, the minimum interval between the presentation of an S2 and the presentation of the S1 of the following trial is beyond the typical 3 sec window for obtaining IOR and is the sum of the RT to S2 on trial n-1, the 3000 msec inter-trial interval, the 500 msec fixation interval, and the 1000 msec S1/S2 SOA. As such, Experiment 2 will clarify results reported for the exogenous-exogenous cell and, in addition, will assess whether IOR occurs for endogenous-exogenous, exogenous-endogenous, and endogenous-endogenous combinations.

Experiment 2: Results

Errors

As seen in Table 7, when subjects made manual responses to both S1 and S2, the greatest loss of data to a single type of error in any one cell was just over 5%. Collapsed over the type of error that could be made (directional errors, misses, anticipations), analysis revealed a significant main effect for S2 ($F(1,17)=17.18$, $MSe=6.04$, $p<.00$): More errors were made overall when the S2 was endogenous ($M=2.6\%$) than when it was exogenous ($M=0.8\%$). The main effects of S1 and of relative locations signalled by S1 and S2 were not significant (both $Fs<1$). Likewise, the two-way interactions between S1 and S2 ($F<1$), between S1 and the relative locations signalled by S1 and S2 ($F<1$), and between S2 and relative locations signalled by S1 and S2 ($F(1,17)=2.08$, $MSe=1.26$, $p<.17$) were not significant. The three-way interaction between S1, S2, and relative locations signalled by S1 and S2 was also not significant ($F<1$).

RTs to S1

RTs for correct responses made to S1 are shown in Table 7. Analysis of these RTs revealed a significant main effect for S1 ($F(1,17)=7.12$, $MSe=592.41$, $p<.02$): When subjects made a manual response to an endogenous S1, RTs to that signal were slower ($M=285$ msec) than when they made a manual response to an exogenous S1 ($M=274$ msec). All other main effects ($Fs<1$), two-way interactions ($Fs<1$), and the three-way interaction ($F(1,17)=3.76$, $MSe=83.62$, $p<.07$) were not significant.

RTs to S2

Manual RTs made without error to S2 are shown in Figure 8, as a function of S1 (exogenous, endogenous), S2 (exogenous, endogenous), and the relative locations signalled by S1 and S2 (different, same). Analysis of these RTs revealed no difference in the speed of responding to S2 as a

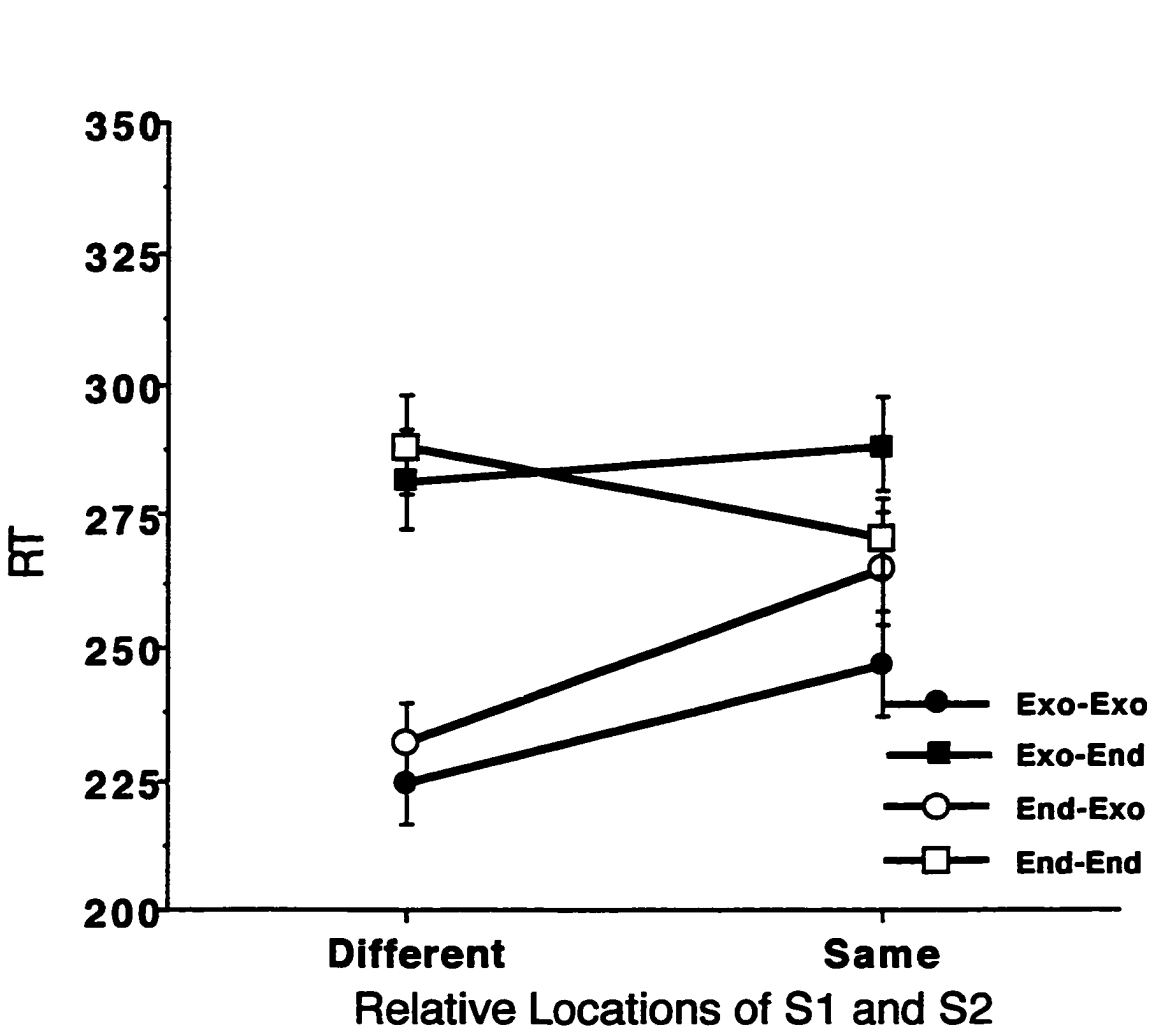

Figure 8. Experiment 2: Manual RT to S2, as a function of the relation between S1 and S2 locations (different, same) and signals (exo: exogenous, end: endogenous).

function of whether the preceding S1 had been exogenous (M=260 msec) versus endogenous (M=264 msec; $F(1,17)=2.75$, $MSe=181.32$, $p<.12$). However, manual responses were significantly faster to an exogenous (M=242 msec) versus an endogenous (M=282 msec) S2 ($F(1,17)=111.94$, $MSe=516.74$, $p<.00$). There was also a main effect for the relative locations signalled by S1 and S2 ($F(1,17)=8.41$, $MSe=510.36$, $p<.01$): RTs were 268 msec when S2 signalled the same location as S1 and were 257 msec when S2 signalled a different location from S1.

The two-way interaction of S1 and S2 was significant ($F(1,17)=18.29$, $MSe=173.34$, $p<.00$). This interaction stems from the observation that RTs to an exogenous S2 were faster when preceded by an exogenous (M=235 msec) versus an endogenous (M=249 msec) S1, whereas RTs to an endogenous S2 were faster when preceded by an endogenous (M=279 msec) versus an exogenous (M=285 msec) S1. The two-way interaction of S1 and the relative locations signalled by S1 and S2 was not significant ($F(1,17)=3.17$, $MSe=135.49$, $p<.09$). However, the two-way interaction of S2 and the relative locations signalled by S1 and S2 was significant ($F(1,17)=16.10$, $MSe=609.86$, $p<.00$). This interaction stems from the fact that, collapsed across S1, an IOR effect occurs only for an exogenous S2: RTs were slower when an exogenous S2 signalled the same (M=256 msec) versus a different (M=228 msec) location than S1; RTs were faster when an endogenous S2 signalled the same (M=279 msec) versus a different (M=285 msec) location than S1.

Finally, with respect to RTs made to S2, the three-way interaction of S1, S2, and the relative locations signalled by S1 and S2 was significant ($F(1,17)=15.05$, $MSe=178.24$, $p<.00$). Where the first and second indicant refer to S1 and S2, respectively, and the parenthetical value indicates the number of subjects who showed an effect in the same direction as the overall mean

(i.e., overall IOR or facilitation), planned contrasts on this interaction revealed a significant 22 msec IOR effect (n=15) for the exogenous-exogenous combination ($F(1,17)=25.00$, $MSe=178.24$, $p<.00$); a significant 33 msec IOR effect (n=18) for the endogenous-exogenous combination ($F(1,17)=53.71$, $MSe=178.24$, $p<.00$); a non-significant 6 msec IOR effect (n=8) for the exogenous-endogenous combination ($F(1,17)=2.12$, $MSe=178.24$, $p<.16$); and, a significant 18 msec facilitatory effect (n=12) for the endogenous-endogenous combination ($F(1,17)=15.79$, $MSe=178.24$, $p<.00$). These results are summarized in Table 9.

RTs to S1 versus IOR

The magnitude of IOR was examined as a function of the speed to make the manual response to S1. Scatterplots of these data are shown in Figure 9. Referring to the S1 and S2s, respectively, the only significant correlation was for the endogenous-exogenous combination ($r=.64$, $F(1,17)=11.04$, $MSe=331.52$, $p<.00$): As RTs to make a manual response to an endogenous S1 increased, so did the magnitude of the IOR effect measured by manual RTs to an exogenous S2.

None of the other correlation coefficients approached significance: For the exogenous-exogenous combination the correlation was 0.27 ($F(1,17)=1.2$, $MSe=449.38$, $p<.29$); for the exogenous-endogenous combination, the correlation was 0.28 ($F(1,17)=1.39$, $MSe=1034.60$, $p<.26$); and, for the endogenous-endogenous combination, the correlation was -0.15 ($F<1$).

IOR as a Function of Block Number

For each of the combinations of S1 and S2, the magnitude of the IOR effect was correlated with the block number in which subjects performed Experiment 2. Indicating that there were no systematic effects of practice on the magnitude of IOR, none of the correlations, shown in Figure 10, were

EXPERIMENT 2 VERSUS THE LITERATURE: SUMMARY OF IOR IN THE MANUAL-MANUAL COMBINATION, AS A FUNCTION OF S1 AND S2

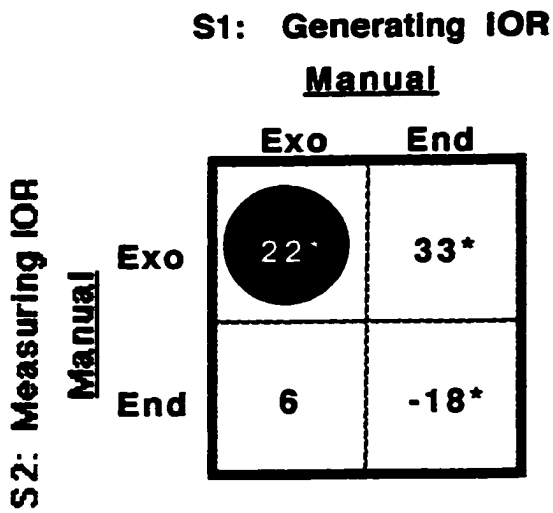

Table 9. Magnitude of IOR in Experiment 2. Significant effects are indicated with "". Positive values reflect IOR, negative values reflect facilitation. For comparison, cells that have been tested in the literature are indicated with circles (the black colouring indicates that IOR was observed).*

MANUAL-MANUAL: CORRELATIONS OF IOR WITH S1 RT

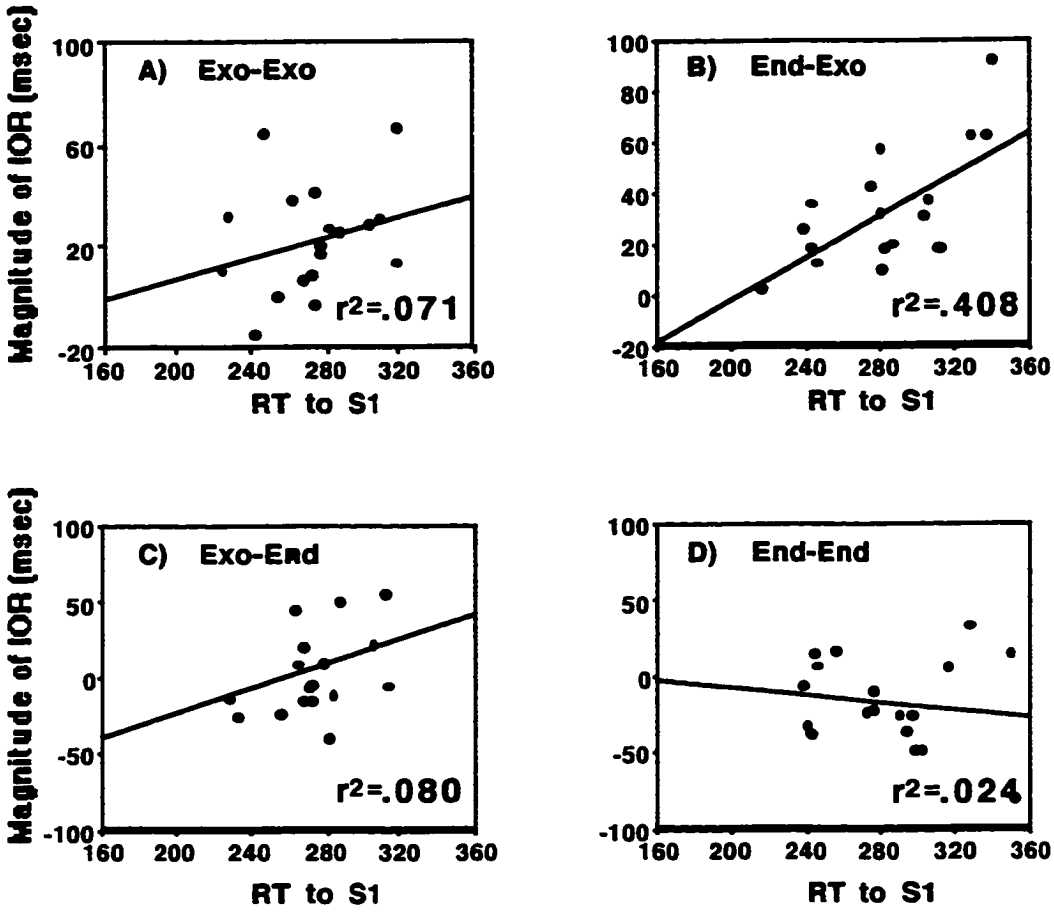

Figure 9. Experiment 2: Scatterplots of S1 latencies versus the magnitude of IOR for the exogenous-exogenous (A), endogenous-exogenous (B), exogenous-endogenous (C), and endogenous-endogenous (D) combinations of S1-S2.

MANUAL-MANUAL: CORRELATIONS OF IOR WITH EXPERIMENTAL BLOCK NUMBER

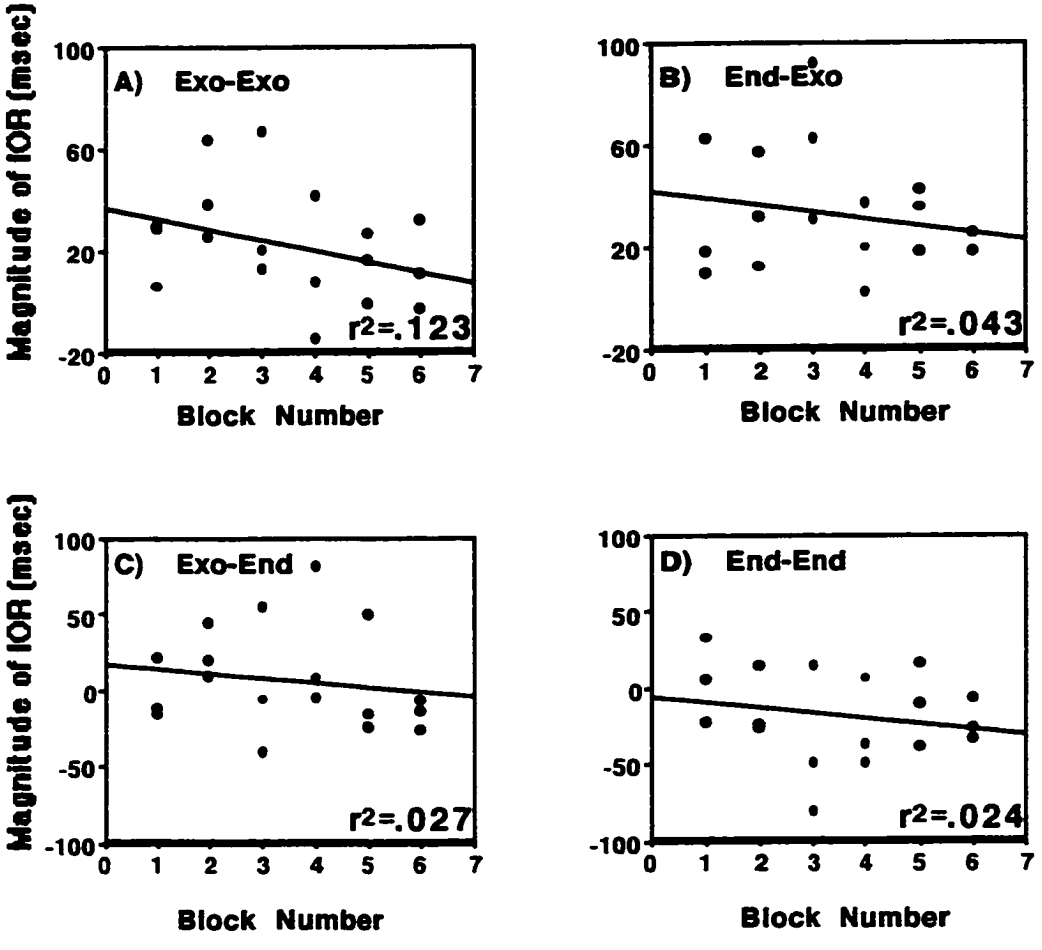

Figure 10. Experiment 2: Scatterplots of the magnitude of IOR versus experimental block number for the exogenous-exogenous (A), endogenous-exogenous (B), exogenous-endogenous (C), and endogenous-endogenous (D) combinations of S1-S2.

significant. For the exogenous-exogenous combination, the correlation was -0.35 ($F(1,17)=2.24$, MSSE=424.2, $p<.15$); for the endogenous-exogenous combination, the correlation was -0.21 ($F<1$); for the exogenous-endogenous combination, the correlation was -0.17 ($F<1$); and, for the endogenous-endogenous combination, the correlation was -0.21 ($F<1$).

Experiment 2: Summary

As seen in Table 9, even after eliminating the confounding of IOR generation and measurement that occurs in response-response tasks which fail to use a discrete-trials procedure, the results of Experiment 2 uphold the conclusion that IOR is obtained for the exogenous-exogenous combination of Manual-Manual responses. In addition, Experiment 2 reveals that IOR occurs for the endogenous-exogenous but not for the exogenous-endogenous or endogenous-endogenous combinations. Notice the correspondence between this pattern of results and that obtained for Experiment 1. Clearly, the visuo-motor inhibition that is suggested by slowed manual RTs to exogenous but not to endogenous S2s does not require that subjects withhold a response to S1: When subjects made manual responses to exogenous or endogenous S1s, similar visuo-motor inhibition was observed.

Beyond this visuo-motor inhibition, there were two surprising observations that require further consideration. The first is in the endogenous-endogenous condition for which significant facilitation was observed. What makes this finding particularly difficult to explain is the fact that it cannot represent either strictly motor response priming nor strictly perceptual priming. Consider that all the cells of Experiment 2 required the execution of two manual responses. Yet, in no other cell was there evidence that responses were primed by repetition - indeed, the finding of significant IOR for the exogenous-exogenous and endogenous-exogenous cells indicates

that motor repetition was slowed relative to motor alternation. Likewise, it is not possible to conclude that the repetition of an identical endogenous signal per se results in perceptual priming. This follows from the observation that the endogenous-endogenous cell of Experiment 1 failed to show facilitation due to the repetition of the same endogenous signal and - as will be seen - significant IOR is obtained for this cell when other combinations of S1-S2 response are employed.

Beyond motor or perceptual priming effects, another possibility for explaining endogenous-endogenous facilitation is that it represents the early portion of a biphasic pattern of results. However, this seems unlikely. No facilitation was observed in Experiment 1 for this cell. Moreover, as will be seen, the Manual-Manual endogenous-endogenous cell is the only one across all 6 experiments to show significant facilitation. Given that facilitation is not consistently observed for endogenous-endogenous combinations, it seems unreasonable to presume that the facilitatory effect observed in Experiment 2 was due to inadequate selection of S1-S2 asynchrony. As such, the rather unsatisfactory conclusion is merely a restatement of the data itself: Repetition of manual responses to repeated endogenous stimuli results in some form of perceptual-response priming that is specific to that response/stimulus combination.

The second surprising finding is the significant positive correlation between RTs to S1 and the magnitude of IOR in the endogenous-exogenous combination. The analysis of correlations between S1 RTs and the magnitude of IOR was to determine whether the control of stimulus timing by SOA rather than RSI might have impacted the findings. The significant positive correlation indicates that the magnitude of IOR tends to increase with the latency of responses to S1. Normally, in response-response paradigms, IOR is

greater at long versus short RSIs. Given that a long-latency response to S1 would effectively decrease the RSI, had the correlation been negative, it would suggest that IOR might have been obtained had RSI rather than S1-S2 SOA been employed. However, given that the correlation was positive, there is little reason to be concerned that the data were compromised by this relation.

These two unexpected findings notwithstanding, the results of Experiment 2 converge with the results of Experiment 1 in suggesting that manual RTs show visuo-motor inhibition following both exogenous and endogenous S1s. There is no change in the pattern of results when S1 requires no response (Experiment 1) versus when S1 requires a manual response (Experiment 2). Experiment 3 will determine whether the nature of this inhibitory effect remains the same when a saccadic response is made to S1.

EXPERIMENT 3: SACCADIC-MANUAL

In Experiment 3, subjects made a saccade to the location indicated by an exogenous or endogenous S1, returned gaze to fixation upon centre brightening, and then made a manual response to an exogenous or endogenous S2. As seen in Table 2, the two cells that have been tested for this Saccadic-Manual response combination are exogenous-exogenous, and endogenous-exogenous; both combinations showed IOR.

As may be recalled, the exogenous-exogenous and endogenous-exogenous combinations were part of Rafal et al.'s (1989) test of the oculomotor view of IOR generation. In their saccade-execution condition, subjects were required to move their eyes to the location that was signalled by either a peripheral luminance cue or else by an endogenous arrow at fixation

(see also Posner & Cohen, 1984). At short SOAs, a manual detection target appeared with 80% probability at the location to which the subject had saccaded; at long SOAs, a central brightening summoned fixation back to centre and a target appeared with equal probability at one of the two peripheral locations. Whether subjects saccaded to an exogenous S1 or to an endogenous S1, IOR was observed for manual RTs to the onset targets.

The current Experiment 3 will attempt to replicate Rafal et al.'s (1989) finding of IOR in the exogenous-exogenous and endogenous-exogenous cells of the Saccadic-Manual response combination. In addition, Experiment 3 will determine whether saccades to endogenous and exogenous S1s also result in IOR that can be measured by manual responses to endogenous S2s.

Experiment 3: Results

Errors

As seen in Table 7, the most data lost due to a single type of error in any cell of the Saccadic-Manual condition was just over 5%. Collapsed over type of error (directional errors, misses, anticipations), an analysis revealed only a significant main effect of S2 ($F(1,17)=13.69$, $MSe=2.35$, $p<.00$), that stemmed from a tendency for greater errors to be committed following an endogenous ($M=1.2\%$) versus an exogenous ($M=0.3\%$) S2. The main effects of S1 ($F(1,17)=1.58$, $MSe=1.03$, $p<.23$) and of relative locations signalled by S1 and S2 ($F(1,17)=2.07$, $MSe=3.43$, $p<.17$) were not significant. The two-way interactions of S1 and S2 ($F(1,17)=2.29$, $MSe=0.99$, $p<.15$), of S1 and relative locations signalled by S1 and S2 ($F<1$), of S2 and relative locations signalled by S1 and S2 ($F(1,17)=1.87$, $MSe=4.49$, $p<.19$), and the three-way interaction of S1, S2, and relative locations signalled by S1 and S2 ($F(1,17)=3.22$, $MSe=.70$, $p<.09$) were all not significant.

RTs to S1

Analysis of the saccadic RTs made to S1 revealed no significant main effects ($Fs<1$). There were also no significant interactions: Between S1 and S2s ($F<1$); between S1 and the relative locations signalled by S1 and S2 ($F(1,17)=1.31$, $MSe=92.63$, $p<.27$); between S2 and the relative locations signalled by S1 and S2 ($F(1,17)=1.02$, $MSe=88.74$, $p<.33$); or, between S1, S2 and the relative locations signalled by S1 and S2 ($F<1$).

RTs to S2

Manual RTs to S2 on those trials for which no errors occurred are shown in Figure 11, according to S1 (exogenous, endogenous), S2 (exogenous, endogenous), and the relative locations signalled by S1 and S2 (different, same). Suggesting that there is an alerting effect of making a saccade to an exogenous signal, analysis of these RTs revealed a significant main effect for S1 ($F(1,17)=14.83$, $MSe=180.23$, $p<.00$) wherein manual RTs to S2 were faster when they followed saccades made to an exogenous S1 ($M=298$ msec; collapsed across the solid lines of Figure 11) than when they followed saccades made to an endogenous S1 ($M=306$ msec; collapsed across the dashed lines of Figure 11); as will be seen, this main effect of S1 did not interact with any other factors. Manual RTs to S2 were also faster when they were made to an exogenous ($M=286$ msec) rather than to an endogenous ($M=318$ msec) signal ($F(1,17)=85.36$, $MSe=441.70$, $p<.00$). With respect to the relative locations signalled by S1 and S2, manual RTs showed significant overall IOR ($F(1,17)=25.68$, $MSe=660.01$, $p<.00$), with a mean RT of 313 msec when the locations were the same, and a mean RT of 291 msec when the locations were different.

The two-way interactions of S1 and S2 ($F(1,17)=3.02$, $MSe=264.04$, $p<.10$) and of S1 and relative locations signalled by S1 and S2 ($F<1$) were not

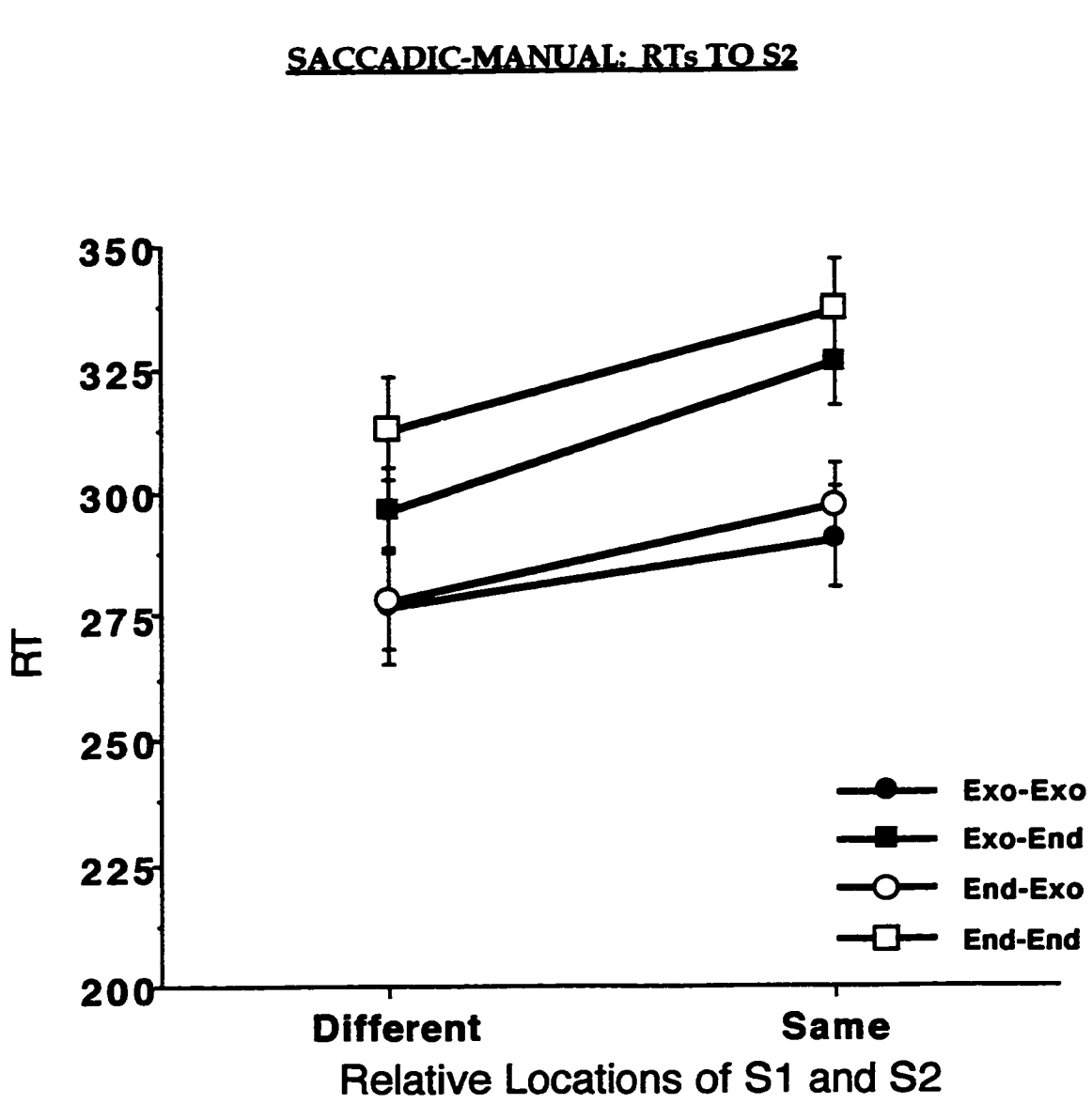

Figure 11. Experiment 3: Manual RT to S2, as a function of the relation between S1 and S2 locations (different, same) and signals (exo: exogenous, end: endogenous).

significant. The two-way interaction of S2 and relative locations signalled by S1 and S2 was significant ($F(1,17)=4.65$, $MSe=217.48$, $p<.05$). This interaction reflects a greater IOR for endogenous (27 msec) than for exogenous (17 msec) S2s.

The three-way interaction of S1, S2, and relative locations signalled by S1 and S2 was not significant ($F(1,17)=1.59$, $MSe=171.83$, $p<.22$). Where the number of subjects showing an effect in the same direction as the overall cell mean is indicated parenthetically, planned comparisons on this non-significant three-way interaction revealed a significant 14 msec IOR effect (n=13) for the exogenous-exogenous combination ($F(1,17)=9.87$, $MSe=171.83$, $p<.01$); a significant 19 msec IOR effect (n=16) for the endogenous-exogenous combination ($F(1,17)=19.05$, $MSe=171.83$, $p<.00$); a significant 30 msec IOR effect (n=16) for the exogenous-endogenous combination ($F(1,17)=46.64$, $MSe=171.83$, $p<.00$); and, a significant 24 msec IOR effect (n=15) for the endogenous-endogenous combination ($F(1,17)=30.58$, $MSe=171.83$, $p<.00$) of S1 and S2, respectively. These results are summarized in Table 10.

RTs to S1 versus IOR

Scatterplots of the relation between IOR and RTs to S1 are shown in Figure 12. Correlations calculated for the magnitude of the IOR effect measured by the manual response to S2 and the speed to make a saccadic response to S1 revealed no significant relations for the exogenous-exogenous ($r=-0.17$; $F<1$), endogenous-exogenous ($r=-0.13$; $F<1$), exogenous-endogenous ($r=0.40$; $F(1,17)=3.12$, $MSe=328.37$, $p<.10$), or endogenous-endogenous ($r=-0.38$; $F(1,17)=2.65$, $MSe=925.87$, $p<.12$) combinations of S1, S2, respectively.

IOR as a Function of Block Number

Scatterplots of the magnitude of the IOR effects and the block number in which subjects performed Experiment 3 are shown in Figure 13.

EXPERIMENT 3 VERSUS THE LITERATURE: SUMMARY OF IOR IN THE SACCADIC-MANUAL COMBINATION, AS A FUNCTION OF S1 AND S2

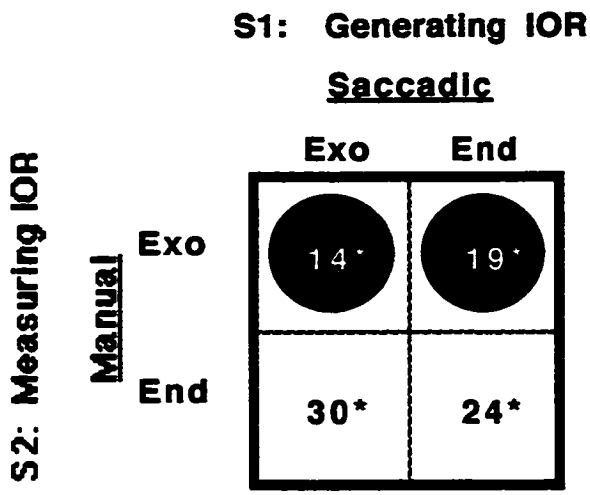

Table 10. Magnitude of IOR in Experiment 3. Significant effects are indicated with "". Positive values reflect IOR, negative values reflect facilitation. For comparison, cells that have been tested in the literature are indicated with circles (the black colouring indicates that IOR was observed).*

SACCADIC-MANUAL: CORRELATIONS OF IOR WITH S1 RT

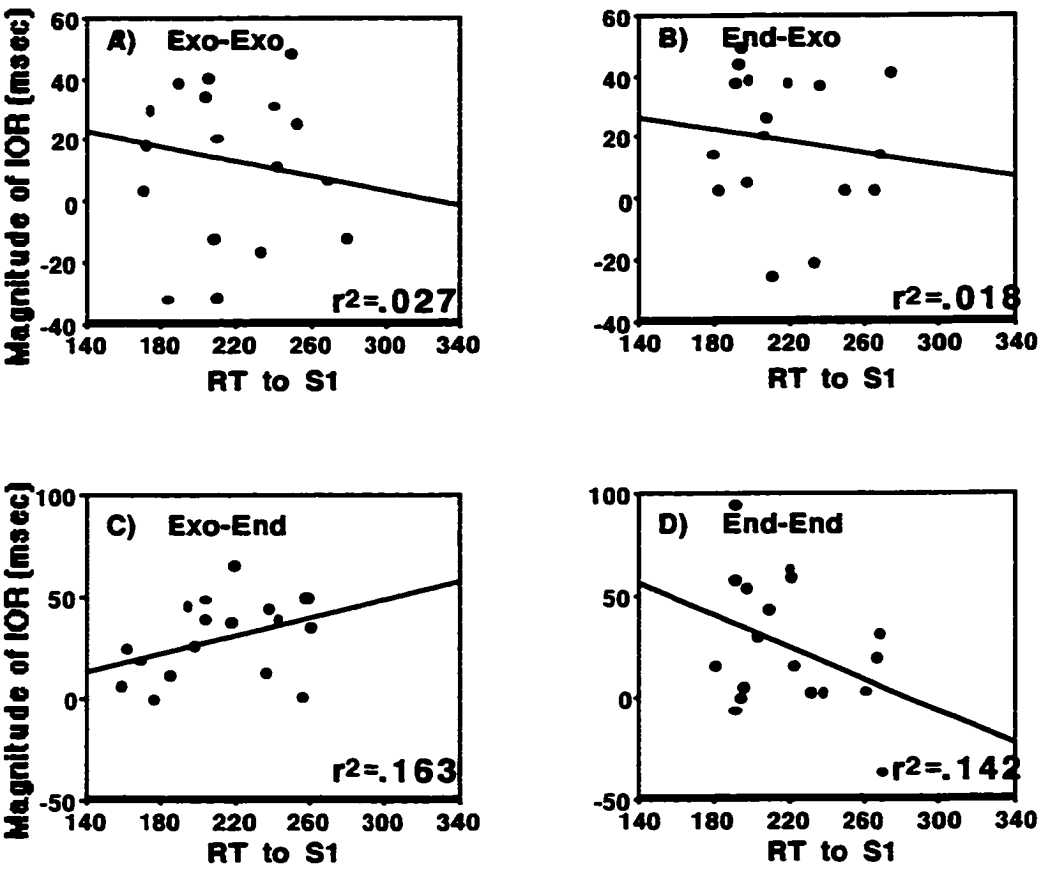

Figure 12. Experiment 3: Scatterplots of S1 latencies versus the magnitude of IOR for the exogenous-exogenous (A), endogenous-exogenous (B), exogenous-endogenous (C), and endogenous-endogenous (D) combinations of S1-S2.

SACCADIC-MANUAL: CORRELATIONS OF IOR WITH EXPERIMENTAL BLOCK NUMBER

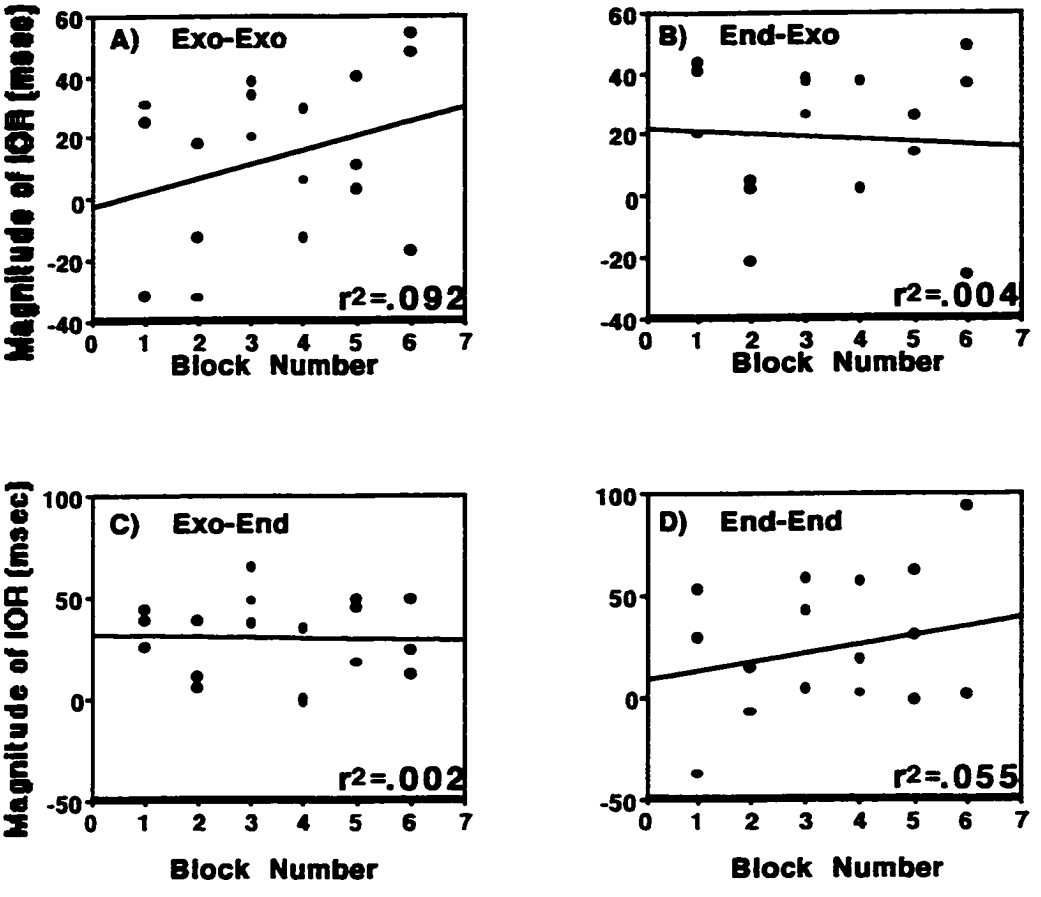

Figure 13. Experiment 3: Scatterplots of the magnitude of IOR versus experimental block number for the exogenous-exogenous (A), endogenous-exogenous (B), exogenous-endogenous (C), and endogenous-endogenous (D) combinations of S1-S2.

Correlational analyses of these data revealed no systematic effects of practice. The correlations were 0.30 for the exogenous-exogenous combination ($F(1,17)=1.62$, $MSe=679.46$, $p<.22$); -0.06 for the endogenous-exogenous combination ($F<1$); -0.05 for the exogenous-endogenous combination; and, 0.23 for the endogenous-endogenous combination ($F<1$).

Experiment 3: Summary

As seen in Table 10, the present Experiment 3 replicated Rafal et al.'s (1989) observation of IOR for the exogenous-exogenous and exogenous-endogenous combinations of Saccadic-Manual responses. The results also revealed significant IOR effects for exogenous-endogenous and endogenous-endogenous combinations. This pattern of results contrasts with those obtained for the No Response-Manual and Manual-Manual response combinations of Experiments 1 and 2, respectively. Whereas Experiments 1 and 2 both revealed a visuo-motor inhibition associated with withholding or else with making manual responses to S1, the results of Experiment 3 suggest that inhibition following a saccadic response to S1 is motoric in nature (i.e., has no visuo-motor component).

This conclusion in favour of a strictly motor inhibition follows from a comparison of IOR effects measured by exogenous versus endogenous S2s. Recall that, theoretically, both signals have the capacity to measure motor response biases and that exogenous signals have the additional capacity to measure inhibitory effects associated with visuo-motor processing/integration. At first blush, the finding of significant IOR for manual responses made to exogenous and endogenous S2s therefore suggests two possibilities. First, the Saccadic-Manual response combination could be revealing a visuo-motor inhibition akin to that observed for the No Response-Manual and Manual-Manual combinations while also

demonstrating additional motor inhibition due to the saccadic response to S1 (thereby accounting for IOR observed for endogenous S2s). Alternatively, the Saccadic-Manual response combination could be revealing *only* a motor inhibition.

As already indicated, the second conclusion - of strictly motor inhibition - is favoured by the present data pattern. This is obvious when the magnitude of the inhibitory effects are examined more closely. If it were the case that the first explanation outlined above were correct, then exogenous S2s would be expected to measure both visuo-motor and motor inhibition whereas the endogenous S2s would be expected to measure only motor inhibition. It follows that exogenous S2s would therefore be expected to show a greater magnitude IOR relative to endogenous S2s, since they would reflect the operation of *two* inhibitory processes (visuo-motor and motor) rather than just one (motor). However, the pattern of results does not support the view that IOR was greater for exogenous versus endogenous S2s. This is true whether the magnitude of IOR is considered in absolute or relative terms.

With respect to the absolute magnitude of IOR, the significant two-way interaction of S2 and relative locations signalled by S1 and S2 demonstrates that IOR for endogenous S2s was actually of *greater* magnitude than IOR for exogenous S2s. Accepting that there was also a main effect for S2, indicating overall slower RTs to endogenous S2s than to exogenous S2s, it is also possible to examine the magnitude of the IOR effects in relative terms. If the overall 17 msec IOR effect for exogenous S2s is taken relative to the 286 msec average RT to these signals, the proportion of IOR:RT was 0.05. In contrast, if the overall 27 msec IOR effect for endogenous S2 is taken relative to the 318 msec average RT to these signals, the proportion of IOR:RT was 0.09. In other words, even in relative proportions, the magnitude of IOR was greater for

endogenous than for exogenous S2s.

Of course, it is not immediately apparent why the postulated motor response inhibition should be greater for endogenous S2s than for exogenous S2s. If these two signals were tapping the same inhibitory mechanism, they might be expected to show similar absolute values - or at least similar relative values. That they do not show the same magnitude inhibition, however, is perhaps accounted for by the fact that the motor inhibition is the same but the signals, nevertheless, are different. That is, the motor inhibition might be countered somewhat by the presentation of a peripheral visual signal (i.e., because there is no visuo-motor inhibition, the visual signal may actually be beneficial) and/or may be exacerbated by the use of a purely symbolic motor command.

Regardless of the reason for the difference, on the strength of the arguments outlined above, the finding that IOR is not greater for exogenous versus endogenous signals suggests that the finding of IOR for all four cells of Experiment 3 reflects the operation of strictly motor inhibition. Unlike when subjects withhold or else make a manual response, when subjects make a saccadic response to an exogenous or endogenous S1, there is no evidence of a visuo-motor inhibition. Instead, saccadic responses show the singular ability to generate a manual motor response inhibition that does not depend on having to process a peripheral target.

MANUAL RESPONSES AS A MEASURE OF IOR: SUMMARY

The results of Experiments 1-3 are summarized in Table 11, along with relevant literature. Table 12 superimposes the current findings on a summary table of predictions that were derived from existing accounts of IOR. As seen, the conjunctive predictions (i.e., those which derive from the

EXPERIMENTS 1-3 VERSUS THE LITERATURE: SUMMARY OF IOR WITH MANUAL RT AS THE DEPENDENT MEASURE, AS A FUNCTION OF S1, S2, AND S1-S2 RESPONSE COMBINATIONS

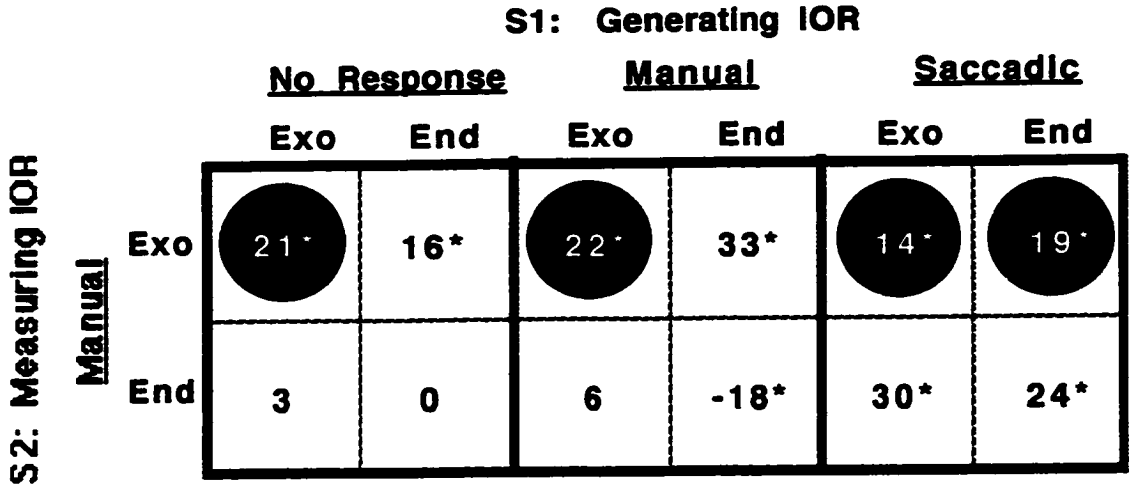

Table 11. Magnitude of IOR in Experiments 1-3. Significant effects are indicated with "". Positive values reflect IOR, negative values reflect facilitation. For comparison, cells that have been tested in the literature are indicated with circles (the black colouring indicates that IOR was observed).*

EXPERIMENTS 1-3: COMPARISON OF THE PRESENT FINDINGS WITH PREDICTIONS MADE FROM THE LITERATURE

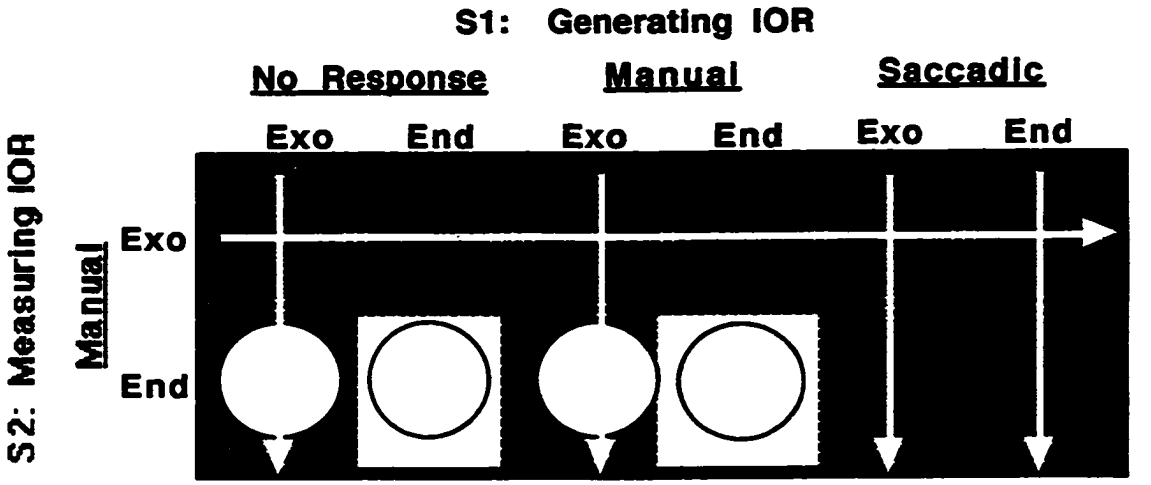

Table 12. Predictions made on the basis of the existing literature are shown with hatched bars and arrows. The present findings for Experiments 1-3 are summarized with circles. White circles indicate the absence of significant IOR; black circles indicate significant IOR.

view that conditions must favour both the generation and the measurement of IOR in order to observe inhibition) were violated by the present data patterns.

Of course, the present experiments were designed to assess the possibility that the characterization of IOR as a "motor response bias that is generated by oculomotor programming" is fundamentally correct but that it is incomplete with respect to the conditions that generate and measure the effect. The literature converges on the conclusion that IOR is a motor response bias when measured by manual RT. As noted, however, it is uncertain whether this bias depends on having to make a response to peripheral visual information. Indeed, predictions outlined for the measurement of IOR indicated only that manual responses to exogenous S2s may show the effect.

Because there is no published evidence to the contrary, predictions for the measurement of IOR could reasonably be extended to encompass the view that IOR is a general motor response bias that does not require that the response be made to peripheral visual stimulation (i.e., that it is motor rather than visuo-motor). As should be obvious, however, this extension would not rescue the "IOR measures motor inhibition" hypothesis with respect to manual RT measured in the current Experiments 1-3 - a conjunctive view would require that IOR be observed for at least some cells in the No Response-Manual and Manual-Manual conditions when S2 is endogenous. This can be seen in Table 12.

Turning from the issue of what is measured by IOR, now consider the corresponding issue of "IOR is generated by oculomotor activation." On the basis of the existing literature, this hypothesis predicts that IOR would be generated automatically by exogenous S1s (i.e., via automatic oculomotor

activation) and also by saccades made to endogenous S1s. However, when introducing the current experiments, the possibility was raised that oculomotor activation may not be specific to exogenous signals and that unpredictive endogenous signals may likewise be capable of generating IOR - even in the absence of an explicit requirement to execute a saccade.

Unfortunately, expanding the predictions to include the ability of endogenous signals to generate oculomotor programming is not able to rescue the "IOR is generated by oculomotor programming" hypothesis with respect to manual RT. If the conjunctive view includes only the prediction that IOR is measured by an exogenous S2, then it is contradicted by the finding of significant IOR for the endogenous-endogenous cell of the Saccade-Manual condition. If, on the other hand, the predictions for the measurement of IOR are expanded to include the possibility that IOR may be measured by any S2, then the conjunctive view is contradicted by the failure to find IOR for the endogenous-endogenous cells of the No Response-Manual and Manual-Manual combinations.

Clearly, the characterization of IOR that has been derived from the existing literature is unable to account for the pattern of results obtained across Experiments 1-3. Moreover, expanding predictions to encompass hitherto overlooked stimulus combinations does not alleviate the difficulty. The view that IOR is a "motor response bias that is generated by oculomotor programming" cannot be applied carte blanche across combinations of S1/S2 response - stimulus conditions pertaining to S1/S2 are not sufficient to predict when IOR will occur and must, instead, be considered in conjunction with attendant responses.

Knowing that IOR cannot be considered independently of either stimulus *or* response combinations, however, does not address the question

of what is measured by IOR and how that inhibition is generated, nor does it account for consistencies across Experiments. Most notably, withholding or else making a manual response to S1 appears to generate a similar inhibitory process: Visuo-motor inhibition of manual RT. This pattern contrasts with that which is generated by a saccadic response to S1. In this case, a general motor inhibition ensues, whereby manual RT is slowed whether or not the response is directed towards a peripheral onset signal. Possible mechanisms supporting visuo-motor inhibition (in the case of No Response-Manual and Manual-Manual response combinations) and supporting motor inhibition (in the case of the Saccadic-Manual combination) will be considered in the General Discussion.

SACCADIC RESPONSES AS A MEASURE OF IOR

EXPERIMENT 4: NO RESPONSE-SACCADIC

In Experiment 4, subjects were required to withhold a response to an exogenous or endogenous S1 and to make a saccade to the location indicated by an exogenous or endogenous S2. As seen in Table 2, the only two cells of this design that have been studied in the literature are the exogenous-exogenous and endogenous-endogenous.

As may be recalled, the exogenous-exogenous and exogenous-endogenous cells were compared directly in Abrams and Dobkin's (1995) study of the visuo-motor and motor processes inhibited by IOR. They presented subjects with a peripheral luminance cue that was followed by either a central endogenous command arrow (see also Rafal et al., 1994) or else by a peripheral exogenous onset target (see also Abrams & Dobkin, 1994; Maylor, 1985; Rafal et al., 1994; Reuter-Lorenz et al., 1996). Both the

endogenous and the exogenous commands revealed IOR; however, the magnitude of the effect differed, with greater inhibition demonstrated by saccadic RTs made in response to an exogenous versus an endogenous command. It was this difference that led Abrams and Dobkin (1995) to postulate that IOR for exogenous targets might represent inhibition both for visuo-motor processing/integration *and* for motor responses directed to the target location.

The present Experiment 4 will attempt to replicate Abrams and Dobkin's (1995) observation of IOR for exogenous and endogenous saccades that follow an exogenous S1; it will also assess whether there is a difference in the magnitude of any observed effects. Furthermore, this experiment will determine whether an endogenous command that requires no response is also able to generate IOR for exogenous and endogenous saccades.

Experiment 4: Results

Errors

As seen in Table 7, the greatest loss of data in any one cell due to one type of error was just over 6%. Collapsed over type of error (directional errors, misses, anticipations), there was a main effect of S2 ($(F(1,17)=6.49$, $MSe=6.70$, $p<.02$), with more errors made to an endogenous ($M=3.0\%$) versus an exogenous ($M=1.9\%$) S2. The main effects for S1 ($F<1$) and for relative locations signalled by S1 and S2 ($F(1,17)=1.32$, $MSe=4.49$, $p<.27$) were not significant. Likewise, none of the interactions were significant: Between S1 and S2 ($F<1$); between S1 and the relative locations signalled by S1 and S2 ($F(1,17)=2.70$, $MSe=1.43$, $p<.12$); between S2 and the relative locations signalled by S1 and S2 ($F(1,17)=1.43$, $MSe=2.11$, $p<.25$); or, between S1, S2, and the relative locations signalled by S1 and S2 ($F<1$).

RTs to S2

Saccadic RTs for error-free trials are shown in Figure 14, as a function of S1 (exogenous, endogenous), S2 (exogenous, endogenous), and relative locations signalled by S1 and S2 (different, same). Analysis of these RT data revealed no difference in the speed of responding to S2 as a function of whether S1 was an exogenous (M=205 msec) or an endogenous (M=208 msec) signal ($F(1,17)=1.42$, $MSe=358.50$, $p<.25$). There was an effect of S2 ($F(1,17)=59.81$, $MSe=1384.08$, $p<.00$), with overall faster saccadic RTs to S2 when it was exogenous (M=183 msec) versus endogenous (M=231 msec). The main effect of relative location signalled by S1 and S2 was not significant ($F(1,17)=2.49$, $MSe=620.51$, $p<.13$).

None of the two-way interactions of S1 and S2 ($F(1,17)=2.87$, $MSe=158.21$, $p<.11$), S1 and relative locations signalled by S1 and S2 ($F(1,17)=3.605$, $MSe=283.53$, $p<.07$), or of S2 and relative locations signalled by S1 and S2 were significant ($F(1,17)=2.268$, $MSe=161.95$, $p<.15$). The three-way interaction of S1, S2, and relative locations signalled by S1 and S2 was also not significant ($F(1,17)=1.56$, $MSe=169.09$, $p<.23$). Where the number of subjects showing an effect in the same direction as the overall mean is indicated in parentheses, planned comparisons on this three-way interaction revealed a significant 12 msec IOR effect (n=13) for the exogenous-exogenous combination ($F(1,17)=8.15$, $MSe=169.09$, $p<.01$); a non-significant 7 msec IOR effect (n=12) for the endogenous-exogenous combination ($F(1,17)=2.70$, $MSe=169.09$, $p<.12$); a significant 11 msec IOR effect (n=11) for the exogenous-endogenous combination ($F(1,17)=6.01$, $MSe=169.09$, $p<.02$); and, a non-significant 5 msec facilitatory effect (n=10) for the endogenous-endogenous combination ($F(1,17)=1.16$, $MSe=196.09$, $p<.30$). These results are summarized in Table 13.

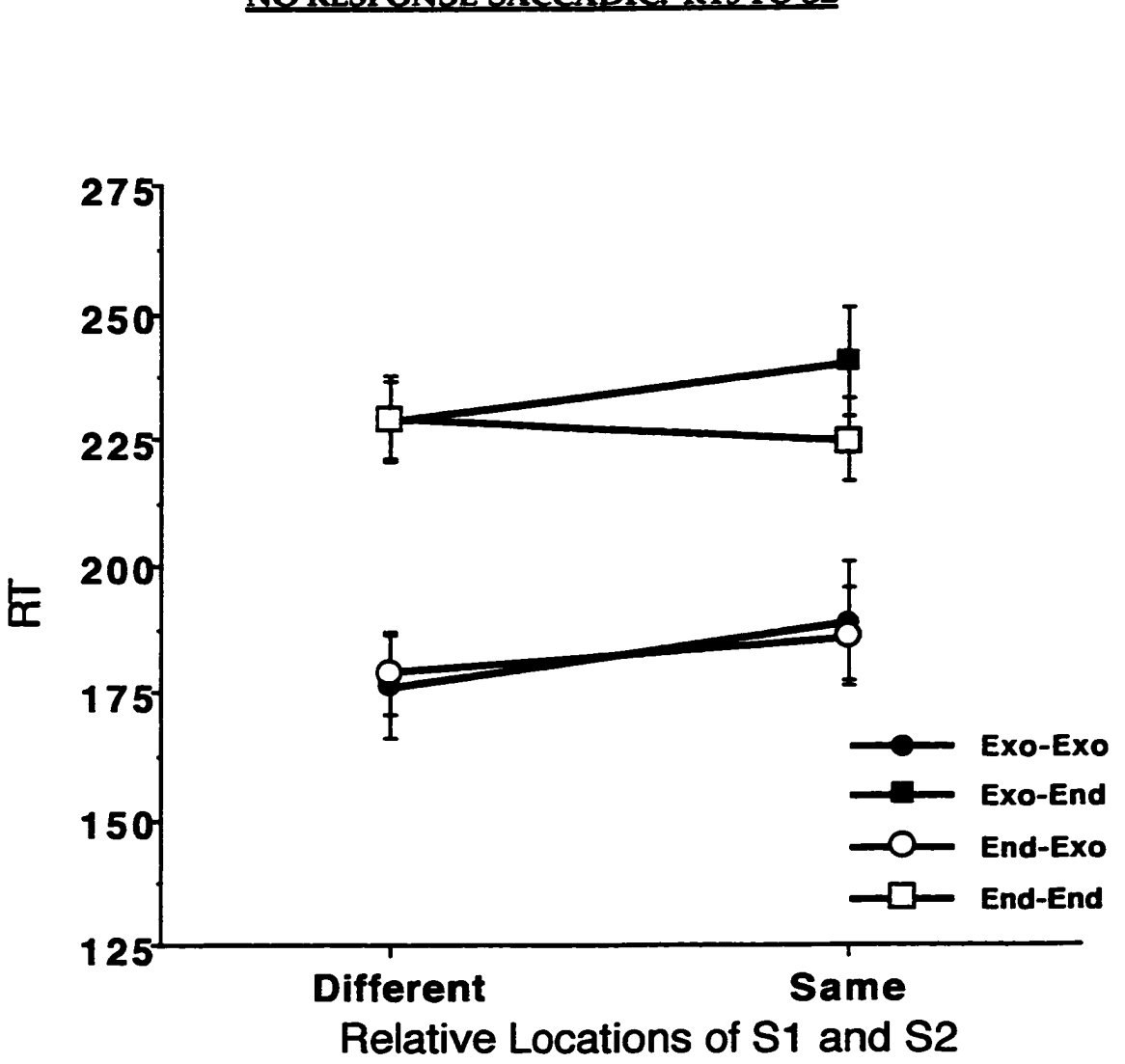

Figure 14. Experiment 4: Saccadic RT to S2, as a function of the relation between S1 and S2 locations (different, same) and signals (exo: exogenous, end: endogenous).

EXPERIMENT 4 VERSUS THE LITERATURE: SUMMARY OF IOR IN THE NO RESPONSE-SACCADIC COMBINATION, AS A FUNCTION OF S1 AND S2

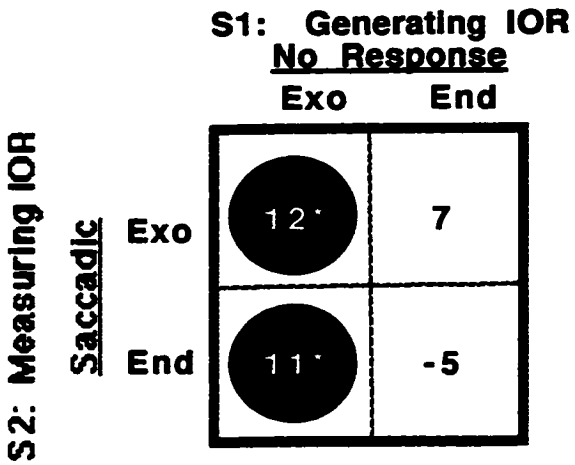

Table 13. Magnitude of IOR in Experiment 4. Significant effects are indicated with "". Positive values reflect IOR, negative values reflect facilitation. For comparison, cells that have been tested in the literature are indicated with circles (the black colouring indicates that IOR was observed).*

IOR as a Function of Block Number

Scatterplots of the block number in which subjects performed Experiment 4 and the magnitude of the obtained IOR effects are shown in Figure 15. Revealing no systematic effects of practice on the magnitude of IOR, none of the correlations were significant. The correlations were 0.07 for the exogenous-exogenous combination ($F<1$); -0.26 for the endogenous-exogenous combination ($F(1,17)=1.11$, $MSe=818.67$, $p<.31$); -0.16 for the exogenous-endogenous combination ($F<1$); and, -0.19 for the endogenous-endogenous combination ($F<1$).

Experiment 4: Summary

As summarized in Table 13, IOR was obtained in the No Response-Saccadic condition for both the exogenous-exogenous and exogenous-endogenous combinations. This replicates Abrams and Dobkin (1995) most generally. However, the present experiment failed to replicate their finding of greater magnitude (by 15 msec) IOR when S2 was exogenous versus endogenous; in the present experiment, there was no difference (1 msec) between the magnitude of IOR obtained in the exogenous-exogenous and exogenous-endogenous cells. The present experiment also demonstrates that IOR is not obtained when an endogenous S1 is employed. This is true whether S2 is exogenous or endogenous.

The pattern of results in this experiment are somewhat surprising in light of Experiment 1. The only difference between Experiments 1 and 4 is the dependent variable - manual versus saccadic RT. In the case of the No Response-Manual response combination, exogenous and endogenous S1s were capable of generating IOR that was reflected in RTs to an exogenous S2. This was taken as evidence that the S1 signals established a visuo-motor inhibition. In the case of the No Response-Saccadic response combination,

NO RESPONSE-SACCADIC: CORRELATIONS OF IOR WITH EXPERIMENTAL BLOCK NUMBER

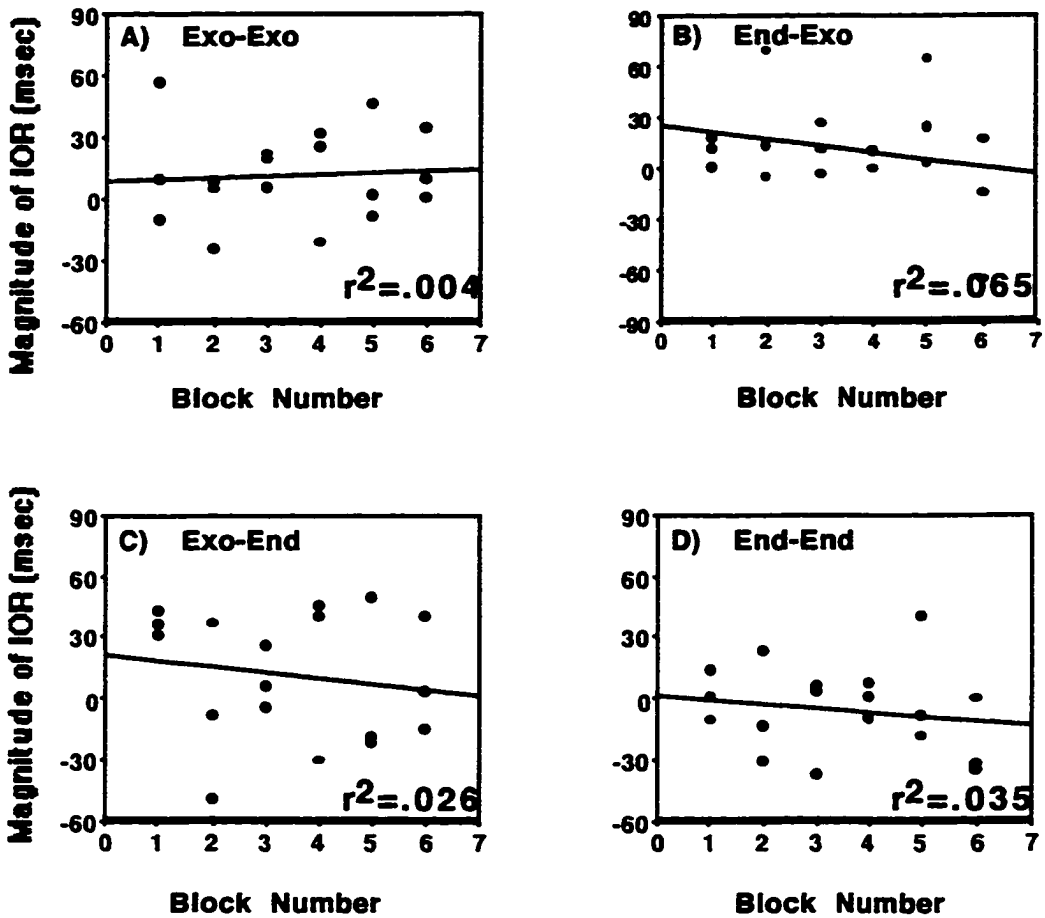

Figure 15. Experiment 4: Scatterplots of the magnitude of IOR versus experimental block number for the exogenous-exogenous (A), endogenous-exogenous (B), exogenous-endogenous (C), and endogenous-endogenous (D) combinations of S1-S2.

however, only exogenous S1s were capable of generating IOR but this IOR was reflected in the RTs to both exogenous and endogenous S2s.

Whereas the critical factor in interpreting the data of Experiment 1 rested in the distinction between the relative ability of exogenous and endogenous signals to *measure* IOR, the critical factor in interpreting the data of Experiment 4 rests in the distinction between the relative ability of exogenous and endogenous signals to *generate* IOR. When preceded by an exogenous S1, equivalent magnitude IOR is obtained for both exogenous and endogenous S2s. This is consistent with the view that IOR measured by saccadic RT reflects a general motor inhibition and that there is not the operation of a distinct visuo-motor inhibition specific to exogenous S2s. This said, the critical determinant of whether IOR is observed or not is the nature of S1 - the motor inhibition depends on subjects withholding a response to an exogenous rather than an endogenous first signal.

Such motor inhibition resulting from withholding a response to an exogenous S1 is consistent with the view that an unpredictive exogenous signal activates the oculomotor system and thereby generates a motor response inhibition. Importantly, if this is the reason why exogenous signals are capable of generating IOR, then it follows that the failure to observe IOR following an endogenous S1 in Experiment 4 reflects the failure of these signals to engage oculomotor programming. This is a critical point because it impinges on explanations that may be posited to account for the ability of the same endogenous S1s to generate visuo-motor inhibition for manual responses. This issue will be reconsidered in the General Discussion.

To recapitulate the present results, Experiment 4 reveals that IOR occurs when S1 is exogenous but not when S1 is endogenous. This appears to reflect a motor inhibition for responses made to S2; the fact that this motor

inhibition occurs only following exogenous S1s suggests that oculomotor programming may be critical to its genesis. If it is the case that motor inhibition in Experiment 4 is established by the automatic activation of an oculomotor program by an exogenous S1, then it follows that the explicit activation of a motor program in the Manual-Saccadic response combination of Experiment 5 may be capable of generating IOR whether the S1 signal is exogenous or endogenous. Of course, this depends on the assumption that oculomotor programming and manual motor programming/execution share neural and/or functional substrates for generating IOR.

EXPERIMENT 5: MANUAL-SACCADIC

In Experiment 5, subjects made a manual response to an exogenous or endogenous S1 and a saccadic response to an exogenous or endogenous S2. Curiously, this response combination does not appear to have been explored in the literature. Nevertheless, the conjunctive view demands that IOR be obtained in the exogenous-exogenous and exogenous-endogenous but not in the endogenous-exogenous and endogenous-endogenous cells of this response combination. The present experiment represents a first attempt to determine whether IOR is obtained for this response combination and, if so, whether it is obtained for the predicted combinations of S1 and S2.

Experiment 5: Results

Errors

As seen in Table 7, the largest data lost to a single type of error in any cell was just over 11%. Collapsed across types of error (directional errors, misses, anticipations), an analysis revealed no significant main effect for S1 ($F<1$). There was, however, a significant main effect for S2 ($F(1,17)=6.39$, $MSe=13.77$, $p<.02$). This main effect stemmed from overall greater errors to

an endogenous (M=4.6%) versus an exogenous (M=3.0%) S2. The main effect of relative locations signalled by S1 and S2 was not significant ($F(1,17)$=1.13, MSe=1.13, p<.30).

The two-way interactions of S1 and S2 (F<1) and of S1 and relative locations signalled by S1 and S2 ($F(1,17)$=1.02, MSe=1.59, p<.33) were not significant. However, the two-way interaction of S2 and relative locations signalled by S1 and S2 was significant ($F(1,17)$=5.99, MSe=4.94, p<.03). This interaction reflects a greater number of errors made when an endogenous S2 signalled the same (M=5.2%) versus a different (M=3.9%) location as S1, and fewer errors made when an exogenous S2 signalled the same (M=2.7%) versus a different (M=3.3%) location as S1. The three-way interaction of S1, S2, and relative locations signalled by S1 and S2 was not significant (F<1).

RTs to S1

Analysis of manual RTs to S1 revealed only a significant main effect of S1 ($F(1,17)$=16.82, MSe=929.87, p<.00), with overall faster responding to exogenous (M=266 msec) than to endogenous (M=287 msec) signals. No other main effects were significant (Fs<1), nor were any of the interactions of: S1 and S2 ($F(1,17)$=3.40, MSe=49.78, p<.08); S1 and relative locations signalled by S1 and S2 (F<1); S2 and relative locations signalled by S1 and S2 ($F(1,17)$=1.38, MSe=166.27, p<.26); or of S1, S2, and relative locations signalled by S1 and S2 ($F(1,17)$=3.05, MSe=110.84, p<.10).

RTs to S2

Saccadic RTs on trials for which no errors occurred are shown in Figure 16, according to S1 (exogenous, endogenous), S2 (exogenous, endogenous), and the relative locations signalled by S1 and S2 (different, same). Analysis of these data revealed no difference in S2 saccadic RT as a function of the S1 to which the preceding manual response was made ($F(1,17)$=2.00, MSe=538.62,

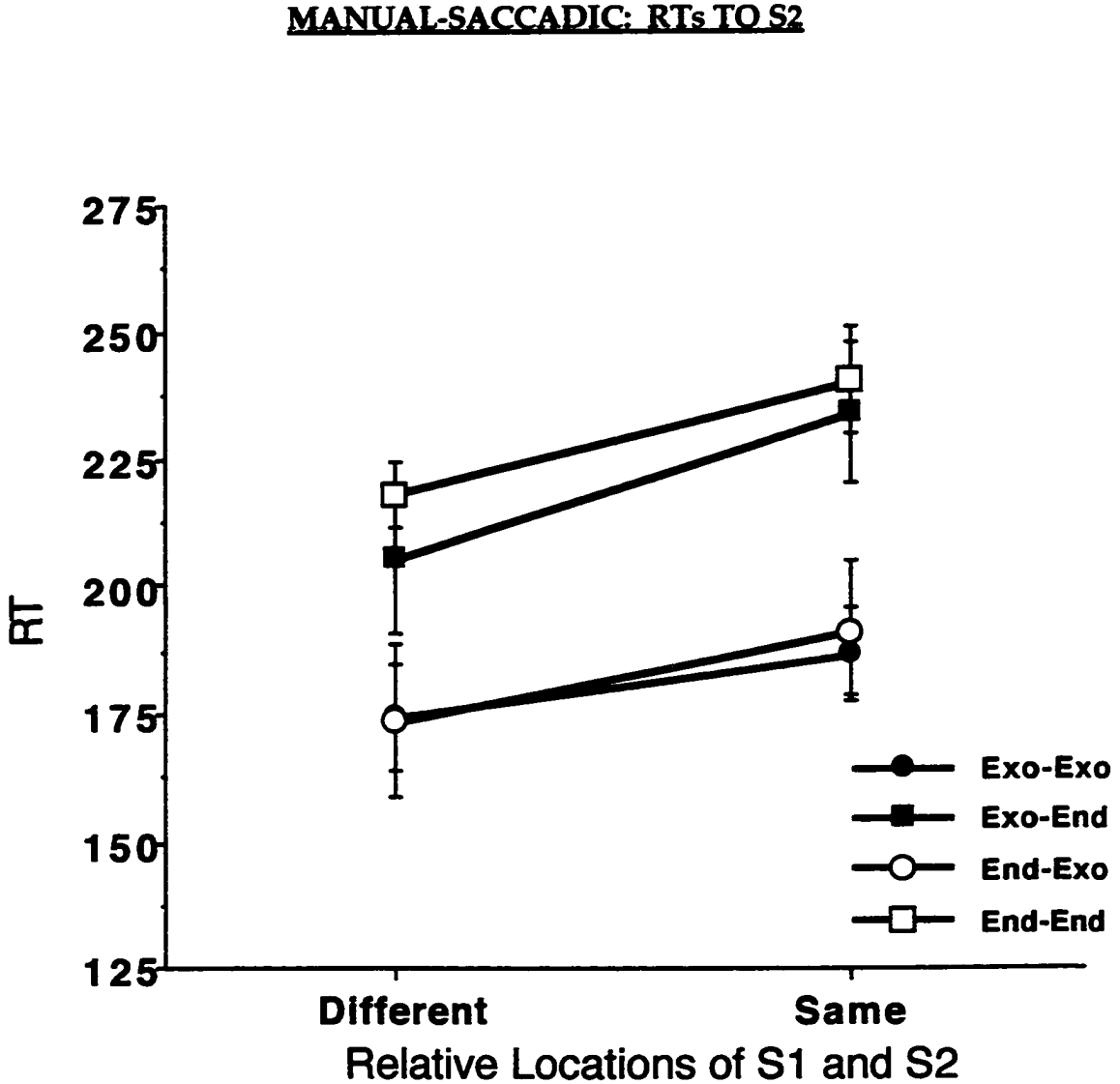

Figure 16. Experiment 5: Saccadic RT to S2, as a function of the relation between S1 and S2 locations (different, same) and signals (exo: exogenous, end: endogenous).

$p<.18$). There was, however, a significant main effect of S2 ($F(1,17)=18.96$, $MSe=3509.27$, $p<.00$): RTs were faster to an exogenous ($M=182$ msec) than to an endogenous ($M=225$ msec) S2. There was also a main effect for relative locations of S1 and S2 ($F(1,17)=25.70$, $MSe=571.95$, $p<.00$). This main effect reflects overall slower RTs to S2 when it signalled the same ($M=213$ msec) versus a different ($M=193$ msec) location relative to S1.

The two-way interactions of S1 and S2 ($F(1,17)=1.05$, $MSe=504.78$, $p<.32$), of S1 and relative locations signalled by S1 and S2 ($F<1$), and of S2 and the relative locations signalled by S1 and S2 ($F(1,17)=2.49$, $MSe=396.35$, $p<.13$) were not significant. The three-way interaction of S1, S2, and relative locations signalled by S1 and S2 was also not significant ($F<1$). However, planned comparisons on this interaction revealed significant IOR effects for all combinations of S1 and S2. Where the number of subjects showing the effect in the same direction as the overall cell mean is shown parenthetically, there was a significant 13 msec IOR effect (n=13) for the exogenous-exogenous combination ($F(1,17)=4.28$, $MSe=342.52$, $p<.05$); a significant 17 msec IOR effect (n=15) for the endogenous-exogenous combination ($F(1,17)=7.77$, $MSe=342.52$, $p<.01$); a significant 29 msec IOR effect (n=16) for the exogenous-endogenous combination ($F(1,17)=21.66$, $MSe=242.52$, $p<.00$); and, a significant 22 msec IOR effect (n=14) for the endogenous-endogenous combination ($F(1,17)=12.91$, $MSe=342.52$, $p<.00$). These results are summarized in Table 14.

RTs to S1 versus IOR

Correlations were used to determine whether the magnitude of IOR as measured by saccadic RTs was related to the speed of making a manual response to S1. Scatterplots of these data are shown in Figure 17. In no case was there a significant correlation between the magnitude of IOR measured by the saccadic response to S2 and the manual RT made to S1. The correlation

EXPERIMENT 5 VERSUS THE LITERATURE: SUMMARY OF IOR IN THE MANUAL-SACCADIC COMBINATION, AS A FUNCTION OF S1 AND S2

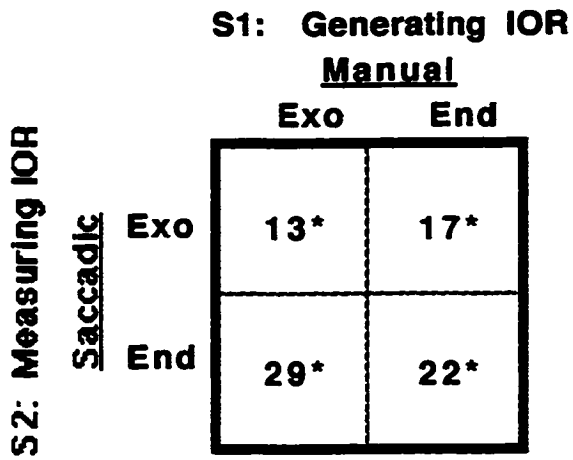

Table 14. Magnitude of IOR in Experiment 5. Significant effects are indicated with "". Positive values reflect IOR, negative values reflect facilitation. As noted in the text, none of these cells have been tested in the literature.*

MANUAL-SACCADIC: CORRELATIONS OF IOR WITH S1 RT

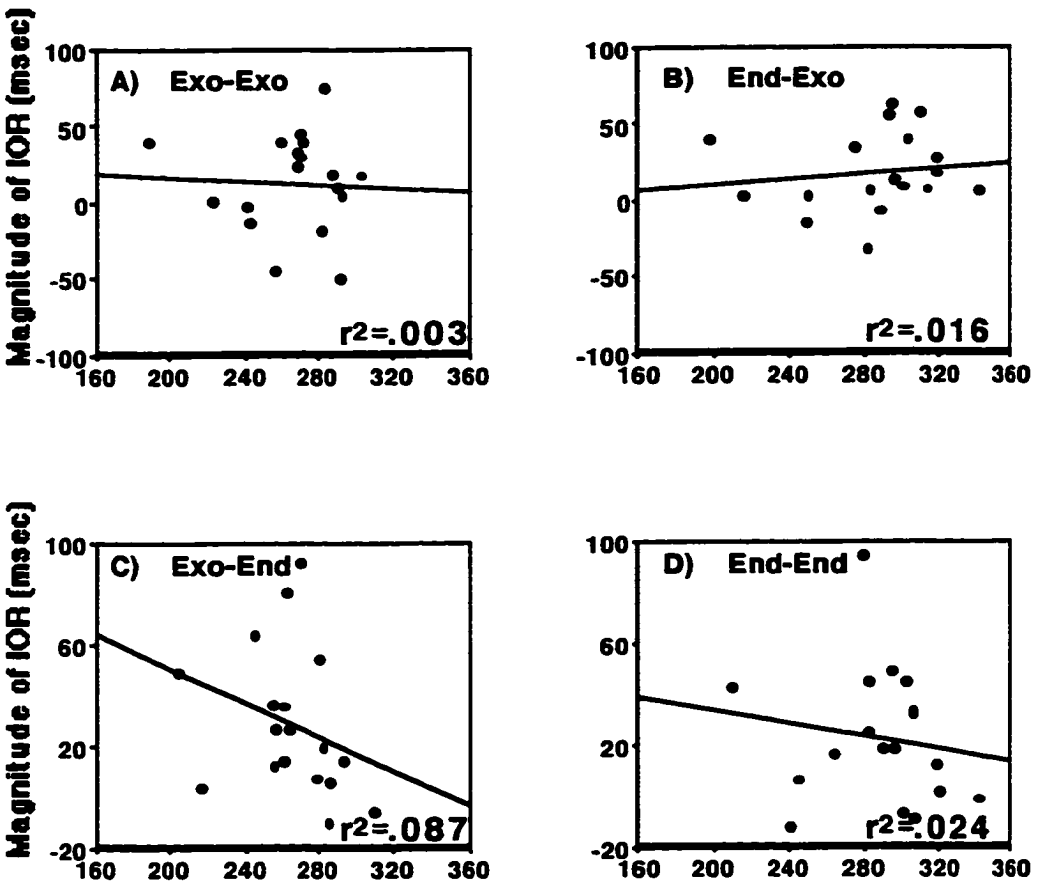

Figure 17. Experiment 5: Scatterplots of S1 latencies versus the magnitude of IOR for the exogenous-exogenous (A), endogenous-exogenous (B), exogenous-endogenous (C), and endogenous-endogenous (D) combinations of S1-S2.

was -0.05 ($F<1$) for the exogenous-exogenous combination; 0.13 for the endogenous-exogenous combination ($F<1$); -0.29 for the exogenous-endogenous combination ($F(1,17)=1.52$, $MSe=815.21$, $p<.24$); and, -0.16 for the endogenous-endogenous combination ($F<1$).

IOR as a Function of Block Number

Scatterplots of the block number in which subjects performed Experiment 5 versus the magnitude of the obtained IOR effects are shown in Figure 18. Correlational analyses of these data revealed no significant relation between the level of practice and the magnitude of IOR for any of the combinations of S1-S2. The correlations were: -0.20 for the exogenous-exogenous combination ($F<1$); 0.16 for the endogenous-exogenous combination ($F<1$); -0.27 for the exogenous-endogenous combination ($F(1,17)=1.23$, $MSe=828.82$, $p<.28$); and, -0.35 for the endogenous-endogenous combination ($F(1,17)=2.28$, $MSe=650.42$, $p<.15$).

Experiment 5: Summary

Experiment 5 is the first to explore the Manual-Saccadic combination of responses. As seen in Table 14, the results demonstrated IOR for all four cells. The observation that significant IOR occurred for exogenous and endogenous S2s as a function of both exogenous and endogenous S1s converges with the conclusions of Experiment 4: Saccadic RTs appear to measure motor inhibition that is generated by the activation of motor programming.

As had been the case for the converse response pairing of Manual-Saccadic in Experiment 3, there was a larger magnitude IOR effect for endogenous than for exogenous S2s. This was revealed in the significant interaction of S2 and relative locations signalled by S1 and S2. Because of the significant main effect of S2 which revealed overall slower responding to endogenous S2s versus exogenous S2s, it is useful to also assess whether the

MANUAL-SACCADIC: CORRELATIONS OF IOR WITH EXPERIMENTAL BLOCK NUMBER

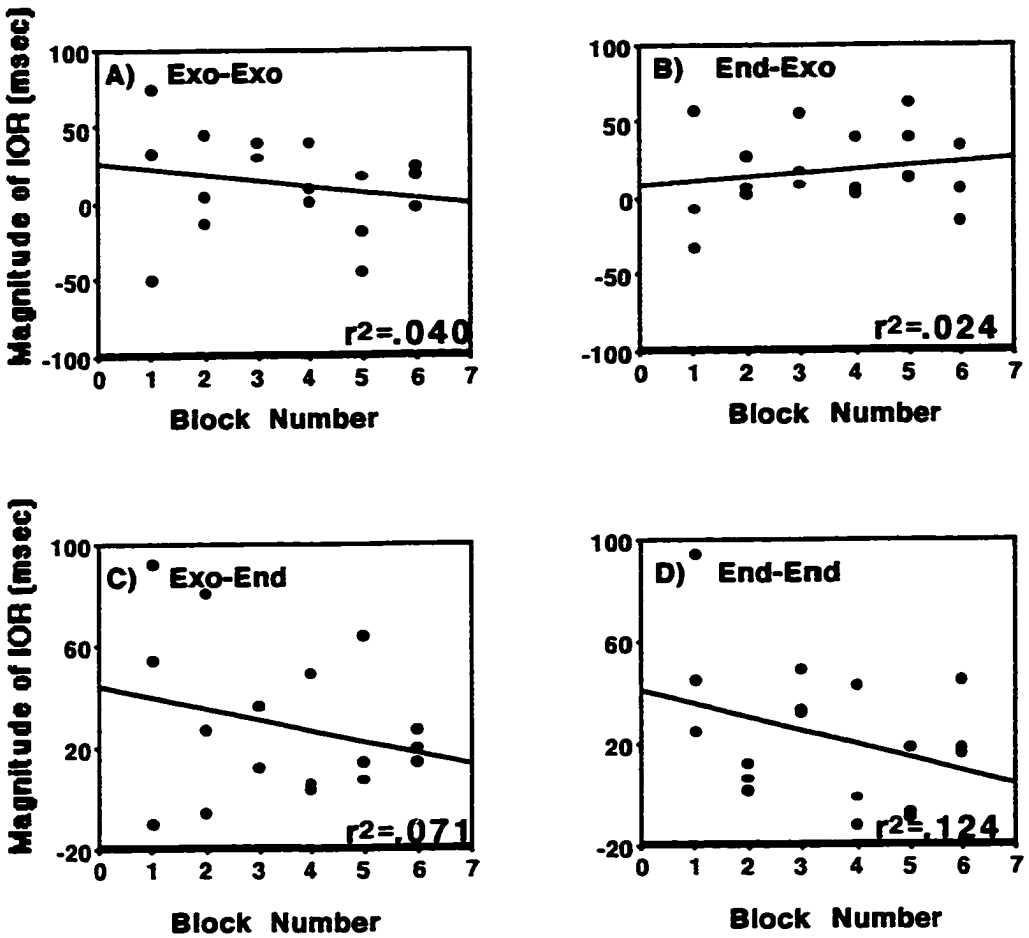

Figure 18. Experiment 5: Scatterplots of the magnitude of IOR versus experimental block number for the exogenous-exogenous (A), endogenous-exogenous (B), exogenous-endogenous (C), and endogenous-endogenous (D) combinations of S1-S2.

relative magnitude of IOR differed according to the nature of the S2 signal. If the 15 msec IOR effect for exogenous S2s is examined relative to the mean saccadic RT of 193 msec, the proportion of IOR:RT was 0.08. If the 26 msec IOR effect for endogenous S2s is examined relative to the mean saccadic RT of 213 msec, the proportion of IOR:RT was 0.12. As such, even in relative terms, the magnitude of the IOR effect for endogenous S2s was larger than for exogenous S2s. This counters any suggestion that there was a visuo-motor inhibition specific to exogenous S2s. Instead, it appears that - like Experiment 4 - saccadic RTs in Experiment 5 measure a motor response inhibition that is independent of peripheral visual processing (i.e., it is not visuo-motor).

The fact that the inhibition is greater for endogenous versus exogenous S2s may be due to the nature of the stimuli themselves. As had been suggested for the difference that also occurred in the Saccadic-Manual cell, the peripheral visual information supplied by exogenous S2s may overcome some of the motor inhibition and/or the purely symbolic nature of endogenous S2s may exacerbate the motor inhibition. Regardless of the reason for the difference, the important point is that IOR is not greater for saccadic RTs to exogenous versus endogenous S2s.

Turning now to how the motor inhibition is generated, the fact that IOR occurs following both exogenous and endogenous S1s when an overt (manual) response is required but only following exogenous S1s when no response is required (Experiment 1) concurs with the suggestion that activation of a motor program is responsible. Whether the critical motor activation is tied to the oculomotor system is a point which will be considered in the General Discussion. Before then, however, examination of IOR when the first response is saccadic rather than manual will shed some light on this issue.

EXPERIMENT 6: SACCADIC-SACCADIC

In Experiment 6, subjects made a saccade in response to an exogenous or endogenous S1, returned fixation to centre upon brightening, and made a saccade in response to an exogenous or endogenous S2. This Saccadic-Saccadic response combination is the only one for which all combinations of exogenous/endogenous S1/S2 have been examined in the literature; for all four combinations, IOR has been observed. Although others have used saccade-saccade paradigms to assess IOR (e.g., Tanaka & Shimojo, 1996; Vaughan, 1984), the quintessential study of relevance to Experiment 6 was that performed by Rafal et al. (1994) who examined all four cells.

Recall that Rafal et al. (1994) required subjects to saccade to an exogenous cue or else in the direction indicated by a centrally-presented arrow; latencies to make reflexive pro-saccades, endogenously-generated anti-saccades, and endogenous saccades to centrally-presented arrows served as the dependent measures in different experiments. When subjects made saccades to exogenous S1s, IOR was observed for both pro- and anti-saccades. In contrast, when subjects made saccades to endogenous S1s, IOR was observed for pro-saccades and for saccades made in response to an endogenous arrow command, but not for anti-saccades. It was on the basis of these results that Rafal et al. (1994) suggested that: 1) Exogenous S1s result in a visuo-motor inhibition wherein subjects are slow to respond on the basis of information presented at the same location as signalled by S1; and, 2) endogenous S1s result in a similar visuo-motor inhibition as well as a motor inhibition that biases against responding in the direction of S1. These two inhibitory effects were presumed to counter one another when Rafal et al. (1994) employed anti-saccades as the dependent measure and no IOR was obtained. However, when endogenous arrows at fixation were instead used as the S2 signal, IOR

occurred.

Because the current Experiment 6 uses centrally-presented arrows as the endogenous S2, there is no concern that any visuo-motor and motor inhibitory effects may oppose one another. Indeed, the Saccadic-Saccadic combination of responses in Experiment 6 will attempt to replicate the conditions of Rafal et al. (1994) that most closely resemble the overarching design of the current study. Such a replication would reveal IOR for all combinations of exogenous and endogenous S1 and S2s.

Experiment 6: Results

Errors

As seen in Table 7, the greatest loss of data in any one cell due to a particular type of error was just over 5%. Collapsed across type of error (directional errors, misses, anticipations), an analysis revealed only a significant effect for S2 ($F(1,17)=8.56$, $MSe=5.26$, $p<.01$), with more errors made following an endogenous ($M=2.3\%$) than following an exogenous ($M=1.2\%$) signal. The main effects of S1 ($F(1,17)=2.33$, $MSe=3.32$, $p<.15$) and relative locations signalled by S1 and S2 ($F(1,17)=2.77$, $MSe=7.73$, $p<.11$) were not significant. None of the two- or three-way interactions were significant (all $Fs<1$).

RTs to S1

When saccadic RTs to S1 were examined, the analysis revealed no significant main effects of S1 ($F(1,17)=2.58$, $MSe=1163.79$, $p<.13$), S2 ($F<1$), or relative locations of S1 and S2 ($F<1$). The two-way interaction of S1 and S2 was significant ($F(1,17)=4.25$, $MSe=668.14$, $p<.05$). Recognizing that S2 events are dummy codes with respect to S1 RT, it happened that RTs to exogenous S1s were faster in conditions when an endogenous ($M=203$ msec) versus an exogenous ($M=208$ msec) S2 followed and, that RTs to endogenous S1s were

slower in conditions when an endogenous (M=217 msec) versus an exogenous (M=213 msec) S2 followed.

The interactions of S1 and relative locations signalled by S1 and S2 ($F(1,17)=3.08$, $MSe=113.93$, $p<.10$), of S2 and relative locations signalled by S1 and S2 ($F<1$), and of S1, S2, and relative locations signalled by S1 and S2 ($F<1$) were all not significant.

RTs to S2

Saccadic RTs on trials for which no errors occurred are shown in Figure 19, as a function of S1 (exogenous, endogenous), S2 (exogenous, endogenous), and the relative locations signalled by S1 and S2 (different, same). Analysis of these data revealed a significant effect of S1 ($F(1,17)=8.20$, $MSe=376.56$, $p<.01$). As had been the case for the Saccadic-Manual response pairing of Experiment 3, when saccades were made to an exogenous S1, subsequent responses to S2 were faster (M=199 msec) than when saccades were made to an endogenous S1 (M=208 msec). This main effect of S1 did not interact with any other factors (see below). There was also a significant main effect of S2 ($F(1,17)=139.10$, $MSe=664.70$, $p<.00$), with faster saccades made to an exogenous S2 (M=178 msec) than to an endogenous S2 (M=229 msec); contradicting a speed-accuracy trade-off, the error data showed fewer errors when saccades were made to an exogenous signal than when made to an endogenous signal. Returning to the S2 RT data, the main effect of the relative locations of S1 and S2 was also significant ($F(1,17)=18.41$, $MSe=868.44$, $p<.00$), with overall slower saccades to S2 when S1 and S2 signalled the same (M=214 msec) versus different (M=193 msec) locations.

None of the two-way interactions approached significance (all $Fs<1$). The three-way interaction of S1, S2, and relative locations signalled by S1 and S2 was also not significant ($F<1$). Where the number of subjects showing an

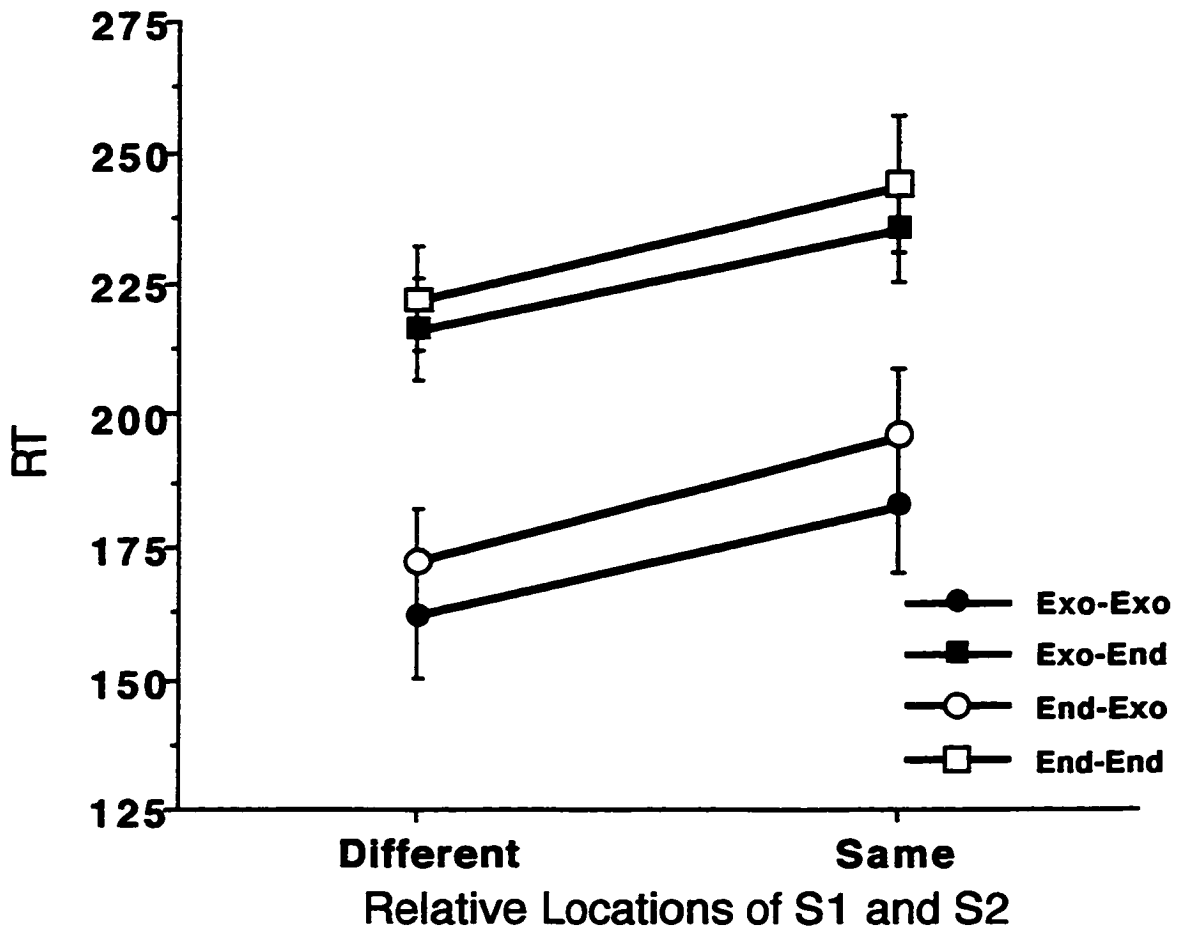

Figure 19. Experiment 6: Saccadic RT to S2, as a function of the relation between S1 and S2 locations (different, same) and signals (exo: exogenous, end: endogenous).

effect in the same direction as the overall cell mean is indicated in parentheses, planned contrasts on the three-way interaction revealed a significant 21 msec IOR effect (n=15) for the exogenous-exogenous combination ($F(1,17)=15.60$, $MSe=245.75$, $p<.00$); a significant 24 msec IOR effect (n=14) for the endogenous-exogenous combination ($F(1,17)=20.32$, $MSe=245.75$, $p<.00$); a significant 19 msec IOR effect (n=15) for the exogenous-endogenous combination ($F(1,17)=13.04$, $MSe=245.75$, $p<.00$); and, a significant 21 msec IOR effect (n=12) for the endogenous-endogenous combination ($F(1,17)=16.51$, $MSe=245.75$, $p<.00$). These results are summarized in Table 15.

RTs to S1 versus IOR

Correlations between the magnitude of the IOR effect as measured by a saccade to S2 and the speed to make a saccadic response to S1 were not significant for any combination of S1 and S2. Scatterplots of these data are shown in Figure 20. The correlation was 0.23 ($F<1$) for the exogenous-exogenous combination; 0.31 ($F(1,17)=1.73$, $MSe=552.67$, $p<.21$) for the endogenous-exogenous combination; 0.15 ($F<1$) for the exogenous-endogenous combination; and, 0.25 ($F(1,17)=1.07$, $MSe=1466.88$, $p<.32$) for the endogenous-endogenous combination.

IOR as a Function of Block Number

Scatterplots of the block number in which subjects performed Experiment 6 versus the magnitude of the obtained IOR effects for each combination of S1-S2 are shown in Figure 21. The -0.45 correlation between block number and the magnitude of IOR obtained for the endogenous-exogenous combination approached, but did not reach, significance ($F(1,17)=3.95$, $MSe=491.24$, $p<.06$). None of the correlations for the other combinations approached significance: For the exogenous-exogenous combination, the correlation was -0.18 ($F<1$); for the exogenous-endogenous

EXPERIMENT 6 VERSUS THE LITERATURE: SUMMARY OF IOR IN THE SACCADIC-SACCADIC COMBINATION, AS A FUNCTION OF S1 AND S2

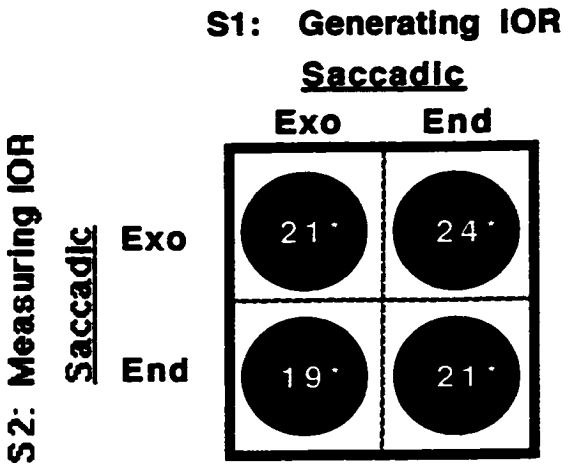

Table 15. Magnitude of IOR in Experiment 6. Significant effects are indicated with "". Positive values reflect IOR, negative values reflect facilitation. For comparison, cells that have been tested in the literature are indicated with circles (the black colouring indicates that IOR was observed).*

SACCADIC-SACCADIC: CORRELATIONS OF IOR WITH S1 RT

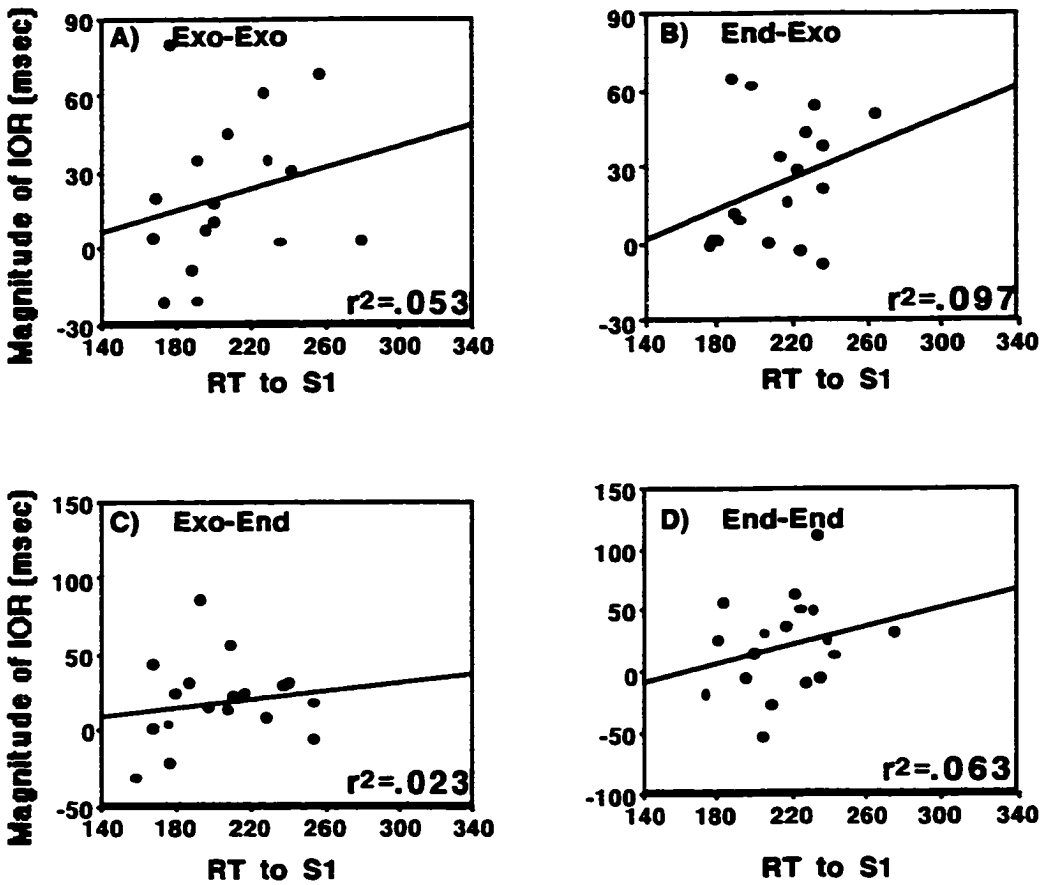

Figure 20. Experiment 6: Scatterplots of S1 latencies versus the magnitude of IOR for the exogenous-exogenous (A), endogenous-exogenous (B), exogenous-endogenous (C), and endogenous-endogenous (D) combinations of S1-S2.

SACCADIC-SACCADIC: CORRELATIONS OF IOR WITH EXPERIMENTAL BLOCK NUMBER

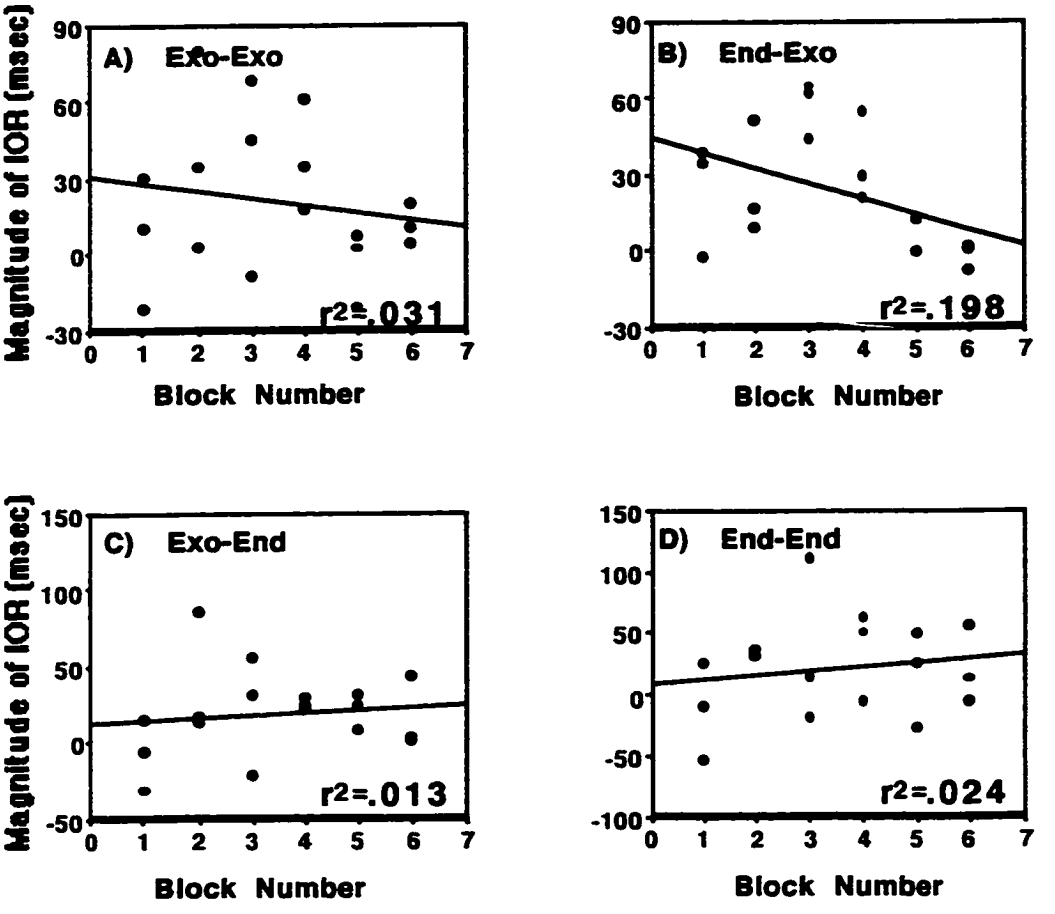

Figure 21. Experiment 6: Scatterplots of the magnitude of IOR versus experimental block number for the exogenous-exogenous (A), endogenous-exogenous (B), exogenous-endogenous (C), and endogenous-endogenous (D) combinations of S1-S2.

combination, the correlation was 0.11 ($F<1$); and for the endogenous-endogenous combination, the correlation was 0.16 ($F<1$).

Experiment 6: Summary

As seen in Table 15, the current Experiment 6 replicated the findings of Rafal et al. (1994). Specifically, IOR was observed for all combinations of exogenous and endogenous S1 and S2s.

As with the Manual-Saccadic response combination of Experiment 5, the pattern of data in the Saccadic-Saccadic response combination of Experiment 6 appears to reflect the operation of a motor inhibition that is generated by the activation of a motor program. The occurrence of IOR for both exogenous and endogenous S2s, and the failure to find any difference in the magnitude of the effects, supports the suggestion that a motor inhibition is being measured by the saccadic RTs. Unlike when the responses to S1 and S2 were in different modalities (Saccadic-Manual, Manual-Saccadic), there was no difference in the magnitude of the IOR effects obtained following exogenous versus endogenous S2s.

To reiterate the findings of Experiment 6: For the Saccadic-Saccadic response combination, IOR is generated by the implementation of a saccadic program to an exogenous or endogenous S1 and this inhibition appears to represent a motor bias against making saccadic responses to exogenous or endogenous S2s.

SACCADIC RESPONSES AS A MEASURE OF IOR: SUMMARY

The results of Experiments 4-6 are summarized in Table 16, along with relevant literature. Table 17 superimposes the current findings on a summary table of predictions that were derived from existing accounts of IOR. Predictions based on a conjunction of methods used to generate and

EXPERIMENTS 4-6 VERSUS THE LITERATURE: SUMMARY OF IOR WITH SACCADIC RT AS THE DEPENDENT MEASURE, AS A FUNCTION OF S1, S2, AND S1-S2 RESPONSE COMBINATIONS

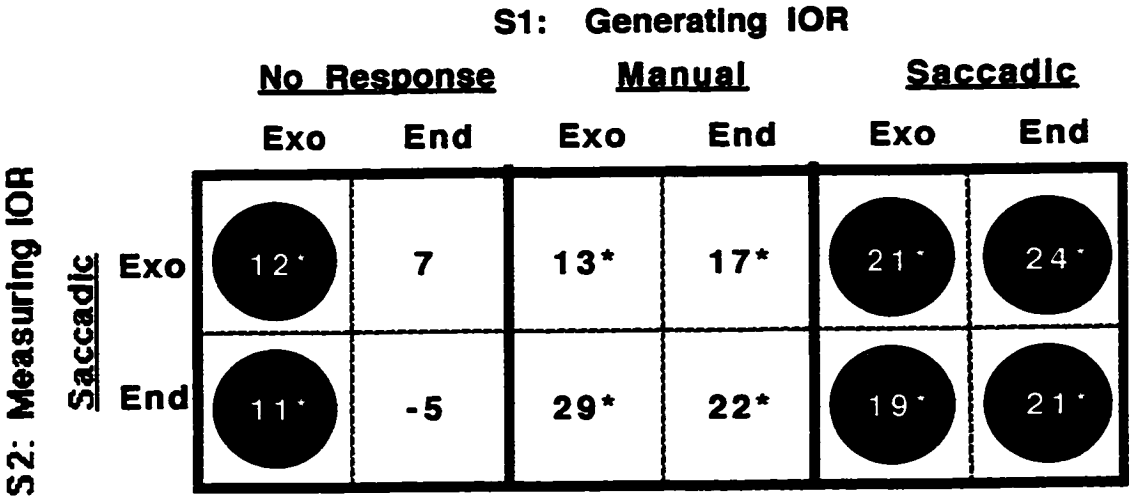

Table 16. Magnitude of IOR in Experiments 4-6. Significant effects are indicated with "". Positive values reflect IOR, negative values reflect facilitation. For comparison, cells that have been tested in the literature are indicated with circles (the black colouring indicates that IOR was observed).*

EXPERIMENTS 4-6: COMPARISON OF THE PRESENT FINDINGS WITH PREDICTIONS MADE FROM THE LITERATURE

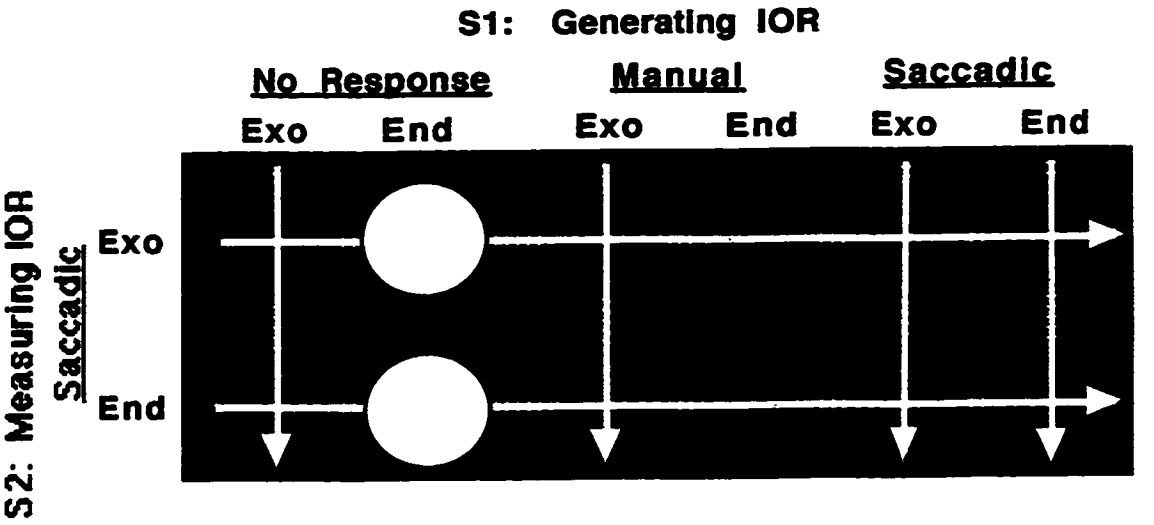

Table 17. Predictions made on the basis of the existing literature are shown with hatched bars and arrows. The present findings for Experiments 4-6 are summarized with circles. White circles indicate the absence of significant IOR; black circles indicate significant IOR.

measure IOR are shown. As was the case with manual RTs as the dependent measure, the results of Experiments 4-6 do not support the predictions that are derived from the literature.

The literature converges on the view that saccadic RTs measure a motor response bias that does not require that the saccade be made to peripheral visual stimulation. Thus, it was predicted that saccadic RTs to both exogenous and endogenous S2s would be sensitive to any inhibitory effects that are generated by S1. Short of contradicting existing literature, it is not possible to withdraw this prediction or to make counter-claims based on the present results. Indeed, the present results suggest only that this motor response bias is sensitive to the methods by which IOR is generated.

With respect to the generation of IOR, the view that IOR results from oculomotor programming led to the prediction that IOR would be generated by exogenous S1s regardless of the response that was required and for endogenous S1s when a saccade was required. As was discussed with reference to Experiments 1-3, one of the goals of the present investigation was to determine whether endogenous cues could likewise generate IOR - perhaps via priority tags that elicit oculomotor programming. However, even if the predictions regarding IOR generation are extended to include this possibility, the present results still cannot be accounted for by the conjunctive view that was derived from the literature. In particular, if endogenous S1s were, in fact, capable of eliciting oculomotor programming, then IOR would be expected in both the endogenous-exogenous and endogenous-endogenous cells of all three response combinations. Contrary to this prediction, there was no IOR in these cells for the No Response-Saccadic combination.

As was the case with manual RT as the dependent measure, the results of Experiments 4-6 demonstrate that IOR must be understood not only in

terms of S1/S2 stimulus combinations, but also in terms of S1/S2 response combinations. Consistent with prevailing views of what is inhibited by IOR, the results using saccadic RT as the dependent measure suggest that it is a motor response bias that is not specific to saccades made to peripheral visual stimulation. Importantly, this motor inhibition is consistent across response combinations - even though the stimulus combinations which support the generation of the inhibition vary. Perhaps even more critically, the fact that IOR for saccadic responses is motoric for all combinations of response whereas IOR for manual responses is motoric for only the Saccadic-Manual combination reveals an important dissociation: Contrary to their application in the literature, saccadic and manual RTs are not different rulers for measuring the same inhibitory effect; the operation of identical S1 conditions are reflected differently in saccadic and manual RTs.

Regardless of this difference between manual and saccadic RT, there is nevertheless a common conclusion: IOR for a dependent measure - whether manual or saccadic RT - must be considered with respect to both stimulus and response conditions that contribute to its genesis and measurement. The operations which may underlie the observed motoric effects for saccadic RTs will be explored in the General Discussion.

GENERAL DISCUSSION

The present study was designed to assess stimulus and response combinations which contribute to the genesis and those which allow for the measurement of IOR. The results of all six experiments are summarized in Table 18 along with relevant literature. For demonstrative purposes, the current findings are also superimposed in Table 19 on the original predictions that were derived from the literature.

EXPERIMENTS 1-6 VERSUS THE LITERATURE: SUMMARY OF IOR, AS A FUNCTION OF S1, S2, AND S1-S2 RESPONSE COMBINATIONS

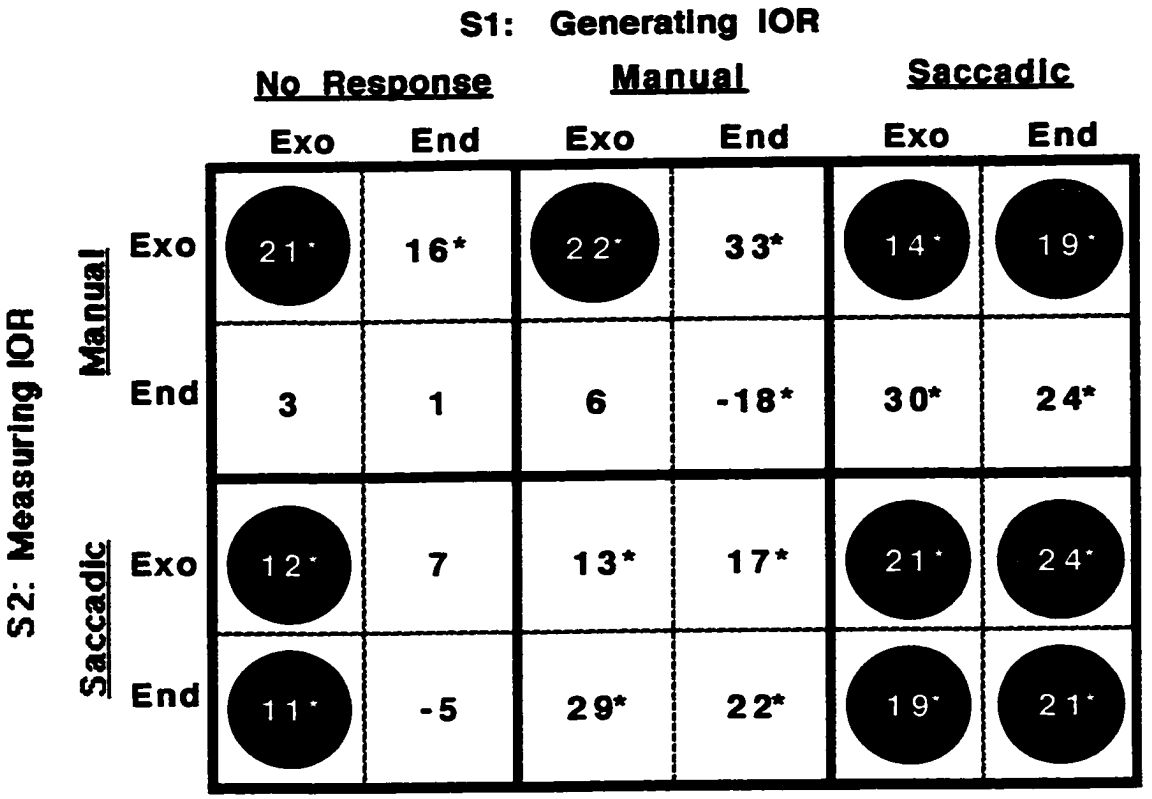

Table 18. Magnitude of IOR in Experiments 1-6. Significant effects are indicated with "". Positive values reflect IOR, negative values reflect facilitation. For comparison, cells that have been tested in the literature are indicated with circles (the black colouring indicates that IOR was observed).*

EXPERIMENTS 1-6: COMPARISON OF THE PRESENT FINDINGS WITH PREDICTIONS MADE FROM THE LITERATURE

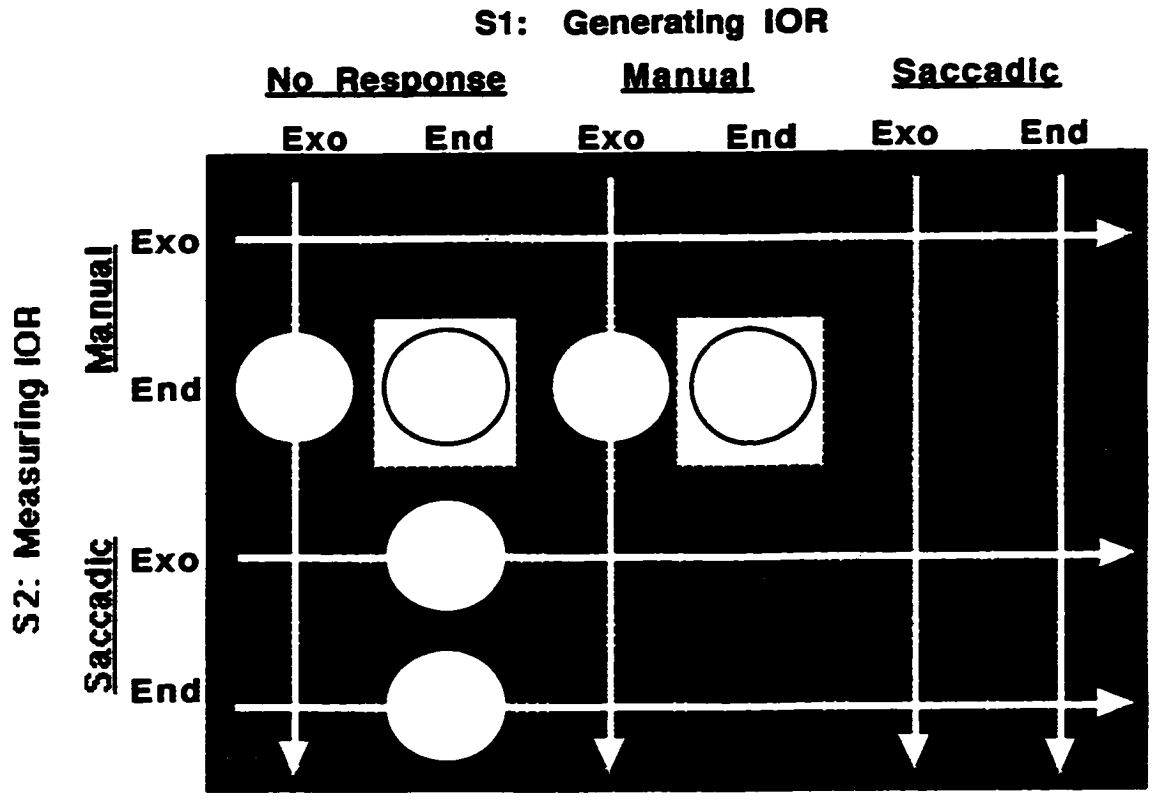

Table 19. Predictions made on the basis of the existing literature are shown with hatched bars and arrows. The present findings for Experiments 1-6 are summarized with circles. White circles indicate the absence of significant IOR; black circles indicate significant IOR.

As seen in Table 18 and emphasized with respect to each of Experiments 1-6, the general findings of all cells that have been tested in the literature were replicated by the present results. Despite this replication, however, the results of the present experiments did not conform to predictions that were based on the literature (as seen in Table 19). This was true whether the predictions were derived exclusively from cells that had been tested in the literature or else expanded to include the possibilities that: 1) Oculomotor programming may be engendered by endogenous S1s; and, 2) that a motor bias for manual responses may not be particular to peripheral visual stimuli. In the absence of blatant conflict with the literature, the implication of the present findings is that views of IOR which have emerged from that literature are not necessarily incorrect - they are merely incomplete.

In examining the stimulus/response combinations under which IOR is obtained, the following will consider the distinction already made between visuo-motor and motor inhibition. Arguments in favour of two dissociable forms of inhibition depend on implied interactions across experiments of the present design. As such, statistical tests of these interactions will be presented. Note, however, that in order to increase the power to observe any interactions that may be present, the following ANOVAs will be recast as 2-way within-subject factorial analyses of IOR, with Experiment (1-6) and S1-S2 stimulus combination (exogenous-exogenous, exogenous-endogenous, endogenous-exogenous, endogenous-endogenous) as factors (cf. Keppel, 1982).

IOR: WHAT IS INHIBITED? A RE-EVALUATION

Clearly, the issue of what is inhibited by IOR cannot be summarized with a blanket statement such as "IOR reflects a motor response bias." Indeed,

two forms of inhibition were measured by the present methods: A visuo-motor inhibition for the No Response-Manual and Manual-Manual combinations; a motor inhibition for the Saccadic-Manual, No Response-Saccadic, Manual-Saccadic, and Saccadic-Saccadic combinations. The implied five-way interaction of S1 response (No Response, Manual, Saccadic), S2 response (Manual, Saccadic), S1 signal (exogenous, endogenous), S2 signal (exogenous, endogenous), and relative locations signalled by S1 and S2 (different, same) did not reach significance when RT was examined in a full factorial ANOVA ($p<.08$); however, lending support to the view that different patterns of inhibition *did* emerge across experiments in the present study, when the power to detect the interaction was increased by recasting the ANOVA as a two-way analysis of IOR, with Experiment (1-6) and S1/S2 combination (exogenous-exogenous, exogenous-endogenous, endogenous-exogenous, endogenous-endogenous) as within-subjects factors, the interaction was highly significant ($F(15,255)=4.13$, $MSe=503.67$ $p<.00$).

As may be recalled, the designation of visuo-motor versus motor inhibition was not intended as a theoretical statement of the mechanisms underlying the observed inhibitory effects. Instead, the designation was intended to summarize the S2 stimulus conditions for which inhibition was revealed: Visuo-motor inhibition refers to IOR that occurred for exogenous but not endogenous S2s; motor inhibition refers to IOR that occurred for both exogenous and endogenous signals. These two labels were not intended to be mutually exclusive and allow for the possibility that the inhibition could be visuo-motor *and* motor; however, a "motor only" designation was made if IOR to an exogenous S2 was not greater than IOR to an endogenous S2.

As indicated in the Introduction, the question of what is inhibited by IOR has typically been addressed by manipulating the S2 stimulus/response

characteristics. The occurrence of both visuo-motor and motor inhibition makes apparent the fact that the manipulation of S2 alone provides an incomplete assessment of what is inhibited by IOR. In fact, the question must be addressed by taking into account not only the response that is used to measure the inhibition, but also the response that is used to generate the inhibition.

With this in mind, the following will explore visuo-motor and motor inhibition in turn. To preface this discussion, it is useful to highlight that the only combinations for which visuo-motor rather than motor inhibition was observed were those that did not explicitly engage the oculomotor system (i.e., the No Response-Manual and Manual-Manual combinations). Given that the literature has converged on explanations of IOR that emphasize oculomotor involvement in both the genesis and measurement of IOR, the visuo-motor inhibition observed for the No Response-Manual and Manual-Manual combinations proves to be contentious with respect to the analysis that was presented in the Introduction. As such, possible mechanisms for visuo-motor inhibition will be explored more fully than for motor inhibition. The issue of *h o w* each type of inhibition is generated will be considered subsequently.

Visuo-Motor Inhibition: IOR for Exogenous but not for Endogenous S2s

As noted, visuo-motor inhibition occurred for the No Response-Manual and Manual-Manual response combinations and not for any combination that included an explicit saccadic response to either S1 or S2 (i.e., because IOR for exogenous S2s was not greater than for endogenous S2s in these conditions, they are said to show motor inhibition only). Importantly, this dissociation of visuo-motor and motor inhibition cannot be accounted

for by appealing to differences in S1 stimuli/responses or S2 stimuli: When subjects withheld or else made a manual response to S1, manual RTs revealed visuo-motor inhibition whereas saccadic RTs revealed motor inhibition. This impression is confirmed by an analysis of IOR as a function of Experiment (No Response-Manual, Manual-Manual, No Response-Saccadic, Manual-Saccadic) and S1-S2 response combination (exogenous-exogenous, exogenous-endogenous, endogenous-exogenous, endogenous-endogenous): There was a significant effect of Experiment ($F(3,51$, $MSe=846.15$, $p<.05$), of S1-S2 combination ($F(3,51)=7.57$, $MSe=693.26$, $p<.00$), and a significant interaction of Experiment and S1-S2 combination ($F(9,153)=4.46$, $MSe=542.54$, $p<.00$).

Of course, the significant interaction of Experiment and S1-S2 combination in the preceding analysis could stem from the large facilitatory effect in the endogenous-endogenous cell of the Manual-Manual response combination of Experiment 2. As such, this analysis was repeated after eliminating the endogenous-endogenous combination from consideration. Again, there was a significant effect of Experiment ($F(3,51)=1.27$, $MSe=949.36$, $p<.30$), of S1-S2 combination ($F(2,34)=1.16$, $MSe=706.44$, $p<.33$), and a significant two-way interaction of these factors ($F(6,102)=4.07$, $MSe=422.52$, $p<.00$). When this interaction was further parceled by examining the pattern of IOR across the exogenous-exogenous, exogenous-endogenous, and endogenous-endogenous combinations as a function of the S2 response (i.e., No Response-Manual and Manual-Manual versus No Response-Saccadic and Manual-Saccadic), a significant interaction of S2 response and S1-S2 response combination ($F(2,34)=10.90$, $MSe=364.14$, $p<.00$) was revealed. Thus, confirming the view that there are different patterns of inhibition, the particular form of IOR that is revealed as a function of S1-S2 stimulus

combinations is dependent upon whether the response used to measure the effect is manual or saccadic.

Give that there does appear to be a dissociation between manual versus saccadic responses to S2 in terms of whether a visuo-motor or a motor inhibition is observed, it follows that an account of visuo-motor inhibition must focus on aspects that are specific to manual versus saccadic responses to S2. This allows two possible mechanisms for visuo-motor inhibition to be ruled out immediately: Visual-sensory processing and oculomotor programming.

With respect to visual-sensory processing, recall that several lines of evidence were identified in the Introduction as being contrary to a purely visual-sensory basis of IOR. Included in this evaluation were the observations that IOR is outside of the temporal range of low-level sensory masking (e.g., Tassinari et al., 1989); shows inter-ocular transfer (e.g., Tassinari & Berlucchi, 1993); is spatially-graded (e.g., Maylor & Hockey, 1987); and, is coded environmentally (Maylor & Hockey, 1987). Nevertheless, the fact that manual RTs in the No Response-Manual and Manual-Manual conditions showed inhibition only when directed by an exogenous S2 begs the question of whether detection of the peripheral visual signal was inhibited by IOR. However, if a visual processing deficit had been engendered by virtue of withholding or making a manual response to S1, then manual and saccadic responses would both have been slowed to a peripheral signal. According to the analysis summarized in Table 18, this did not occur - no IOR was observed for the endogenous-exogenous cell of the No Response-Saccade combination.

Given that exogenous signals are closely linked to activation of the oculomotor system, if it is accepted that visuo-motor inhibition cannot be explained with reference to visual-sensory processing, the next obvious

consideration is that it reflects inhibited oculomotor programming. However, if this were the case, then saccadic responses (which depend on oculomotor programming) should have been impacted by the identical S1 stimuli/responses that produced IOR for the No Response-Manual and Manual-Manual combinations. As seen in Table 18, this did not occur: An endogenous S1 requiring no response generated IOR for manual responses to exogenous S2s but not for saccadic responses to either exogenous or endogenous S2s.

Thus, on the grounds that there is a dissociation between the No Response-Manual and No Response-Saccadic endogenous-exogenous cells, it is possible to dismiss both a visual-sensory processing account and an oculomotor activation account of the visuo-motor inhibition. Although the analysis summarized in Table 18 supports this conclusion, it should be noted that the endogenous-exogenous cell of the No Response-Saccadic combination did not differ significantly from the No Response-Manual combination ($F<1$). As such, it remains possible that a Type II error was operating in the former. As will be seen below, however, a detailed consideration of the motor inhibition account suggests that a visuo-motor inhibition is unlikely to be operating in Experiments 3-6. To foreshadow that argument: Because IOR was not greater for exogenous versus endogenous S2s, it is more parsimonious to conclude that a single motor inhibition was operating in Experiments 3-6, rather than assuming that a visuo-motor inhibition was operating for exogenous S2s and that a separable motor inhibition was operating for endogenous S2s (except when a response is withheld to a preceding endogenous S1 in the No Response-Saccadic condition!). Nevertheless, parsimony aside, the present dismissal of a visual inhibitory effect rests on the analysis presented in Table 18 (i.e., a binary

decision regarding whether IOR is present/absent) and should therefore be accepted with caution.

Accepting the view that visuo-motor inhibition is specific to manual responses and therefore cannot be tied to visual processing or oculomotor programming, two possible mechanisms present themselves. The first presumes that the visuo-motor inhibition actually operates at the interface of visual processing and manual programming/execution. On this view, IOR represents a deficit for using peripheral visual signals to program and/or execute a manual response. This reflects a *modality-specific* form of the motor response bias that was outlined in the Introduction. Although a reasonable description of the obtained inhibitory effect, to the extent that manual and saccadic RTs show a dissociation with respect to the endogenous-exogenous cells when no response is made to S1, this view suffers from the fact that it requires that IOR be represented in different *response* systems - in the motor system for manual responses and in the oculomotor system for saccadic responses. In other words, it presumes that manual and saccadic RTs reflect different inhibitory effects rather than different components of the same inhibitory process. This possibility cannot be denied on the basis of the current findings. The second plausible explanation resurrects an attentional view of IOR. However, rather than assuming that the visuo-motor inhibition represents inhibition of attentional orienting generally, this view postulates that it is inhibition of exogenous orienting per se that is inhibited by visuo-motor IOR.

For an exogenous attentional view of the visuo-motor inhibition to hold, it must be able to account for the supposition that manual RTs revealed visuo-motor inhibition when saccadic RTs did not (recall that this supposition depends on the existence of significant IOR for the endogenous-

exogenous cell of the No Response-Manual but not for the No Response-Saccadic combination). There is obviously a tight link between covert exogenous attention and saccadic eye movements: Preparation to execute a saccade to a peripheral onset target speeds the detection of signals arising at the location of the saccade goal (e.g., Chelazzi et al., 1995; Hoffman & Subramaniam, 1995; Kowler et al., 1994; Posner & Cohen, 1980; Shepherd et al., 1986); conversely, deployment of covert attention to a peripheral onset target speeds the execution of a saccade (Kowler, et al., 1994; Remington, 1980). This functional relation, however, does not imply that the deployment of covert exogenous attention and saccadic programming are identical processes. Indeed, important dissociations between saccadic orienting and covert attentional orienting to exogenous cues are reported by Shulman (1984) and by Reuter-Lorenz and Fendrich (1992). Given the centrality of these dissociations for applying an attentional account to the current data, these studies will be presented in some detail.

Shulman (1984) was interested in determining whether a temporal field bias observed by Posner and Cohen (1980) for saccades would be revealed by covert attentional orienting. Under monocular viewing conditions, Posner and Cohen (1980) had presented subjects with target dots at 10 degrees eccentricity. On a third of the trials, the left or the right target dot led by 150 msec; on a third of the trials, the left or the right target dot led by 500 msec; and, on a critical third of the trials, both dots were onset simultaneously. The subjects' task was to move their eyes in the direction that felt most comfortable. Not surprisingly, when there was 150 msec or 500 msec lead time, subjects moved their eyes in the direction of the target dot that actually led. However, on trials when the two dots were onset simultaneously, subjects tended to move in the direction of the temporal field (relative to the

open eye).

To determine whether a temporal field bias represents an isolable component of the saccadic eye movement system, Shulman (1984) repeated Posner and Cohen's (1980) basic paradigm using covert attentional cues rather than eye movement targets. Under monocular viewing conditions, he presented subjects with exogenous cues to the left or right of fixation. These were onset with an asynchrony of 0, 15, 30, 50, 75, or 125 msec. At an SOA of 125 msec relative to the second cue, a target requiring a manual detection response was onset with equal probability to the left or right. The temporal field bias hypothesis predicted the capture of exogenous covert attention by the temporal hemifield cue. This did not occur. When the two cues were onset with a non-zero SOA, then regardless of field, target detection was always fastest at the location of the second cue (although see Rafal, Henik, & Smith, 1991); and, critically, when the two cues were onset with 0 msec SOA, the speed to make the target detection was the same in both fields, indicating the absence of a bias to attend to the temporal field cue.

A dissociation between saccadic and covert exogenous orienting was likewise reported by Reuter-Lorenz and Fendrich (1992). Reuter-Lorenz and Fendrich (1992) were interested in exploring a tenet of the pre-motor theory of attention (Rizzolatti et al., 1987; Sheliga, Riggio, & Rizzolatti, 1994; see also Hughes & Zimba, 1987) which states that RT costs for reorienting covert visual attention across the vertical midline is accounted for by the speed to reprogram the direction of a saccade. To test this hypothesis, they presented subjects with four horizontally-arranged outline boxes, one of which was brightened at the beginning of a trial. In one condition, subjects were required to make a speeded manual detection response to a target that appeared subsequent to the cue; in another, subjects were required to make a

speeded saccade.

Challenging the pre-motor theory, Reuter-Lorenz and Fendrich (1992) showed that neither the manual nor the saccadic measure revealed greater costs for having to reorient across the vertical meridian versus having to reorient an equivalent distance within a hemifield. Nevertheless, there was a dissociation, wherein manual RTs revealed greater costs when an invalid cue was eccentric to the target rather than vice versa; the opposite was true for saccadic RTs. Because the overall magnitude of the cueing effect was the same for both dependent measures, Reuter-Lorenz and Fendrich (1992) argued that the dissociation was not due to the imposition of an additional stage of processing for one measure that was not present for the other. Instead, they suggested that attentional reorienting and saccadic reprogramming are accomplished by parallel, independent mechanisms. Their conceptualization of the production of manual and saccadic responses to invalidly-cued targets is shown in Figure 22.

Independence of saccadic and attentional orienting to exogenous signals is consistent with the need to identify a basis for visuo-motor inhibition that could impact manual responses without coincident impact on saccadic responses. Moreover, couching an explanation in terms of exogenous covert orienting provides the possibility of a common neural substrate (even if not completely overlapping pathways) for inhibitory effects that are measured by manual and saccadic RTs. In particular, beyond its role in saccadic orienting, there is evidence from both humans and monkeys that the SC subserves exogenous covert orienting.

With regard to the human data, PSP patients tested in an attentional orienting paradigm show marked impairment for responding to both validly- and invalidly-cued targets (Posner, Cohen, & Rafal, 1982; Rafal, Posner,

REUTER-LORENZ AND FENDRICH'S (1992) MODEL OF SACCADIC PROGRAMMING AND ATTENTIONAL REORIENTING

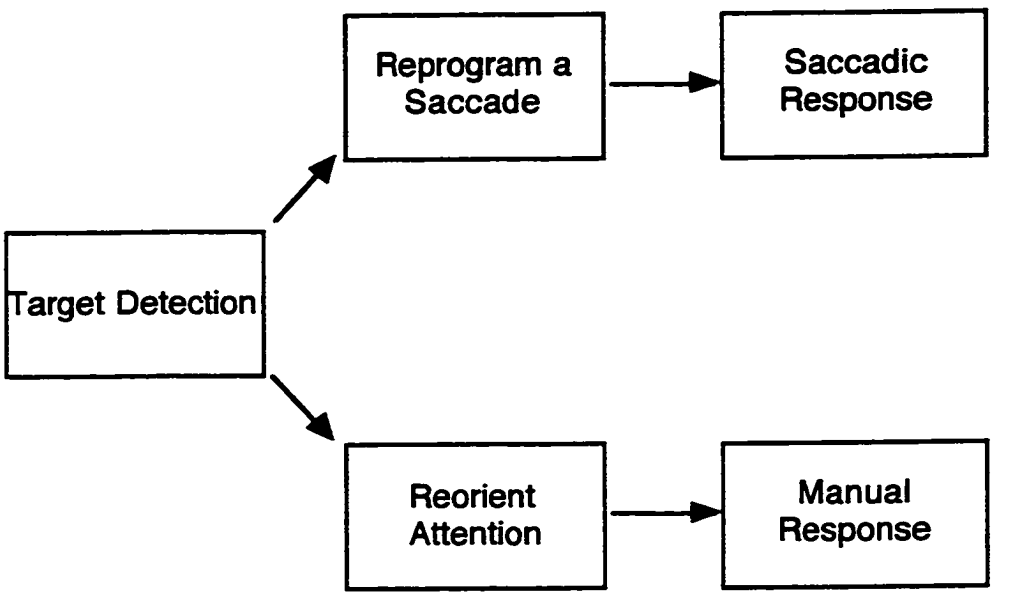

Figure 22. Reuter-Lorenz and Fendrich's (1992) model of saccadic programming and attentional reorienting. In this conceptualization, the production of manual and saccadic responses is accomplished via two distinct, parallel pathways.

Freidman, Inhoff, & Bernstein, 1988). This pattern of impaired attentional orienting to cues and targets alike is consistent with the view that lesions of tectal and peri-tectal regions result in a deficit to move covert attention to the location of an exogenous signal. Similar effects are observed following SC lesions in monkeys (as reported in Colby, 1990). In particular when muscimol (a GABA agonist) is injected unilaterally into the SCs of monkeys trained to maintain fixation in an orienting task, RTs are slowed to both validly- and invalidly-cued targets that appear contralesionally. Desimone, Wessinger, Thomas, and Schneider (1989) likewise report deficits in covert orienting to stimuli that appear in the receptive fields of SC cells that are electrically rather than chemically lesioned. And, the obverse finding that stimulation of the SC results in a shift of attention to the contralateral field is reported by Gattas and Desimone (1992).

This role of the SC in exogenous attentional orienting makes a modified attentional account of the present data more parsimonious than the alternative explanation in terms of inhibition at the visual-(manual)motor interface. Whereas both views are consistent with the observed dissociation of manual and saccadic RTs with respect to visuo-motor inhibition, only the modified attentional account is consistent with a unitary inhibitory process. In particular, rather than arguing that visuo-motor inhibition is modality-specific, by virtue of a common collicular basis with saccadic eye movements, the modified attentional account allows for the possibility that visuo-motor and motor inhibition are tapping different facets of the same inhibitory mechanism. Of course, if an attentional view is to be adopted, it must be able to accommodate not only the current data, but also that which is reported in the literature.

Recall that an attentional view of IOR was discounted in the

Introduction, on the grounds that no impairment is observed for either the speed or quality of perceptual processing. With respect to the speed of developing a percept, it was observed that neither temporal order judgement tasks (e.g., Maylor, 1985; Posner et al., 1985) nor those that measure the frequency of illusory line motion (e.g., Schmidt, 1996b) are sensitive to IOR. And, with respect to the quality of perceptual processing, it was observed that IOR does not occur for RTs that are based on non-localization discrimination responses. However, these failures to observe IOR for either the speed or quality of perceptual processing (i.e., due to attentional allocation) can be explained if a modified attentional view is applied in lieu of an attentional account that fails to distinguish between exogenous and endogenous systems.

Recall that in the temporal order judgement and the frequency of illusory line motion paradigms, subjects are typically presented with an exogenous cue to which no response is required. Following this cue at a variable onset asynchrony, subjects are then presented with a lead and a following target dot (temporal order judgement) or else with an onset line about which a judgement of illusory motion is required. The critical issue is whether exogenous attention is slow to move to the location that had been cued. When making judgements of temporal order or illusory line motion, subjects are usually instructed to sacrifice speed for accuracy. As such, the failure to observe IOR for making such judgements is not prima facie evidence against an attentional basis of IOR: Sluggish movement of attention to the previously-cued location could be lost in the response time delay associated with making an accurate judgement.

Consistent with this view, Gibson and Egeth (1994) report conditions under which the latency to make temporal order judgments reflects IOR even when accuracy is not impacted. In particular, Gibson and Egeth (1994) found

that when the onset of the two target dots was nearly simultaneous (e.g., 14 msec), neither RTs nor accuracies reflected the operation of IOR. However, when there was a delay between the two targets dots, then IOR occurred for RT but not for accuracy. These are precisely the results that would be expected based on an exogenous attentional account of what is inhibited by IOR. In particular, when the two dots are onset simultaneously, the bilateral visual input would not bias the capture of attention to one location versus the other. In contrast, when there is a delay between the two target dots, then the onset of the lead dot would capture covert exogenous attention. As such, to the extent that the movement of covert attention is slowed by IOR, it is under conditions of non-simultaneous target onset that IOR would be expected to impact RT.

Gibson and Egeth's (1994) finding of IOR for the speed to make a temporal order judgement indicates that the typical emphasis on accuracy in this task might have obscured the presumed inhibition for exogenous attentional orienting. Attentional mechanisms of IOR might have similarly been obscured in non-spatial discrimination tasks intended to index IOR. This is because the tasks which have typically failed to reveal IOR for non-spatial discriminations have: 1) Presented target stimuli that would be sensitive to endogenous - but not exogenous - attentional orienting; and/or, 2) confounded the generation and measurement of IOR.

With respect to the nature of the target stimuli, consider a study performed by Pontefract and Klein (1988). They presented subjects with a horizontal display of three stimulus markers, each comprised of four dots at the corners of an imaginary box and one dot in the centre. One of the two peripheral stimulus markers could brighten and, after an SOA of 100 or 500 msec, one of the two boxes expanded or contracted via respective movement

of the four corner dots away from or toward the centre dot. When subjects were required to detect the size change, IOR was observed at the long SOA; however, when subjects were required to make a manual discrimination based on the type of size change (expansion, contraction), no IOR was obtained. This pattern of data was foundational for Klein and Taylor's (1994) arguments against an attentional locus of IOR: They reasoned that if attentional allocation were inhibited by IOR, then perceptual processing (as indexed by the discrimination task) would have suffered at the previously-cued location. By re-considering the attentional processes that would be elicited by the demands of the detection and discrimination tasks, however, it is possible that Klein and Taylor's (1994) evaluation is compromised by their neglect of the exogenous/endogenous distinction.

As has already been explicated, exogenous attentional orienting is accomplished bottom-up via activation of transient visual channels whereas endogenous attentional orienting is accomplished top-down via the generation of spatial expectancies. Importantly, these two forms of orienting are not simply different means for allocating the same limited pool of attentional resources; instead, they are isolable orienting systems that are subserved by different mechanisms and that contribute to unique aspects of perceptual processing (e.g., Briand & Klein, 1987; Klein et al., 1992). This isolability is acknowledged by the suggestion that IOR may impact the rapidity with which exogenous attention is deployed to a previously-cued location, without impacting the rapidity with which endogenous attention is deployed. By extension, if the perceptual demands of a target task require the allocation of endogenous attentional resources, then the unimpeded movement of those resources may eclipse any inhibitory effects that may be operating on the speed to allocate exogenous attention.

Applying this analysis to Pontefract and Klein (1988), it is possible that their detection and discrimination tasks summoned different attentional resources and, therefore, were differentially sensitive to the effects of exogenous attention. Specifically, it is likely that the local luminous flux due to the expansion/contraction of the target stimulus initiated the movement of exogenous attention. In contrast (in the absence of a strong perception of movement), the perceptual discrimination of size change would likely have required the endogenous allocation of attention to a representation of the visual array - in order determine whether the change made the boundaries of the target marker larger or smaller than the non-target markers (i.e., it was a relative size judgement). If so, then Pontefract and Klein's (1988) results are consistent with the notion that IOR represents inhibited exogenous attention. This conclusion follows from two suppositions: 1) That their simple detection task would have been sensitive to slowed exogenous orienting to the location of the luminous flux; whereas, 2) their discrimination task would have required the allocation of endogenous attention in order to analyze the nature of the size change; this uninhibited movement of endogenous attention would have overshadowed the inhibited movement of exogenous attention. To the extent that this analysis is correct, then a perceptual discrimination that does not depend on top-down comparison of target/non-target stimuli should reveal IOR.

A study by Tanaka and Shimojo (1996) that satisfied this condition but that nevertheless failed to obtain IOR can be challenged from a different vantage point. Tanaka and Shimojo (1996) had subjects fixate a central cross. Relative to fixation, a single coloured target rectangle appeared 6 degrees visual angle to the upper right and upper left. Target location (left, right), colour (red, blue), orientation (vertical, horizontal), RSI (100, 300, 500, 1200

msec) were all varied randomly. Subjects performed in different sessions, defined by the required response: 1) detection; 2) localization; 3) colour discrimination; 4) orientation discrimination. No cues were presented. Instead, a subjects were presented with a continuous series of targets; IOR was assessed as slowed RT to a target on trial N that shared the same discriminated target attribute as the target on trial N-1. When RTs were thus analyzed according to the positional relationship between targets on consecutive trials, the results demonstrated IOR for the detection and localization tasks but facilitation for repeated-location targets in the colour and orientation discrimination tasks. These findings were replicated using luminance discrimination, vernier acuity, and judgements of line length.

Note that all of Tanaka and Shimojo's (1995) discrimination targets were presented singly and were defined by features or simple conjunctions of features. Given that exogenous attention is attracted to abrupt luminance onsets and subserves visual search by conjoining free-floating features into a single percept (Briand & Klein, 1987), Tanaka and Shimojo's (1995) discrimination targets were ideal for assessing inhibitory influences on exogenous attention. Even so, their paradigm was not. By employing a response-response paradigm for both their detection and their discrimination tasks, Tanaka and Shimojo (1995) confounded the methods used to measure IOR with the methods used to generate IOR (see also Kwak & Egeth, 1992; Terry, et al., 1994). As such, it is difficult to determine whether non-spatial discrimination tasks fail to generate IOR (e.g., because endogenous attentional resources are maintained at the peripheral location in order to perform the discrimination) and/or whether they fail to measure IOR as Tanaka and Shimojo (1996) maintain. When this confound is eliminated by using a cue-target procedure with singly-presented onset targets, then IOR is, in fact,

observed for non-spatial manual discriminations of colour (Lupianez et al., 1997), identity (Danziger et al., 1997; Pratt et al., 1997), and line orientation (Handy, Jha, & Mangun, 1997).

Of course, the fact that - at least under some conditions - IOR reflects inhibition for the speed and quality of perceptual processing does not obligate an attentional account. Indeed, using successful demonstrations of inhibited perceptual processing as evidence for an attentional view violates modus tollens (see the Introduction). Nevertheless, of the two plausible alternatives that can account for the visuo-motor inhibition observed in the No Response-Manual and Manual-Manual conditions of the present study, the attentional hypothesis is favoured. By postulating that visuo-motor IOR for these response combinations is due to the inhibited movement of covert exogenous attention, dissociations between manual and saccadic RTs in the present study can be accounted for; a common substrate for IOR assessed by manual and saccadic responses can be assumed; and, inconsistencies in the literature can be explained. On the strength of the view that IOR for No Response-Manual and Manual-Manual response combinations is therefore due to inhibited exogenous attentional movements, the ongoing debate between an attentional and motoric basis for IOR can be quelled. Rather than debating *whether* IOR is attentional, the current findings suggest that the literature should, instead, delineate the conditions that determine *when* IOR is attentional.

Motor Inhibition: IOR for Exogenous and Endogenous S2s

In addition to a visuo-motor inhibition for the No Response-Manual and Manual-Manual combinations, the present results also revealed a strictly motor inhibition. Recall that the designation of motor inhibition was made

when IOR was observed for responses to both exogenous and endogenous S2s. In other words, motor inhibition captures a general reluctance to respond to an S2 that signals the same direction as a preceding S1 - regardless of where S2 is presented. As seen in Table 18 and noted earlier, under this definition, motor inhibition was observed for any response combination that included a saccade (whether to S1 or S2): Saccadic-Manual, No Response-Saccadic, Manual-Saccadic, and Saccadic-Saccadic.

A bias against responding in the direction of a preceding S1 is consistent with conceptualizations of IOR that were derived from the literature. With respect to manual RTs, endogenous S2s had not been explored previously. As such, in making predictions, it was unclear whether a manual motor bias would be specific for responses made to peripheral stimuli or whether it would also occur for responses made to central signals. The current study demonstrates that a manual motor response bias is not limited to exogenous S2s. However, the present findings qualify the view of a general motor response bias by demonstrating that this inhibition occurs only when a saccade is made to the preceding S1: On the strength of previous arguments, the inhibition observed in the No Response-Manual and Manual-Manual conditions was attentional, rather than motoric.

With respect to saccadic responses, the literature suggests that the motor bias is a general reluctance to respond in the direction of the preceding S1 and therefore does not depend on the nature of the S2 signal. The present results are broadly consistent with this prediction. However, the fact that IOR does not occur for saccades that follow an endogenous S1 to which a response is withheld indicates the saccadic motor response bias depends, in part, on the conditions used to generate the inhibition.

The fact that motor inhibition can be observed for manual and saccadic

RTs - even if under limited conditions - argues that whatever is inhibited must be common to both responses and must not depend on the nature of the S2 stimulus. In their original conceptualization of what is inhibited by IOR, Klein and Taylor (1994) argued that IOR is a motor inhibition that results from an impaired ability to respond based on target localization. In the case of saccadic responses (which require localization of the targeted location), such an account can accommodate the fact that a motor bias occurs for both exogenous and endogenous S2s. However, two challenges arise in the case of manual responses.

The first challenge to a localization view of what is inhibited by motor IOR arises from the suggestion that manual detection responses may reveal motor inhibition. In particular, the exogenous-exogenous and endogenous-exogenous cells of the present Saccadic-Manual condition replicate results obtained by Rafal et al. (1989) using manual detection responses (see also Posner & Cohen, 1984). It is therefore reasonable to assume that the motor bias revealed via the comparison of exogenous and endogenous S2s would be replicated if manual detection, rather than localization, responses were used. The second challenge stems from the observation that manual responses to endogenous S2s revealed motor inhibition in the Saccade-Manual condition. The possibility of motor inhibition for detection responses can be accommodated by a localization view of motor IOR if it is assumed that the need to detect peripheral information results in implicit localization (cf. Klein & Taylor, 1994); the fact of motor inhibition for manual responses made to central arrows is not accommodated as easily.

Despite these challenges, a localization view of motor IOR is rescued, in part, by the dependence of motor inhibition on the occurrence of a saccadic response. Consider that the execution of a saccadic response - whether to S1

or S2 - requires the accurate localization of the targeted location. As such, the saccadic system must specify the vector coordinates of a targeted location. If it could be assumed that the act of making a saccade is capable of both generating and accessing inhibition within a spatial map that specifies the target vector, then it is conceivable that manual and saccadic responses would suffer from the degraded coding of direction and/or amplitude. Although highly speculative, by emphasizing the critical role that saccades play in the generation/measurement of motor IOR, such an account would be consistent with an SC basis of the effect.

Regardless of the mechanism underlying the observed motor inhibition, the present results emphasize that what is inhibited by IOR depends on the combination of S1 and S2 responses. In the case of Saccadic-Manual, No Response-Saccadic, Manual-Saccadic, and Saccadic-Saccadic combinations, there is a general bias against responding in the same direction as a preceding S1. This contrasts with the apparent inhibition of exogenous attention which was revealed for the No Response-Manual and Manual-Manual combinations. The following will turn to a consideration of S1 stimulus/response factors that generate visuo-motor and motor inhibition.

IOR: HOW IS THE INHIBITION GENERATED?: A RE-EVALUATION

As was the case with the issue of what is inhibited, the question of how IOR is generated cannot be answered with a blanket statement such as "IOR is generated by oculomotor programming." There is clearly an interaction between the stimulus/response combinations that are used to measure and those that are used to generate IOR. As such, the question is not "how is IOR generated?" but rather "how are visuo-motor and motor IOR generated?" The following will explore the generation of visuo-motor and motor

inhibition in turn.

Visuo-Motor Inhibition: Generating IOR for Exogenous S2s

As observed in Table 18, whether a visuo-motor or motor effect was observed for manual RTs was determined by the S1 response. A comparison of the S1 response conditions therefore suggests a fruitful means for exploring how visuo-motor inhibition is generated. Fundamental to this analysis is the observation that IOR occurred for manual responses that were made to endogenous S2s that followed a saccadic response, but not those that followed no response or else a manual response to S1; this was true whether S1 responses were directed by exogenous or endogenous signals. This suggests that the mechanism which underlies the generation of visuo-motor IOR is not the same as that which underlies the generation of motor IOR. By focusing on conditions that may have led to inhibition for endogenous manual responses, it is therefore possible to eliminate possible explanations for how the visuo-motor inhibition was generated.

For now, consider only the exogenous S1 conditions of the No Response-Manual, Manual-Manual, and Saccadic-Manual combinations. What are the possible processes initiated by the exogenous S1 signal in the Saccadic-Manual condition that were not similarly initiated in the No Response-Manual and Manual-Manual conditions? When a saccade is made to an exogenous S1, a vector must be programmed in response to the signal and then executed. If it were the case that the motor inhibition observed for manual RTs that follow saccades to exogenous S1s was generated by oculomotor programming, then the No Response-Manual and Manual-Manual conditions would have demonstrated the same pattern of IOR. This is because even in the absence of saccadic execution, exogenous S1s generate

oculomotor programming. Conversely, given that the same pattern of IOR was not observed for the No Response-Manual and Manual-Manual combinations (i.e., there was no IOR in the exogenous-endogenous cells) it follows that the inhibition observed for these response combinations is not likely generated by oculomotor programming.

Recall, however, that in addition to generating oculomotor programming, exogenous signals initiate orienting of exogenous covert attention. Because covert orienting can occur independently of saccadic programming, postulating that IOR in the No Response-Manual and Manual-Manual conditions may have been generated by attentional orienting is not contradicted by the pattern of data observed in the Saccadic-Manual cells. However, before an attentional view can be forwarded, it is necessary to expand the analysis of visuo-motor inhibition to the case where the S1 signal is endogenous.

Postulating a role for attention in the generation of visuo-motor IOR introduces a quandary with respect to endogenous S1s. Recall that a defining feature of IOR is the inability to observe inhibition following endogenous covert orienting (e.g., Posner & Cohen, 1984; Rafal et al., 1994). As such, it would seem dubious to claim that the endogenous S1s of the present study were, in fact, able to generate IOR. However, before this possibility is rejected, consider a distinction that was forwarded by Friedrich, Rader, and Knauts (1997). In a study of spatial expectancy effects on attentional orienting, Friedrich et al. (1997) found that manual detection responses were facilitated for targets that appeared in the direction of a centrally-presented arrow - even in the absence of incentive to attend in the cued direction. The facilitatory effect observed with unpredictive cues was of smaller magnitude than when a probabilistic meaning was attached to the arrow. On the basis of these

findings, the authors suggested that a central arrow engages two attentional operations. The first is the initiation of attentional orienting that occurs because of the well-learned symbolic meaning associated with a directional arrow. This initiation of attentional orienting occurs even when the cue does not predict target location. Once attentional orienting has been initiated, then the second process is the allocation of attentional orienting according to any probabilistic meaning associated with the cue. If the arrow points to the location most likely to contain a target, then attention is allocated and maintained at the cued location; if, on the other hand, the arrow points to the location least likely to contain a target, then attention is directed away from the location to which the arrow is pointing.

Accepting this distinction between the initiation and subsequent allocation of endogenous attention, it is therefore possible to assert that previous failures to observe IOR following endogenous covert orienting may have been due to the probabilistic nature of the cues. If this is the case, then the present suggestion that IOR *is* generated by an unpredictive endogenous signal is not at variance with previous failures to define a role for endogenous attentional orienting. As such, the present findings converge on the possibility that visuo-motor IOR in the No Response-Manual and Manual-Manual conditions is generated by covert attention[8].

Of course, reviving an attentional hypothesis for how visuo-motor IOR is generated must be reconciled with the literature. Three lines of evidence were presented in the Introduction as being contrary to an attentional view of IOR generation. The first was based on Maylor's (1985) demonstration that IOR for saccadic RTs can occur in the absence of facilitation at early SOAs. As the present study makes apparent, this finding does not challenge the current attentional hypothesis: The IOR that is

accessed by saccadic responses is not identical to that which is accessed by manual responses. The second line of evidence was Lambert and Hockey's (1991) demonstration that facilitation and IOR are not equivalently affected by practice. In particular, they noted that facilitation is eliminated by practice when high salience cues are used whereas the magnitude of IOR remains virtually unchanged over blocks of trials. This dissociation of early facilitation and IOR is potentially damaging for an attentional account of how IOR is generated. However, given that practice effects have not been consistently observed (e.g., Maylor, 1985; Maylor & Hockey, 1987) a replication of this finding would seem to be in order before giving it too much credence. The third line of reasoning that was originally used to discount an attentional view pointed to inconsistencies in the outcome of studies designed specifically to test for the role of covert orienting in the genesis of IOR. These inconsistencies were only alluded to in the Introduction and so will be explicated more fully at this juncture.

On the view that covert visual orienting cannot be allocated simultaneously to non-adjacent spatial locations[9], Posner and Cohen (1984) and Maylor (1985) reasoned that to the extent it is established by attentional orienting, IOR should be exhibited when a cue is presented unilaterally at one of two possible target locations but not when cues are presented bilaterally. The results were inconclusive. Whereas Posner and Cohen (1984) showed no reduction in the magnitude of IOR for bilateral versus unilateral cues, Maylor (1985) reported that the magnitude of IOR was halved in the bilateral condition. A third study, performed by Tassinari and Berlucchi (1993), resolves the controversy by suggesting that the choice of cue-target SOAs may have determined the discrepant results of Posner and Cohen (1984) and Maylor (1985). At least for SOAs longer than 600 msec, Tassinari and

Berlucchi's (1993) results are consistent with an attentional basis for IOR.

What these studies make apparent is that arguments which counter an attentional basis of IOR are not considerably stronger than arguments in support of an attentional basis. Given that an attentional view seems best able to account for the present study and cannot be easily discounted with respect to the existing literature, the cautious conclusion of the present analysis is that visuo-motor inhibition is generated by covert attentional processes. It is unknown whether the postulated initiation of endogenous orienting by a central arrow would be expected to operate on the same SC mechanisms that were outlined previously for the allocation of exogenous attentional mechanisms. Clearly, the supposition that IOR is generated by attentional processes engaged by unpredictive exogenous and endogenous cues would benefit from the specification of a neural substrate that could mediate the measurement of these effects by manual responses to exogenous but not endogenous S2s.

Motor Inhibition: Generating IOR for Exogenous and Endogenous S2s

As seen in Table 18, motor inhibition was observed for the Saccadic-Manual, No Response-Saccadic, Manual-Saccadic, and Saccadic-Saccadic combinations. Where motor inhibition is defined as the observation of IOR for responses made to both exogenous and endogenous S2s, it is clear that the generation of this effect is not specific to any particular S1 stimulus/response combination.

With respect to manual RTs, execution of a saccadic response -whether to an exogenous or an endogenous S1 - was the only condition capable of generating motor IOR. This has two implications. First, oculomotor programming is not sufficient to produce motor inhibition of manual

responses. This conclusion follows from the fact that the exogenous S1s of the No Response-Manual and Manual-Manual conditions are expected to engender oculomotor programming and yet did not yield a similar impact on manual RT as the exogenous and endogenous S1s of the Saccadic-Manual condition. Second, the preparation/execution of any motor response is not sufficient to produce motor inhibition of manual RTs. This follows from the fact that a similar pattern of inhibition was not observed in the Manual-Manual and Saccadic-Manual conditions, both of which required an explicit response to S1. As such, the generation of manual motor inhibition requires the explicit execution of a saccadic response.

When an explicit saccadic response is made to S2, however, the S1 conditions that generate motor inhibition are more varied. Examination of the pattern of data across the No Response-Saccadic, Manual-Saccadic, and Saccadic-Saccadic combinations reveals that any motor activation is sufficient to generate motor inhibition for saccadic RTs. For example, consider the No Response-Saccadic condition. Here, according to the analysis summarized in Table 18, an exogenous S1 generated motor inhibition whereas an endogenous S1 did not. The most obvious distinction between these S1 conditions is that the former results in oculomotor programming whereas the latter does not. This implicit activation of a motor response is sufficient to produce inhibition of saccadic responses. Likewise, explicit activation of a manual or saccadic response was also effective in generating IOR for saccadic RTs.

The fact that the S1 conditions capable of generating motor inhibition differ according to whether the IOR is measured by manual or saccadic RTs would seem to suggest that manual and saccadic RTs are not assessing the same type of inhibition. However, as discussed previously, there is a shared

characteristic of the response combinations that reveal motor inhibition: They all require a saccadic response to either S1 or S2. As such, it is more parsimonious to assume that the engagement of the oculomotor system - whether with respect to S1 or S2 - is capable of generating and/or accessing a general motor inhibition that can be measured by manual and saccadic RTs alike. A speculative SC-based mechanism for this inhibition was suggested earlier and will not be reiterated here.

Regardless of the underlying mechanism, it is clear that the execution of a saccadic response to either S1 or S2 sets the stage for obtaining motor inhibition of manual and saccadic RTs. The need to conjointly specify the responses to S1 and S2 in order to predict motor IOR buttresses the rationale for the present studies. Recall that one of the motivations for manipulating S1/S2 stimulus/response combinations is that they may interact in critical ways that cannot be characterized via the analysis of a single response or stimulus combination. Indeed, this is the case for motor inhibition. Rather than being defined solely by how IOR is generated or how it is measured, the occurrence of a motor bias depends on making a saccade to *either* S1 or S2.

INHIBITION OF RETURN

Experiments 1-6 of the present study represent a foray into 24 stimulus-response combinations that were expected to clarify the means by which oculomotor activation leads to a motor bias. Of course, the view that IOR is generated by oculomotor programming and measured as a motor response bias was based on the analysis of the 10 cells which had been tested previously in the literature. Examination of all 24 cells indicates that current conceptualizations of IOR must be re-considered (keeping in mind the possibility of S1/S2 stimulus/response interactions) and identifies several

points that are critical to this re-evaluation. First, the tendency to interchange manual and saccadic RTs as measures of IOR is not warranted. Second, isolated comparisons of S1-S2 response combinations must be viewed with caution, since it is only when patterns of IOR are examined across combinations that a picture regarding the nature of IOR emerges. And, third, the "either-or" debates that have abounded in the literature should clearly be superceded by the specification of the conditions under which contending views may hold.

The results of the present study suggest that IOR consists of two separable effects: 1) Slowed manual RTs to exogenous stimuli that appear in the same direction as a preceding exogenous or endogenous S1; and, 2) a bias against responding in the same direction as signalled by a preceding S1 when the saccadic system is engaged. The first form of inhibition - which has been referred to as visuo-motor - appears to be due to inhibition of exogenous covert attention. This inhibition for exogenous movements, in turn, appears to be generated by covert attentional operations associated with S1. The second form of inhibition - which has been referred to as motor - reflects a general motor response bias. This bias, in turn, requires execution of a saccadic response to S1 or S2. Provided that this condition is satisfied, then implicit or explicit activation of the manual or oculomotor response system is responsible for generating the IOR.

As has been alluded to, both proposed components of IOR are consistent with an SC basis of the inhibitory effect. Measuring the consequences of slowed exogenous orienting and slowed motor responding due to a deficient representation of target localization are consistent with SC mechanisms. However, the present discussion has judiciously avoided advocating a strong position on the neural processes underlying the present

pattern of results. This is because such a position would require considerable speculation with regard to the means by which various S1/S2 stimulus/response combinations generate and access a neural representation of inhibition. For example, there is no basis upon which to identify a role of the SC in the initiation of endogenous orienting (via the onset of an uninformative arrow). And there is a need to further specify the means by which executing a saccade to S1 has the same implications for measuring IOR as executing a saccade to S2. Even if represented within the SC, the mediation of IOR by attentional and oculomotor factors suggests that IOR may be better characterized within the context of an orienting system rather than in the context of a neural structure.

The challenge for future research is to determine whether the proposed dual-process model of IOR withstands further empirical scrutiny. And although such specification is premature at this point, the eventual development of a plausible neural model of the present findings would help develop and constrain theories regarding the generation and measurement of IOR.

FOOTNOTES

1 Note that RT facilitation versus inhibition of peripheral targets is determined by comparing cued versus uncued locations rather than by comparing these locations independently to the uncued centre location. The reason for this is clear: By virtue of occurring at the location where the receptor density is greatest and where attentional resources were actively focused, foveal targets were responded to faster than the peripheral targets - regardless of the spatial relation between cue and target. That the RT effects for the peripheral targets do represent facilitation of the cued location at short SOAs and inhibition of the cued location at long SOAs is indicated by a subsequent study (Posner & Cohen, 1984) that presented targets with equal probability at four peripheral locations to the left and right as well as above and below fixation. At short SOAs, RTs were faster at the cued location than at any other; at long SOAs RTs were slowest at the cued location than at any other.

2 There is some debate regarding whether a visual transient is either necessary (e.g., Yantis & Hillstrom, 1994) or sufficient (e.g., Folk, Remington, & Johnston, 1992; and Yantis', 1993, rebuttal) for exogenous orienting. Although exogenous cues may not capture attention in a manner inconsistent with subjects' intentions (Yantis & Jones, 1991; Yantis & Jonides, 1990), in the absence of strong endogenous control, a visual transient is capable of facilitating performance at a cued location independent of the predictive validity of the cue (Theewes, 1991).

3 Chronometric analysis of spatial cueing effects sometimes compares cued and uncued target RTs to a neutral condition in which attention is spatially dispersed. Relative to the indicated costs+benefits measure of attentional allocation, the use of a neutral cue allows for the relative parsing

of RT benefits due to orienting to a valid cue and RT costs due to orienting to an invalid cue. The validity of obtained costs and benefits clearly depends on the representativeness of the neutral condition as a true baseline (see Jonides & Mack, 1984 for cautions regarding the selection of an appropriate neutral condition).

4 Luminance summation typically results in similar functions for forward and backward masking (see Kahneman, 1968 for a review). The implication is that if the early facilitatory effects in an orienting paradigm are due to luminance summation, cues should be equivalently effective at negative SOAs (i.e., when the target precedes the cue) as at positive SOAs.

5 Although the arguments are made for visual stimuli, it should be noted that visual, auditory, and somatosensory output all converge on the SC (e.g., Stein & Meredith, 1990; Wallace, Meredith, & Stein, 1993) so that an SC basis of IOR does not prohibit finding the effect in other than a visual modality.

6 Abrams and Dobkin (1995) did not frame their study in terms of visuo-motor versus motor contributions. Instead, they spoke of visual versus motor contributions to IOR. The essence of their rationale is the same as that outlined here. The decision to avoid their use of the word "visual" is based on the outlined evidence which denies visual or perceptual bases of IOR. Although it remains possible that their so-called visual contribution does represent an effect at the visual receptors or in the perceptual analysis of the target, it is presumed on the basis of the weight of evidence that what they were measuring was, in essence, a visuo-motor as opposed to a strictly visual effect.

7 In exploring the spatial coding of IOR measured by exogenous and endogenous saccades, Abrams and Dobkin (1995) revealed the possibility that

the two may be dissociable. Following on the work of Tipper et al. (1991) who showed evidence of object-centred IOR, Abrams and Dobkin (1995) examined visual and motor effects in dynamic displays. They repeated the described methods, except that stimulus boxes started at locations above and below fixation and rotated clockwise through 90 degrees during the 300 msec presentation of the cue at fixation. In separate experiments, the saccade command was a exogenous visual signal or an endogenous motor signal. When the command was exogenous, IOR occurred for targets that appeared in the previously-cued stimulus box; when the command was endogenous only, there was no evidence of object-based IOR. This dissociability of visual and motor contributions is theoretically important. However, in light of the majority of studies that have used static displays and therefore confounded space-based and object-based effects, the ability to dissociate the effects is not paramount to the present analysis.

8 Note that the automatic orienting effects of an arrow cue are presumed to be the result of endogenous attentional mechanisms. Although the automaticity of the effect could be used to argue that the arrow engages exogenous attentional resources, there are two reasons why this view is not favoured - one is based on semantics, the other is based on the quest to interpret the data in a consistent manner. On the issue of semantics, the operational definition provided for exogenous orienting (see the Introduction) is that exogenous attention is captured by stimulation of transient visual channels. By this definition, orienting to a peripheral location based on a central arrow cue is not exogenous. Beyond this semantic issue, automatic orienting of attention in accordance with the direction signalled by an unpredictive arrow cue is unlikely to be accomplished by the same mechanisms that orient attention to the location of a peripheral onset

cue. If they were, then the No Response-Manual and Manual-Manual conditions should have revealed IOR to endogenous as well as to exogenous S2s.

9 Although the view that attention cannot be split over non-adjacent spatial locations is generally held, the only studies to have explored this hypothesis have used endogenous cues (cf. Kiefer & Siple, 1987; McCormick & Klein, 1990; Posner, Snyder, & Davidson, 1980; although see Castiello & Umilta, 1987 and McCormick, Klein & Johnston's, in press, challenge to their data).

APPENDIX A

Below is a summary table of the analyses presented in the body of the text. Significant effects are marked with "*" and non-significant effects are marked with "-". Note that empty columns exist for studies where the measure in question was not applicable. In the abbreviated captions, N=No Response, M=Manual, S=Saccadic.

	S1/S2 Response Combinations					
	N-M	M-M	S-M	N-S	M-S	S-S
Average Errors						
S1	-	-	-	-	-	-
S2	*	*	*	*	*	*
Relative Locations	-	-	-	-	-	-
S1xS2	-	-	-	-	-	-
S1xRelative Locations	-	-	-	-	-	-
S2xRelative Locations	-	-	-	-	*	-
S1xS2xRelative Locations	-	-	-	-	-	-
RTs to S1						
S1		*	-		*	-
S2		-	-		-	-
Relative Locations		-	-		-	-
S1xS2		-	-		-	*
S1xRelative Locations		-	-		-	-
S2xRelative Locations		-	-		-	-
S1xS2xRelative Locations		-	-		-	-
RTs to S2						
S1	-	-	*	-	-	*
S2	*	*	*	*	*	*
Relative Locations	*	*	*	-	*	*
S1xS2	-	*	-	-	-	-
S1xRelative Locations	-	-	-	-	-	-
S2xRelative Locations	*	*	*	-	-	-
S1xS2xRelative Locations	-	*	-	-	-	-
IOR vs RT to S1						
Exo-Exo		-	-		-	-
End-Exo		*	-		-	-
Exo-End		-	-		-	-
End-End		-	-		-	-
IOR vs Block Number						
Exo-Exo	-	-	-	-	-	-
End-Exo	-	-	-	-	-	-
Exo-End	-	-	-	-	-	-
End-End	-	-	-	-	-	-

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IMAGE EVALUATION
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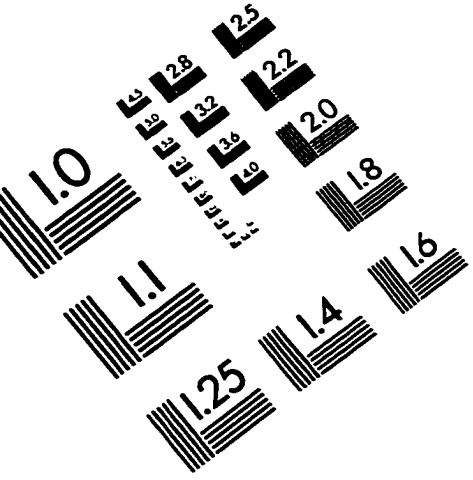

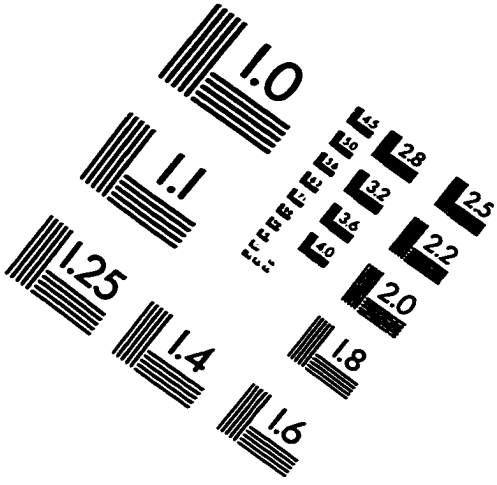

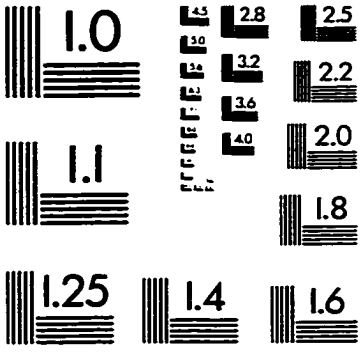

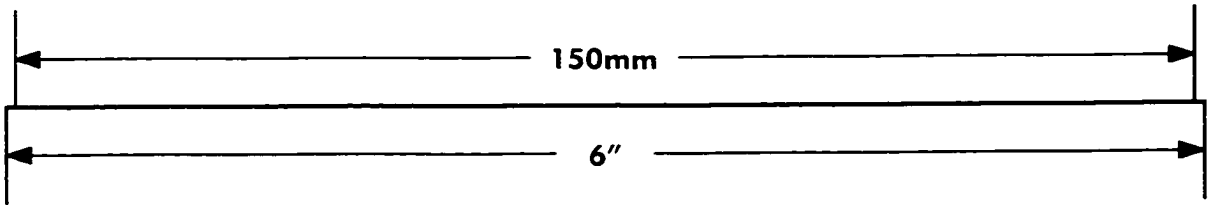

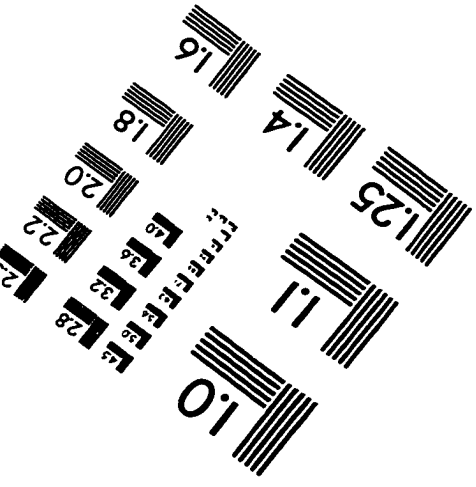

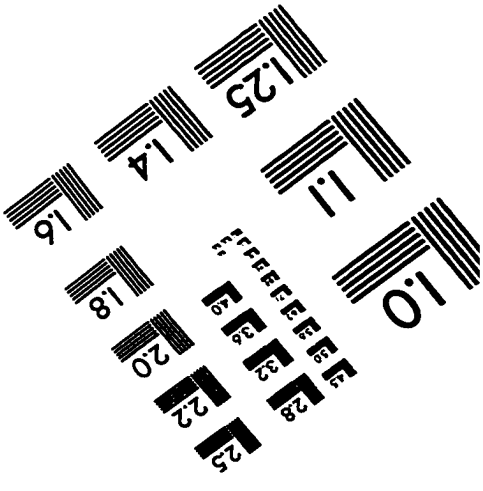

APPLIED IMAGE, Inc
1653 East Main Street
Rochester, NY 14609 USA
Phone: 716/482-0300
Fax: 716/288-5989