Visual Attention with implications for Unilateral Spatial Neglect.

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Statement of Copyright.

Declaration.

The work contained in this thesis was carried out by the author between 1989 and 1992 while a postgraduate student in the Department of Psychology at the University of Durham. None of the work contained in this thesis has been submitted in candidature for any other degree.

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Abstract.

Recent models of visual attention (eg. Rizzolatti et al., 1987) have suggested that a similar system orients visual attention as is used to produce a saccadic eye movement. This thesis provides further support for the link between the attentional and eye orienting systems and has incorporated ideas from recent models of saccade generation.

The time taken by normal subjects to initiate a saccade ('latency'), is examined in Chapters two, three and four. Subjects were given attentional instructions and saccades made to either: unilateral single, or, bilateral double, targets. Latency to attended targets was not greatly enhanced, while latency to non-attended targets was greatly slowed. The results support both the premotor model of visual attention and models of visual attention that emphasise the inhibitory consequence of directed attention. Bilateral double targets produced an additional slowing on saccade latency, which could reflect a further automatic attentional inhibition produced in the contralateral field by the stimulus onset.

Fixation point offset (in 'gap' situations) is known to reduce saccade latency, which has been attributed to prior attentional disengagement (Fischer, 1987). In Chapter two, the use of a gap situation produced a generalised speeding which was independent of the effects of directed visual attention. This suggests that active fixation affects a separate component to that involved in orienting visual attention to a spatial location. This idea is incorporated into a model which emphasises the inhibitory consequences of attentive fixation.

Chapters six and seven report the findings from an experimental investigation of a patient (B.Q.) with a 'unilateral spatial neglect', a condition often attributed to a deficit of visual attention. The 'gap' paradigm was shown to be effective at reducing the severity of B.Q.'s contralateral neglect. In contrast to normal subjects, bilateral double targets did not have an inhibitory effect on her saccade latency. These findings are explained in terms of a model that neglect results in part from the loss of attentional inhibition for the ipsilesional side of space and in part an inability to switch off contralesional inhibition produced during active fixation.

A functional model is proposed in Chapter eight to account for the findings. This supports the close link between the attentional orienting and saccade programming systems. An additional implication of the findings is that models of visual attention and saccade generation need to consider the inhibitory consequences of directing attention to a spatial location.

Dedication

To Mum and Dad with love and thanks.

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Love and thanks to Clapp

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Chapter 1
Introduction

1.1 Background.

The term attention when used in its broadest sense is often regarded as being synonymous with concentration, however attention is also commonly used to refer to the process which selects part of an incoming stimulation for further processing. For example William James (1890) described attention in the following way:

"Everyone knows what attention is. It is the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. Focalisation, concentration, of consciousness are of its essence. It implies withdrawal from some things in order to deal effectively with others" (pp 403-404).

The need for such a selection process arises as the human cognitive system is thought to be of limited capacity and could not process all of the information in the optical array simultaneously. As the human cognitive system is thought to have a limited capacity, psychologists have suggested that attention serves to cut down the amount of stimuli that enter the processing system. A great deal of work has been carried out on the stage at which the selection process occurs (Broadbent, 1958; 1982; Deutsch and Deutsch, 1963; Treisman, 1964). *Visual attention* is the area examined in this thesis and refers to the processes which are thought to select certain regions of the visual environment for more efficient processing, at the expense of less efficient processing for other locations. Visual perception operates within the space viewed by the observer, so visual attention as a process which facilitates visual perception can be thought to have spatial properties. An alternative suggestion is that we do not attend to regions of space, but to the objects located within the space (Duncan, 1984). The idea of object based allocation attention has also been incorporated in another view of visual attention. Assuming that the representation of the visual scene is mapped at coarse and fine levels, visual attention could operate by activating object representations (icons) at a specific spatial scale (Nakayama, 1989).

Attention can be thought of as enhancing detection of stimuli within a specific region of space and can also play a role in grouping features as part of object recognition. Treisman's feature integration theory (Treisman and Gelade, 1980; Treisman, 1988) provides a model of the role of attention in object perception. According to this theory the early stages of visual perception involve parallel processing of the basic features of the stimulus such as; colour, orientation, size etc. The stimulus features are coded in separate 'feature maps', and these individual features can be combined or integrated into an individual percept by attentional processes. The features can be integrated by attending to the location of the object, and also by the stored knowledge of the characteristics of certain objects that would group together features that are known to go with a specific object. According to feature integration theory attention is applied serially to specific locations to combine correctly all of the features coded in the maps.

This thesis is concerned with the spatial account of visual attention. The spatial characteristics of attention were studied by an experimental examination of the link between saccadic eye movements and movements of visual attention, in normal subjects. The link between the eye movement and attentional system was examined by measuring the effect of directing visual attention on the time taken to make a saccade ('saccade latency') to a peripheral target. The second part of the thesis studied a patient with a disorder, which is commonly thought of as resulting from a deficit to the visual attentional orienting system ('unilateral spatial neglect'). The patients ability to report and orient her eyes to stimuli was examined under conditions which manipulated visual attention. The final section of the thesis proposes a model of the attentional/eye orienting system, which can account for the experimental results with normal subjects and the results obtained with the patient.

Shifts of visual attention in normal viewing situations are closely linked to movements of the eyes. An eye movement is required in normal viewing conditions to bring the attended stimulus (or location) onto the foveal region of the retina, thus enabling detailed processing to take place. It has been shown experimentally that it is also possible for a person to shift their attention 'covertly' to a stimulus location in the absence of an 'overt' eye movement (Posner, 1980). If a person is attending to a peripheral location without having moved their eyes to that location then it can be shown that a novel event occurring at that location will be responded to more efficiently than an event at a non-attended location (Posner et al., 1978; Posner, 1980). The evidence for an attentional selection system which can move independently of the eyes has been provided by measuring subjects performance on a variety of different tasks; manual reaction time tasks, saccadic reaction time experiments, target discrimination tasks and visual search. These different techniques have made use of conditions which are thought to alter the orienting of attention, so as to produce changes in performance measures.

Posner and Petersen (1990) emphasised the idea that the attentional system is separate from the system involved in the information processing of the stimulus event, and went on to divide the attentional system into three separate subsystems. These are the orienting component, detection component and vigilance component, each one being thought of as having has a different cognitive function, and involving a different anatomical substrate. For vision the attentional orienting component is thought to involve the selection of a particular region to enable a more detailed analysis of stimuli at this location, at the expense of less detailed processing for other regions. The detection component is a more general component which serves to enable conscious processing of the sensory signal as well as information stored in memory. The third component is involved in maintaining a vigilance or alert state, which affects the rate at which attention can respond to a stimulus. This distinction between the different attentional sub-systems is an important one, however it is the nature of the orienting component in visual attention that will be the primary concern in the following discussion.

One of the most widely quoted illustrations of a covert shift in attention is from Posner et al.'s (1978) manual reaction time experiment. In this experiment an arrow cue at the point of fixation, was used to direct the subjects attention covertly, to a spatial location, while their eyes

remain at the central fixation point. The provision of such a cue improved performance, indicated by faster reaction times for targets located at the **valid** (cued location) termed **benefits**, and produced slower reaction times for targets appearing at the **invalid** (non cued location) termed **costs**. The size of the costs and benefits obtained were defined with respect to RT's obtained in a 'neutral' condition, in which there was no cue at all. The improvement in performance is thus attributed to the facilitative effect of visual attention for targets at the cued location, and costs are attributed to the time taken to move attention from the cued location to the non cued location.

Posner et al. (1984), suggested that orienting attention in itself involves three separate processes. Initially attention must be 'disengaged' from the current attentional fixation, then it can be 'moved' to a new location, before being 'engaged on the stimuli of interest at the new location. These processes are all involved in the basic cued reaction time experiment described above. In the neutral condition, attention must first be disengaged, then moved to the stimulus location following target onset and then engaged on the novel stimulus. The advantage with valid targets in the cued condition is termed a 'benefit' on RT performance and is thought to reflect the prior disengagement and movement of attention to the target location. The increase in RT's for invalid targets compared to the neutral condition is called a 'cost'. The cost is thought to reflects the extra time required in addition to the disengagement and engagement processes, for attention to move from the cued location, to the opposite (invalid), location. This analysis of costs and benefits in terms of comparing cued performance with a neutral condition is controversial as the neutral cue condition could also affect the performance measure (Hughes and Zimba, 1987).

Experiments of attentional orienting typically use a cue which indicates the location that attention should be oriented towards. There are two different cueing procedures which are used to direct attention, termed symbolic and peripheral cueing. **Symbolic** (or central) cueing involves the use a symbol (arrow, number etc.), located centrally at fixation while **peripheral** cueing involves presenting a sensory flash coincident with the target location. The use of these two cues has been shown to have different effects on the performance measures which has lead to the suggestion that they operate on different attentional orienting systems (Jonides, 1981; Müller and Rabbitt, 1989; Nakayama and Mackeben, 1989; Posner, 1980). Subjects have internal control over the spatial allocation of attention when a symbolic cue is used so this is thought to be controlled by the **voluntary** orienting system, an abrupt peripheral onset produces a reflexive allocation of attention which is thought to involve the **automatic** orienting component. The evidence supporting the idea of two components of attentional orienting is discussed in section [1.2].

The idea of visual attention moving in space enhancing stimuli processing for contiguous regions of the field, has lead to the metaphor of a 'spotlight' being applied to describe the attentional movement (James, 1890; Posner, 1980). The premise is that visual attention can move across the visual array enhancing cognitive processing for stimuli falling within its beam in much the same way that a spotlight beam illuminates the surrounding world.

Two variations on the spotlight theme have been suggested to account for attentional orienting, these are the zoomlens model (Eriksen and St. James, 1986), and the gradient model (Downing and Pinker, 1985). Some of the evidence supporting these models and a description of the differences between, them is discussed in relation with results that are thought to be incompatible with these metaphors, in section [1.3].

An alternative to the idea of attention acting as a spotlight enhancing the processing of attended areas is provided in the inhibition model of Hughes and Zimba (1985; 1987) and Zimba and Hughes (1987). They suggested that directing visual attention to one location serves to produce broad areas of inhibition for other non-attended spatial locations. The spatial distribution of the inhibition is governed by the summation of two broad areas of inhibition, one for the left and right hemifields and one for the upper and lower hemifields. According to this view directing attention to one location will produce inhibition for the area of space in the opposite quadrant, attention being spatially restricted by the horizontal and vertical meridians. According to this model directing attention will not produce a facilitatory effect for the attended area. The predictions from the hemifield inhibition model and the spotlight models, are very different in terms of the cost/benefit analysis of target detection for stimuli presented at attended and non-attended spatial locations. The experimental evidence supporting and refuting these different theories will be discussed in section [1.4].

Under normal viewing conditions movements of the eyes and movements of attention are usually coincident. A fast, ballistic, eye movement, called a 'saccade' is responsible for moving the eye, so as to direct the fovea onto the target. The 'premotor' model of visual attention emphasises the link between saccadic eye movement and attentional orienting systems (Rizzolatti et al., 1987; Tassinari et al., 1987). Rizzolatti et al. (1987) proposed that the same neural mechanisms that control a saccadic eye movement could also control covert movements of visual attention. Covert orienting of attention involves the programming of the corresponding saccadic eye movement, but the final execution of the saccade is suppressed. Rizzolatti et al.'s premotor model stated that the features of the motor response are specified independently. When a cue has been presented, the motor programme containing both the amplitude and direction of the required saccade are programmed separately. The motor response to a stimulus appearing at the cued location is therefore facilitated, compared to responses made to a stimulus at a non cued location which require some aspects of the motor programme to be altered. The premotor model is described in section (1.5).

Shepherd, Findlay and Hockey (1986) examined the relationship between saccadic eye movements and visual attention by using a reaction time experiment in which attention was cued by target expectancy, while a central arrow cue indicated the direction that subjects should prepare to make an eye movement. The results showed that preparing to make an eye movement to a location speeded RT's to that position, and directing attention to a location speeded an eye movement. They also showed that it is not possible for an eye movement to be prepared in one direction and attention to be moved in another, implicating the role of the saccadic system in the attentional orienting system.

The link between attention and saccadic eye movements has been supported in studies of saccade latency, which have shown that under certain attentional manipulations saccades with short latencies are produced. Saslow (1967) showed that saccade latency is reduced by prior fixation point offset before the saccade target appears ('Gap paradigm'). Fischer and Breitmeyer (1987) performed experiments using the gap paradigm in which observers were required to attend to a peripheral stimulus (attentional stimulus), whilst keeping their eyes on a central fixation point, observers moved their eyes from fixation to a target stimulus when it appeared at an eccentric location. Saccade latency was shown to be reduced if the attentional target was turned off, before the target stimulus appeared. Under these conditions saccades of extremely short latencies termed 'express saccades' were produced, which Fischer proposed are due to the attentional system being in a disengaged state prior to target onset, enabling an eye movement to be made in less time than would normally be required. The gap paradigm and its effects on saccade latency are discussed in section [1.6].

1.2 Two components of visual attention.

Experiments on visual attention have typically used symbolic cues at fixation (central cues), or sensory peripheral flashes at the target locations (peripheral cues), to orient attention. Central cues such as arrows, or numbers, involve higher level cognitive process to interpret the cue, to enable attention to be voluntarily directed to that location. This attentional system has been termed the 'Endogenous' (Posner, 1980), 'Sustained' (Nakayama and Mackeben, 1989), or 'Voluntary' (Müller and Rabbitt, 1989) attentional component, to reflect that it is a voluntary system, under the control of central cognitive processes. Peripheral cueing, using a sensory flash coincident with the target location, has been shown to automatically summon attention to that location. The automatic orienting of attention also overrides the voluntary orienting by central cues (Müller and Rabbitt, 1989). This system has been termed the 'Exogenous' (Posner, 1980), 'Transient' (Nakayama and Mackeben, 1989), or 'Reflexive' (Müller and Rabbitt, 1989), attentional component; indicating that it produces an automatic summoning of attention which is outside cognitive control. The voluntary and automatic components of attention are not thought of as two completely separate mechanisms of attentional orienting, but are considered to be two sub-components of a single attentional orienting system. The evidence to support these claims comes from the effects the two cue types have on measures of visual attention, over different time course.

Müller and Rabbitt (1989) examined the 'reflexive' and 'voluntary' components of visual attention, in a target discrimination task. Subjects were required to indicate if a target was the same as, or different from, a comparator stimulus and to indicate both the target and cued locations. Peripheral and central cues were used over a range of 'stimulus onset asynchronies' (SOA's), to examine the time course of the two orienting components. The performance measure was the subject's probability of making a correct same/different response and a correct position response, as a function of SOA. Peripheral cues produced a faster initial rise in performance with valid cues, with a broad peak at 175 ms SOA. A slow decrease in performance

occurred with SOA's up to 400 ms and thereafter performance remained at a fairly constant level. The central arrow cues produced a gradual slow rise in performance with SOA's up to 400 ms which then remained at a constant level. Invalid peripheral cues were found to produce a greater decrement in performance than did invalid central cues. When attention was oriented to a cued location with central, or peripheral cues, a second irrelevant peripheral flash was found to decrease performance. This implies that attention is automatically summoned by a peripheral flash, even when the subject is aware that attending to the flashes will be detrimental to task performance. These results are taken to support the idea of a fast acting reflexive orienting component which is not under cognitive control and a slower acting voluntary orienting component which will be interrupted by the reflexive component. Müller and Rabbitt suggested that these two components of attention could both operate on the same limited capacity attentional orienting system, but could have different underlying neural locations. Posner and Cohen (1984) suggested that the reflexive component is linked to the mechanisms which control saccadic eye movements, while the voluntary orienting system could involve cortical control particularly the parietal lobe, which is an area often linked with spatial orienting.

Shepherd and Müller (1989) used central and peripheral cues, over short and long cue to target (SOA) intervals, to examine the spatial distribution of attention. With central cues at short SOA's, the benefits on RT performance were equal for both near and far target locations, but with longer SOA's the facilitatory effect was restricted to the cued location. With peripheral cues a strong facilitation effect was present at the earliest SOA's and was spatially restricted to the cued location. This result shows that peripheral cues are more effective than central cues at focusing attention which could be due to the peripheral cues having a faster effect at narrowing an attentional beam to the cued location. The data is used to support a model of attention with an initially broad focus which is narrowed to focus on the cued location.

Todd and Van Gelder (1979) performed an experiment which indicated the different effects that the reflexive and voluntary orienting systems have on saccadic reaction times. Saccade targets were either the onset of a target (onset) or the offset of all but one distractor targets, which left a single target without the sensory transient change (no-onset). They showed that an abrupt target onset produces much faster saccadic RT's than does a procedure in which there is no abrupt onset. This is consistent with known differences between transient and sustained retinal ganglion cells. The transient cells are fast conducting and stimulated by abrupt transient changes, whereas sustained cells have slow conducting velocities and are stimulated by stationary stimuli (Breitmeyer and Ganz, 1976). Todd and Van Gelder suggested that an abrupt target onset will activate the transient visual channels which project to the superior colliculus to give a map of the target location. Stimuli in the no onset condition will not activate the transient visual system, so the eye movement must be activated by the sustained system only. A further interesting result was that if the number of distractor stimuli was increased in the no onset condition, saccade latency was decreased. This result is counter intuitive as it would be thought that increasing numbers of distractor stimuli would give a decrease in performance, as there are more possible target locations for the saccade. Todd and Van Gelder further suggested that the offset of a stimulus has a general non specific facilitative affect on

performance, possibly due to increasing arousal. However this result is also consistent with Nakayama and Mackeben's (1989) idea that the offset of stimulus activates a transient attentional channel, which automatically orients attention to that location. It is possible that the greater the number of offsets there are, produces a greater activation to the transient attentional system, which accounts for the increase in the facilitation of saccade latency with increasing numbers of offset stimuli.

1.3 Spotlight and Zoomlens accounts of visual attention.

Movements of visual attention have been compared to those of a 'spotlight' which moves across visual space, enhancing target detection if the beam is coincident with target location. The spotlight metaphor used to account for the orienting of attention is a convenient one and has become one of the most popular in accounting for a wide variety of experimental results. Posner (1980), used the term 'orienting' to mean the aligning of attention with an input which enables a second cognitive process of 'detection' to take place. The spotlight metaphor implies that attention moves to a peripheral location and improves the processing of a stimulus at that location. Recent studies have been concerned with examining how the attentional spotlight moves in space, to show if the movement is smooth (analogue), or a jump from one location to another. Other work has examined the size of the spotlight beam and also examined if the beam size is fixed, or variable. There is also an increasing amount of work which is thought to be incompatible with the spotlight metaphor. The issues of how the spotlight could move in space, the size of the spotlight beam and the evidence that questions the spotlight analogy are described in this section.

1.3.1 The movement of the attentional 'spotlight'.

Posner (1980) interpreted the results of his manual reaction time experiments (eg Posner et al.,1978) in terms of the movement of the attentional spotlight. In their basic experiment a central arrow cue was used to direct attention to one of a number of boxes on the horizontal axis, in which the target stimulus could appear. The cue indicated the target location on 80% of trials, while on the remaining trials the target appeared in one of the non-cued locations. A cued target is termed a 'valid' target, whilst a non-cued target is termed an 'invalid ' target. Performance was compared with RT's made in a 'neutral' condition in which a cue indicated that the target could occur with equal probability at any location. Reaction times were shown to be fastest in valid trials and slowest in the invalid trials, with neutral trials producing intermediate reaction times. This result is interpreted in terms of attention having moved to the cued location, thus producing the fastest reaction times. On invalid trials attention will have to move from the cued location to the non-cued (invalid), location a process that will take time and produce a slowing of RT's.

An experiment performed by Tsal (1983) enabled an estimation of the speed that the spotlight moves across space to be estimated. Tsal used a vocal reaction time experiment, with valid cues for targets at three eccentricities. An examination of the point at which the benefit on

reaction times became constant (asymptotic) was examined for the three eccentricities. The theory being that after a certain length of time (SOA) attention will have moved from fixation to the cued location so performance will not improve further with more time. A comparison of the SOA's at which there was no further improvement on RT performance, for each target eccentricity revealed that attention does appear to move across space in an analogue way, at a speed of 1 degree every 8 ms.

If shifts in visual attention occur in an analogue way, comparable to a spotlight beam moving across real space, then the movement of attention would require a certain amount of time to travel a particular distance. Attention would also have to pass intermediate spatial locations before reaching the final cued location. This dependence on time taken is referred to as a 'time locked' movement of attention (Posner, 1980). The analogue movement of attention is supported by several results from cueing experiments. Shulman, Remington and McLean (1979) used a central cue, to covertly orient attention to an 18 degree location, at which the target appeared on 70% of trials. On the remaining trials ('invalid'), the target occurred at an intermediate position on the cued side, or on the side opposite to the cue. The time course of the shift of attention was examined by using different SOA intervals, from cue to target onset. A subtraction of reaction times to the near (unexpected) and far (cued) target locations showed that the near location advantage is small with short SOA's of up to 100 ms and increases to a maximum with SOA's of 200 ms, before falling off with longer SOA's of 300-500 ms The interpretation is that visual attention has moved to the intermediate (near), location by 150 ms, producing fast RT's for near targets. At longer SOA's visual attention is assumed to have moved on towards the cued (far), location so that there is no near target detection advantage as attention will now be aligned with the far location. Humphreys and Bruce (1989) questioned the interpretation of Shulman's results as being compatible with the spotlight metaphor. Shulman's data shows evidence of a facilitation for the far cued location at SOA's of 200 ms, when the spotlight is assumed to be aligned with the near cued location. If the spotlight was, as suggested, aligned with the near cued location by 200 ms, then it is difficult to explain the benefits shown for the far location using the idea of a spotlight with a fixed beam size.

Recent work by Shepherd and Müller (1989) has compared predictions from the analogue attentional movement, and focusing of attention, models, and obtained data which undermine the analogue movement of attention. Subjects were cued using arrow, or peripheral cues, in a covert orienting RT experiment. Targets appeared at near and far locations on both side of fixation, following SOA's of 50, 150, 200 and 500 ms. The aim was to compare the benefits obtained when comparing the specific cued location to the uncued location in the same field, to a neutral cue condition. Cuing the near location with an arrow cue, produced equal benefits for both the near and far locations at short SOA's. As SOA increased the benefit for the far location decreased and finally became a small cost (at 500 ms SOA). When the far location was cued a comparable pattern of results was obtained, with equal benefits for near and far locations at short SOA's, and a decline in the benefit with increasing SOA for the near target. Large costs were shown for near and far targets in the field opposite to the cue. The equal benefits obtained for near and far locations at short SOA's is incompatible with an analogue

movement explanation, which predicts a greater benefit for the near location as the spotlight moves across space. The decrease in benefits for the non cued location is compatible with a focusing explanation. Initially attention is broadly distributed covering both the near, and far locations producing equal benefits. As SOA increases the beam becomes narrower around the cued location, this produces a decline in performance for the non cued location. When peripheral cues were used there was a benefit for the cued location only at short SOA's which declined as SOA increased. This is compatible with the idea that the attentional beam is focused faster with peripheral cues, than arrow cues, and that attention is not maintained at the cued location for a long time interval. The idea of attention being comparable to a spotlight beam of fixed diameter, is not supported by the finding of equal benefits for the near and far target locations, which were some 10 degrees apart. The analogue movement idea is not supported, as the intermediate near location should be facilitated at short SOA's as the beam moves across space. The proposal that the beam moves across space with a constant velocity (Shulman et al., 1979; Tsal, 1983) is not supported as there were no effects of target eccentricity on performance. A focusing of attention model with an initial broad beam which is narrowed following the cue can explain most of the obtained results.

1.3.2 The spatial distribution of the attentional 'spotlight'.

An alternative to the idea that attention is evenly distributed across a relatively small area (spotlight) has been the suggestion that attention is more concentrated at the fovea and decreases towards peripheral locations. Downing and Pinker (1985) used a cued reaction time experiment to examine the distribution of attention across visual space. They interpreted their results as showing that attention is unevenly distributed at different retinal locations, implying that there is a gradient of the distribution of attention. In their (second), experiment a central number cue was used to indicate which of ten horizontally displayed boxes the subject had to attend to. Costs and benefits were plotted (cued reaction time minus neutral reaction time), for each of the ten stimulus positions. Attentional facilitation was shown to decrease, with increasing retinal eccentricity. The rate at which the facilitation decreased was different for different retinal locations, as the slope of the curve fell off most steeply for cued locations near to the fovea, while a more gradual decline was shown for peripherally cued locations. The greatest benefits were for cued foveal locations, than for peripheral cued locations. These results are consistent with the attentional spotlight analogy, if it is expanded to incorporate the idea that the beam can be narrowed to a greater extent at the fovea than in the periphery. Visual attention falls off more rapidly for regions with a greater cortical representation and finer resolution than for regions where the resolution is coarse.

The size of the spotlight beam has been estimated to be of a fixed size of about 1 degree, by Eriksen and Eriksen (1974), who used a choice reaction time task where subjects had to decide which target letter was present on a trial. The target letters were presented along with distracting letters, which were either from the same response category as the targets or from a different category to the targets. Distracting letters were located spatially close to the target (less than 1 degree) or further away over 1 degree. The slowest reaction times were

obtained when the distracting letters were from the opposite response category to the targets. However this slowing was only apparent if the distractors were located close to the target letters, within an area of approximately 1 degree visual angle. The conclusion which Eriksen and Eriksen (1974) reached was that the spotlight beam is of a fixed size of about 1 degree.

Later work by Eriksen and St.James (1986), used a similar task to suggest a variation on the spotlight model, to incorporate the idea of an adjustable beam size. This variation suggests that visual attention operates like a 'zoomlens' with a variable beam size, attention initially being widely distributed and then focused at the target location (Eriksen and Yeh, 1985; Eriksen and St.James, 1986). The zoomlens analogy proposes that the spatial extent of the beam can be made to vary, and as the beam size gets larger there is a decrease in the processing efficiency of stimuli falling within the beam. Eriksen and St. James (1986) provided experimental evidence which supports the zoomlens account. Subjects were required to discriminate between the target letters S and C as quickly as possible. The targets were presented in circular arrangement along with seven distractor elements, following a precue (underlining of letter position). The manipulations included, the SOA between precue and display onset, the number of adjacent locations that were cued (1, 2, or 3), the presence of 'response competitive' distractor letters and the distance of these distractors from the target (1, 2, 3, spaces from the target). The response-competitive distractors were either the same letter as the target letter ('compatible') or the other possible target letter ('incompatible'). The important manipulations for the zoomlens account involved the effect of the incompatible distracting letters (at the three locations), following one, two or three precues, over increasing SOA's. The zoomlens account would predict that the size of the attentional distribution will be larger with more precues and narrow with only one precue. The zoomlens being thought to be set initially to cover the whole of the display and is narrowed after a certain time, following the precues. There will be more interference with incompatible distractors 2/3 positions away from the target at short SOA's when the beam is broadly distributed, than in a single cue condition following a long SOA in which the zoomlens has been narrowly focused.

The results obtained by Eriksen and St.James (1986) confirmed these predictions. The greatest slowing was shown for an incompatible letter one position from the target and the least when it was three positions away. Importantly, performance was equally impaired at low SOA's regardless of the spatial position of the distractor. Reaction time performance improves with increasing SOA, and the improvement was more pronounced for a distractor at three positions away than for distractors two or one position away. This is consistent with the idea that the zoomlens is narrowed after the longer SOA's, so distractors three positions away have much less effect on performance than do distractors that are closer. Reaction time performance increased as the number of cued positions increased, this is consistent with the notion that as the size of the attentional field increases the amount of processing resources within the field decreases. However the increase in RT's is also consistent with the idea that cueing more locations makes the task of discrimination more difficult.

Driver and Baylis (1989) offered an alternative explanation to the results of Eriksen and

Eriksen (1974) and Eriksen and St. James (1986) which questions both the spotlight and zoomlens interpretations. Driver and Baylis suggested that attention may in fact be assigned to perceptual groups on Gestalt principles of organisation. The finding of Eriksen and Eriksen (1974) that near distractors produce a greater slowing of RT's than far distractors could be interpreted either; as near distractors falling within the attentional spotlight, or alternatively; in terms of a grouping explanation. The grouping explanation is that the near distractors are grouped with the target letter, because of their spatial proximity, which will result in attention being allocated to this region. To dissociate between these two explanations of attentional allocation Driver and Baylis used a variation of Eriksen and Eriksen's (1974) experiment by using distracting letters that were near and far from the target letter and were grouped by using motion as a variable to effect grouping. Subjects were required to respond to a central letter which moved down the array along with two far distractors, while two near distractors remained stationary. The distractor targets were of two types; congruent (same category as target) and incongruent (letter assigned to incorrect response category), the incongruent distractors should produce the greatest slowing to RT performance. The spotlight explanation predicts more interference from the near incongruent distractors, while the grouping explanation predicts more slowing from the far incongruent distractors which will be grouped by the cue of apparent motion. Distant distractors that moved along with the target produced a greater slowing of RT's than did near targets that remained stationary. This result appears to support the grouping, but not the spotlight hypothesis of visual attention. A variation on this experiment was to move the near distractors while the target and far distractors remained stationary. The grouping hypothesis should again predict a greater slowing for the far distractors which will be grouped by virtue of their absence of movement. An Eriksen interference effect was again noted for incongruent distractors and the far distractors produced a greater slowing than the near distractors. These experiments show how the effect of proximity can be reversed by motion of distractors. This suggests that attention may be assigned to perceptual groups, rather than features that are spatially close together as the spotlight account would require.

To summarise this section the evidence reviewed shows how directing spatial attention to a spatial location has the effect of improving target detection at that location and also reduces performance for targets at non cued locations. These results have been interpreted as attention moving through space like a spotlight. The work of Eriksen (Eriksen and Eriksen, 1974; Eriksen and St. James, 1986) has refined this account to incorporate an adjustable beam size which results in the analogy of a zoomlens being applied. Driver and Baylis (1989) showed how stimuli located spatially further from the target can have a greater distracting effect if they are 'grouped' by common motion, than near stimuli not grouped to the target. This finding poses a problem for both the spotlight and zoomlens account of visual attention. The next section reviews further experimental evidence which indicates little advantage for directing attention to a specific location, with large costs for targets at non-attended locations. The lack of a benefit for directing attention to a spatial location has resulted in an alternative model of visual attention termed the 'Hemifield activation hypothesis' by Klein and McCormick (1989), but will be referred to here as the 'hemifield inhibition model' of attention based on the work of Hughes and Zimba (1985, 1987) and Zimba and Hughes (1987).

1.4 Hemifield inhibition model of visual attention.

Hughes and Zimba (1985) obtained results from a cued reaction time experiment which are not compatible with the spotlight account of visual attention. Attention was cued to a location either 2 or 6 degrees left or right of fixation, using a central arrow cue in an otherwise empty visual array. Occasional 'probe' flashes appeared at other locations within the cued and uncued hemifield. The reaction times to the probe stimuli were compared to stimuli appearing at the cued locations (expected) locations and also to reaction times in 'neutral' trials in which a left and right pointing arrow cue indicated that targets were equally likely to occur at both locations. A baseline measure of reaction times to targets appearing at each location without any precues was also used and showed the typical increase of reaction time with eccentricity. The results showed that reaction times to probes in the valid hemifield were equivalent to reaction times at the cued location. There was also little improvement in performance for targets in the cued hemifield compared to the neutral trials, indicating that there is no real benefit on RT performance for targets in the cued direction. A significant cost was obtained for targets and probes in the uncued hemifield, shown by equal reaction times for stimuli at all locations. Hughes and Zimba further considered the possibility that the failure to obtain significant benefits within the attended hemifield could be due to the difficulty observers may have had attending to an unmarked visual location. To examine this possibility they used a precue flash which appeared above the expected target location. On valid trials the target appeared below the precue location, on invalid trials it appeared 10 degrees away but in the same hemifield. Reaction times on valid trials, were shown to be a non significant 7 ms faster than on neutral trials which again shows a failure of directing attention to produce any benefits. If attention is focal (like a spotlight) then costs and benefits should be obtained within a hemifield. As there were no significant benefits on performance compared to a no cue condition the major effect of directing attention is thought to be inhibitory. Hughes and Zimba put forward the view that directing visual attention results in a modest benefit in the expected hemifield with a large cost produced by wide spread inhibition in the opposite hemifield. The inhibitory effect is thought to operate in terms of the visual half fields so the inhibitory effect would be apparent for movements of attention separated by the vertical meridian.

The experiments of Hughes and Zimba (1985) directed attention covertly along the horizontal meridian only. The conclusion being that the inhibitory effect of visual attention operates in terms of the visual half fields, which have distinct anatomical correlates and are represented separately in each hemisphere. However, it may also be possible to direct attention in the upper and lower hemifields, or in each of the visual quadrants, in which case attention could be distributed in terms of both the horizontal and vertical meridians. Hughes and Zimba (1987) examined this possibility by directing attention along the vertical axis and measuring RT's on valid, invalid and probe trials with stimuli located 6 degrees above and below central fixation. Probe targets could occur at locations both on and off the vertical meridian in the attended (upper) and unattended (lower) meridians. The attended and unattended 6 degree locations were 'marked' in this experiment by the presence of two small dots presented

on the display along with the central fixation point. The results replicated the findings of directing attention along the horizontal axis with a small (non significant) advantage for valid cued targets and a large (significant) cost for invalid targets. Probes in the attended hemifield on the vertical axis produced relatively uniform levels of performance. Probes located in the attended hemifield, but away from the attended vertical axis, produced a small non significant cost of some 4 ms, except for probes at 10 degrees from the vertical axis which produced a 17 ms slowing which reached significance. Large costs were obtained in the non-attended hemifield, for all invalid targets and probes. The greatest drop in performance was found for invalid targets at the marked location. These results are consistent with the idea that both the horizontal and vertical meridians serve to spatially restrict the extent of directed attention.

In a further experiment Hughes and Zimba (1987) examined the effects of directing attention along the oblique meridians. The results again showed small benefits for stimuli at all locations in the attended quadrant and large costs for stimuli in the hemifield diagonally opposite the cued quadrant, which require a movement of attention crossing over the horizontal and vertical meridian. Smaller costs were incurred to stimuli in the quadrant on the attended side (horizontal crossing), and in the opposite quadrant (vertical crossing). A further decrease in performance was shown for targets at the marked locations in the attended quadrant and to a greater extent for the marked location in the unattended quadrant, which is again interpreted as showing that the markers have a further inhibitory effect on target detection. The results of Hughes and Zimba are incompatible with the spotlight analogy of visual attention, as their cost-benefit analysis rarely showed any evidence of benefits of target detection on valid trials, which is in contrast to other findings such as: Shulman, Remington and McLean (1979); Posner (1980) etc. The interpretation of the large costs for stimuli in non-attended quadrants is that the effects of directing visual attention, is to produce two large area of inhibition for the left and right hemifields and the upper and lower hemifields which can operate jointly to produce inhibition in visual quadrants as well as half fields.

The failure of Hughes and Zimba (1985; 1987) to find any evidence of benefits on RT performance in valid trials is of interest for two reasons. The first is simply to know why other workers have found similar sized costs and benefits in comparable tasks. The second and more important consequence of the failure to find any benefits for targets located in the attended hemifield, is that it is incompatible with the spotlight analogy. Zimba and Hughes (1987) suggested that the main difference between experiments which do show benefits on RT performance and those that do not is the amount of structure contained in the visual display. Studies which have found benefits typically contain a structured display, such as marker boxes at the target location (Downing and Pinker, 1985; Posner et al., 1978; Posner and Cohen, 1984; Shulman et al., 1979), or distracting features in a discrimination task (Eriksen and Eriksen, 1974; Eriksen and St. James, 1987). Hughes and Zimba (1987) used only a small target location marker, and found little evidence of any benefits, interestingly performance was reduced in the attended hemifield for targets at the marked location. Zimba and Hughes (1987) compared the effects of directing attention to target locations, when the display contained small markers (0.37 degrees) and compared it to performance in a display without markers. Visual

attention was again oriented covertly using arrows and peripheral flashes as cues, to indicate likely target locations along the horizontal axis. A cost-benefit analysis comparing cued RT's (valid/invalid) to a neutral (both locations cued) condition. The results again showed the pattern of much greater costs in invalid trials than benefits obtained in valid trials. The presence of markers had the effect of increasing the costs obtained for invalid targets at each eccentricity. Adding markers in the visual display has the effect of elevating RT's at all eccentricities in the invalid direction. When the landmarkers are not present performance is constant across all eccentricities. If a cost benefit analysis is performed by subtracting the valid RT's from the invalid RT's at the same eccentricity, then the benefits obtained for targets in the attended hemifield will be greater when markers are used to indicate the target location. As RT's now increased with increasing distance from cue to target location and the benefits in the attended hemifield are significant the use of the markers has produced a pattern of results compatible with the spotlight analogy. Zimba and Hughes argue that this pattern is not due to the time taken for attention to move across the visual array, but is actually due to an inhibitory effect of the markers.

Zimba and Hughes (1987) second experiment aimed to examine the effects of directed visual attention, within the attended and non-attended hemifield, with and without markers. In this experiment the cue indicated that targets were likely to appear at 12 degree location, while probes appeared at eight intermediate locations in the attended and non-attended hemifields. The results again showed a small benefit of some 6 ms for valid trials, with much greater cost of some 30 ms for invalid trials. A general decrement in performance was apparent when the markers were present. A comparison of RT's obtained for targets and probes in the attended hemifield showed that there was little difference between the cued location RT's and probe RT's. This finding was apparent in the marked and unmarked condition and fails to support the spotlight hypothesis. Costs in the non-attended hemifield were also uniform for targets at the cued location and at the probe locations. A decrement in performance was shown in the marked condition for targets appearing at the 12 degree location in the non-attended hemifield, which confirms that the presence of the marker produces an inhibitory effect. This decrement is termed the 'landmark effect' by Zimba and Hughes. The results obtained failed to produce any costs within the attended hemifield which would have been required to support the spotlight hypothesis. In a further variation Zimba and Hughes used larger markers (1 degree squares), at ten target locations in both hemifields. This experiment used peripheral cues rather than central cues to reduce any doubts about which box was being cued. This experiment produced significant costs for invalid targets within the attended hemifield. The size of these costs increased with distance of the target from the cued location. This result is consistent with the results of Downing and Pinker (1985) and the spotlight interpretation. The presence of the markers appeared to be inhibitory and this inhibitory effect increased with eccentricity, these two factors were examined in a last variation of the basic experiment. In this instance performance was compared in a condition using landmarkers of a constant size at all locations, with the use of landmarkers that were scaled according to the cortical magnification factor. This meant that the landmarkers increased in size from 1 degree square, at a viewing angle of 1 degree, to 6 degrees square at 10 degrees visual angle. The use of unscaled markers produced costs in the attended and non-attended hemifields. When the markers were scaled there were no

differences in RT's to targets in the attended hemifield, which was comparable to the results obtained in an unstructured visual display. The implication is that experiments that have shown a graded decrease in performance for non-attended targets within the attended hemifield which are interpreted as a spotlight of attention moving across visual space are in fact a result of an inhibitory effect produced by large markers of the target locations. In the absence of markers, or if the markers are scaled according to the cortical magnification factor a pattern of results consistent with the hemifield inhibition hypothesis are obtained.

The hemifield inhibition model of attention is useful in showing that one effect of directing attention is to produce a slowing of RT's for targets in the non-attended hemifield/quadrant. Their work with marked and unmarked visual arrays has shown that an extra inhibitory effect is introduced by the presence of the marker boxes, which could explain the significant benefits obtained in many of the experiments of directing visual attention. However, their results are also compatible with another model of directing visual attention the 'premotor' account (Rizzolatti et al., 1987; Tassinari et al., 1987). The premotor explanation is based on the motor planning required to make an eye movement to the cued location, large costs for targets in the non-attended hemifield are explained by the time required to cancel a motor direction programme and reprogrammed a movement in the opposite direction. The experiments supporting this view are outlined in the next section.

1.5 Premotor model of attention.

Tassinari et al. (1987) performed a covert manual reaction time experiment to five small LED's located along the horizontal axis. The cues were a written instruction of the likely target location presented at the start of each block of cued trials, or an instruction to attend to all locations on neutral trials. This procedure was used to remove problems of interpreting the cue on neutral trials which could alter the subjects response strategy. Tassinari et al. (1987) in contrast to Hughes and Zimba (1985; 1987) obtained significant benefits for targets in the attended direction compared to the neutral trials, and large costs if the target was in the non-attended hemifield. There were no costs for non-attended targets appearing within the attended hemifield. Tassinari et al. suggested an explanation that directing attention operates in terms of motor planning of an overt saccadic eye movement. The covert orienting reaction time experiments used to demonstrate the affects of visual attention involve the eyes remaining fixed at a central location, however the motor responses required to make an overt movement could still be programmed. The preprogramming of the overt movement could give targets in the attended direction an advantage over targets in the neutral condition. Targets in the non-attended direction will require the cancellation of the initial programme and the computation of a new direction programme. Invalid targets in the attended hemifield will not require the recalculation of direction, but only a small readjustment to the size of the movement. This explains why there are no costs obtained in the attended hemifield for targets at the non cued location. Directing attention to a specific location enables a motor response to be planned in that direction. Any stimulus appearing in the same direction, but at a non-attended location will

require only a small adjustment to that programme. In neutral trials there will be no preprogramming of the direction component until the target has appeared, resulting in longer reaction times in the neutral than attentional condition.

The premotor account is strengthened by a series of experiments performed by Rizzolatti et al. (1987) who directed attention in a covert orienting manual reaction time task, along both the horizontal and vertical meridians. These experiments were devised to dissociate between accounts of visual attention which suggest a movement across visual space (Posner, 1980), and predict costs for invalid targets within the attended hemifield, and the hemifield inhibition account of Hughes and Zimba (1985), which predicts no such costs. This experiment also examined the effect of movements of attention which cross either the vertical or horizontal meridians to see if there is something special in terms of allocating attention in the left and right hemifields. Rizzolatti's experiment used four horizontal target locations in the upper or lower visual fields, and vertical target locations in the left or right hemifield. The subject fixated on a central location and was cued by a central number cue (digits 1 to 4) to a specific target location, the cue was valid on 70% of trials. In neutral trials all target locations were equiprobable, cued by a 0 at fixation. All of the target locations were marked by boxes. The same pattern of results was obtained directing attention along the horizontal and vertical axis, although RT's were marginally faster overall in the vertical direction. A small benefit for valid targets (7 ms) was shown, while invalid trials produced much greater costs (32 ms) when compared to the neutral trials. A significant slowing of RT's was obtained for invalid targets occurring in the same hemifield as the cued location (attended hemifield). This result does not support the hemifield inhibition hypothesis of Hughes and Zimba (1987), but could be explained by the presence of the markers used in the visual display (Zimba and Hughes, 1987). Rizzolatti et al. prefer an explanation based on the time to programme the direction and exact distance of a motor response (Rosenbaum et al., 1982; 1984). They suggest that there is a strong link between the neural mechanism which moves the eyes and that which covertly orients attention. As movements of visual attention are closely linked to movements of the eyes, covert orienting could reflect a voluntary suppression of the final execution of the saccade programme. The eye movement programme will specify the direction to move in, as well as the exact location. These two coordinates could be programmed separately in series, or in parallel. Modification to either the direction or location aspect of the computation will require a certain amount of time. These assumptions can be used to explain why responding to invalid targets which requires a movement of attention crossing either the horizontal, or vertical meridians, results in a larger cost in RT performance, than invalid targets in the same hemifield. Invalid targets in a non-attended hemifield will require the initial motor programme to be changed to incorporate a new direction and specific location. Invalid targets in the attended direction will require a change to the distance programme only, resulting in a smaller cost. This model also accounts for the speeding of RT's in the valid trials compared to the neutral trials as valid trials enable the motor programme to be preprogrammed. The small facilitation obtained by valid cues implies that the programming of the motor response is very quick, while the cancelling of a programme in invalid trials requires a larger amount of time. Rizzolatti et al.'s model states that a cue is used to build up a motor programme which takes a certain length of time, in normal circumstances this would

result in a saccadic eye movement being made to bring the fovea onto the target.

The large cost obtained when a movement of attention crosses the vertical meridian is compatible with models of saccade generation that make a distinction between the programming of direction and amplitude. Becker and Jürgens (1979) model of of saccade programming uses two separate mechanisms that compute the decision to initiate a saccade ('when' component) and the spatial location of the saccade ('where' component). The when component is responsible for triggering the saccade and is also concerned with the direction of the saccade. In this model the direction of the saccade must be specified before the amplitude of the saccade. It is the direction processing which is thought to account for the resulting latency of the saccadic eye movement. Changes to the direction of the saccade by cueing a location in one hemifield while the target is presented in the opposite hemifield, will require a modification to the direction programme and would account for the observed increase in the latency of the saccade when a target is tracked from one hemifield to another. This model of saccade generation is consistent with Rizzolatti et al.'s explanation of the increase observed with manual RT's when an attentional crossing of the vertical meridian occurs.

Abrams and Jonides (1988) have questioned the fixed order of hierarchical programming suggested by Becker and Jürgens (1979) model. They used the movement cueing technique of Rosenbaum et al. (1984) which they suggested prevented any interference between the programming of features of saccades and visual/perceptual processes. Abrams and Jonides obtained evidence that saccade direction and amplitude can be programmed separately, but there is not necessarily a fixed order of sequential programming as suggested by the model of Becker and Jürgens (1979). Cueing either the saccade direction, or the saccade amplitude, reduced saccade latency when compared to a condition in which the cue specified neither amplitude or direction, of the required saccade. It is possible to criticise the cueing technique used by Abrams and Jonides on the grounds that the cue appeared as an abrupt onset in the hemifield opposite to that in which the target appeared, which could be expected to automatically summon attention to the opposite side (Todd and Van Gelder, 1979). This could produce an interference effect between attentional factors and advanced programming of saccade parameters which they had initially set out to avoid. Indeed when they repeated their basic experiment but using cues presented in the same hemifield as the target the results suggested that the saccade amplitude and direction were not programmed separately. Abrams and Jonides explain these conflicting findings in terms of the voluntarily preparation of a saccade being performed in terms of a separate direction and amplitude computation without the fixed order inherent in Becker and Jürgens model, while a sudden abrupt onset can organise the programme more 'holistically' solely in terms of the final desired location.

Shepherd and Müller (1989) obtained RT data (examined in section 1.3.1) to examine the analogue movement of attention and obtained results more consistent with a focusing of attention explanation. They also suggest that their data can be explained more adequately in terms of the premotor theory of attention. They showed equal benefits for near and far targets

on the cued side with short SOA's. The benefits decreased for the non cued location in the cued field as SOA increased. This is consistent with models of saccadic eye movements that incorporate the idea that a change in the direction computation takes more time than a change to the amplitude of the saccade (Rizzolatti et al., 1987). The facilitation of the whole hemifield following a central cue is attributed to preparing a saccade in the appropriate direction. The decline in facilitation for the non cued location reflects the exact amplitude of the saccade having being computed with longer SOA's. The large costs for targets in the non cued field reflects the effects of having to reprogramme the direction of the saccade, producing equal large costs for targets in the non cued hemifield.

The link between the oculomotor system and the visual attentional system has been indicated in the experiments of Shepherd, Findlay and Hockey (1986). They measured manual RT's and saccade latencies to targets when spatial attention was manipulated by target probability and saccades were cued at the same time by a central arrow cue. Manual RT's were shortened by target probability and preparing to make a saccade to that location. When the saccade was made to a low probability location, manual RT's to targets at the high probability location were delayed, which suggests that attention cannot be moved in the opposite direction to a saccadic eye movement. So it appears that covert orienting of attention is possible without an eye movement being made, but it is not possible to move the eyes to a location without moving attention in the same direction.

The oculomotor explanation of visual attention has not been supported in all experimental work. Klein (1980) investigated the effects of directing an eye movement to a specific location on both saccadic RT's and manual RT's, and concluded that visual attention is not controlled by the eye movement system. This dual task, directed subjects to make a saccade to a target location either left or right of fixation, while the target could appear at either of the two locations. The direction of the eye movement was constant across blocks of trials. On twenty percent of trials a manual response was required to one of the peripheral locations signalled by the brightening of the marker dot. The prediction of the oculomotor explanation is that when the manual response coincides with the location of the saccadic eye movement, the RT should be faster than in trials when it occurs at the opposite location. Saccades were shown to be some 40 ms faster for targets in the attended direction than in the non-attended direction. Manual RT's were found to be the same for targets at the saccade location and the opposite location which fails to support the oculomotor hypothesis. This independence between directing the eyes and detecting a stimulus implies that visual attention does not move towards the saccade target, refuting the idea that visual attention is linked to overt shifts of the eyes. One possible explanation of this failure of finding any costs and benefits of the manual RT's is that the dual task was a complicated one indicated by the generalised slowing of saccade latency and RT's when compared to responses made in blocks of saccades, or manual RT's, only. Manual RT's are some 100 ms slower in the dual task condition compared to the single response blocks which could mask any costs or benefits obtained by directing attention. Klein's data cautions against this explanation as there was no difference between error rates for targets at the saccadic location and opposite location. The failure to find evidence of an increase in the

size of costs for crossing the vertical meridian with saccades is a problem for the premotor account, but this may reflect the use of peripheral cueing procedure. Some support for this view is obtained from Umiltá et al. (1991) who failed to show the meridian effect following peripheral cueing on manual RT's, which they explain by an inhibitory process that builds up following a peripheral cue and favours responses made on the non cued side (Tassinari et al., 1989).

Crawford and Müller (1992) used peripheral cueing of saccadic eye movements, to investigate the hierarchical programming of saccade direction and amplitude explicitly implied in Rizzolatti et al.'s premotor model. They obtained benefits for targets that appeared at the exact cued location only. There was no advantage for invalid targets presented on the cued side (same hemifield) which require a change in the amplitude programme without any change to the direction programme and more importantly no increase in the size of the costs for invalid targets in the non cued hemifield which do require a change to the direction component of the saccade programme. In contrast repeating the experiment with peripheral cueing of manual RT's, they found a general advantage for targets in the cued hemifield and an increase in costs for targets in the non-attended hemifield. Crawford and Müller interpreted these results as showing that for saccadic eye movements the amplitude and direction are programmed holistically, whereas the motor programme for manual RT's is programmed in a hierarchical way as suggested by Rizzolatti et al.. The conclusion being that the failure to find the increase with costs associated with crossing the vertical meridian with saccades, while it is shown with manual RT's shows that the same underlying system is not responsible for covert attentional orienting as is responsible for producing saccadic eye movements. However, the failure of Crawford and Müller to find the meridian crossing effect with saccade latencies may be due to the use of peripheral cues; as Umiltá et al. (1991) failed to find the meridian crossing effect with manual RT's following peripheral cueing. A second factor cautions against rejecting the premotor model on the findings of Crawford and Müller, it can be seen that the mean saccade latencies obtained in their experiment are slower than would be expected for normal subjects in this type of cued saccade task. This may be due to the use of peripheral cues building up an inhibition for targets on the cued side which reduces the magnitude of the meridian crossing effect as suggested by Umiltá et al. (1991).

A further problem for the premotor model is accounting for increases in manual RT's observed as the distance from the cue to target locations increases (Shulman et al., 1985; Tassinari et al., 1987; Tsal, 1983), as saccade latency is not affected by distance of the target from fixation at eccentricities of up to 20°degrees (See for a review: Findlay, 1983). Rizzolatti et al. argued that manual RT's should not change with distance of target from fixation, as attention moves to the cued location once the motor programme is completed. The results of Remington and Pierce (1984), which showed that RT's are unaffected by target eccentricity, supports this claim, but Rizzolatti et al.'s own results did show a significant slowing of RT's with eccentricity, but only when comparing the 2 degree and 12 degree locations. They do not explain why other workers have obtained eccentricity effects in manual RT experiments which poses a problem for their model of the saccadic system controlling covert orienting of attention. The issue of visual attention being controlled by the eye movement system is still an unresolved issue and one

which will be examined in this thesis.

1.6 The gap effect and visual attention.

The experiments described thus far have used cueing procedures to covertly and overtly direct attention and have examined the facilitatory and inhibitory effects produced on manual reaction times and saccade latency. The following section examines a different procedure in which the attended stimulus is removed prior to target onset which has been shown to speed overt responses, which could be due to an acceleration of attentional deployment. In this type of experiment a temporal interval (or gap) is present between the fixation offset and target onset which has resulted in the term 'gap' paradigm being applied to this type of study.

Saslow (1967) showed that saccade latency was reduced if a central fixation point was removed before the onset of the peripheral saccade target in a 'gap' condition, compared to conditions in which the fixation remained on while the target was presented in an 'overlap' condition. The maximum speeding on latency was noted if the fixation point was removed 150 ms before the target onset, the slowest saccades were observed if the fixation point remained on for overlaps of over 100 ms. A '0 gap' in which fixation offset and target onset were coincident produced intermediate latency saccades. Saslow rejected the idea that the speeding of saccades is due to the offset of the fixation point acting as a warning signal, enabling the saccade to be prepared before the target appears. If this was the case then overlaps of over 100 ms would not give any speeding of saccade latency. Saslow's explanation is that the presence of a fixation point produces microsaccades, which leave the saccadic system in a refractory period that inhibits the generation of the next saccade.

Ross and Ross (1980) examined the effects of visual onset, offsets and a change of events at fixation and showed that there was a reduction in saccade latency associated with all three conditions, but the greatest facilitation was found with stimulus offset. Prior fixation offset, onset and change all produced a reduction of saccade latency, indicating that all are sufficient to act as a warning event. However, with gap intervals of 0 and 100 ms the offset of the stimulus showed mean saccade latencies of 230-250 ms, some 40-60 ms faster than in the onset and change conditions where a mean latency of 270-310 ms was produced. Longer gap intervals of 300 and 600 ms showed comparable mean saccade latencies of 240 ms in the onset, offset and change conditions. In a second experiment overlap intervals in which the stimulus offset, and onset occurred after the saccade target appeared were used, and showed that saccade latency is slowed by the onset of a stimulus after target onset, but is not affected by the offset of the stimulus after target onset. The delay associated with the onset of the stimulus could be due to a disruption of the ongoing saccade programme, or it could be due to the presence of microsaccades which inhibit saccadic generation (Saslow, 1967). Alternatively the foveal onset could activate the transient attentional channels, summoning attention back to the foveal location, causing a delay in orienting attention to the target and in moving the eyes to the

peripheral location. The conclusion is that all three events (offset, onset and change) act as warning signals which reduce saccade latency. Onset and change produce an initial slowing of saccades due to an inhibitory process which interferes with saccade generation at intervals up to 150 ms after peripheral target onset.

The results of many experiments have revealed evidence that a facilitation effect is also obtained with the offset during the overlap of fixation and target (Reulen, 1984; Ross and Ross, 1980; Saslow, 1967). This is shown by a speeding of latency with fixation offset occurring at intervals up to 150 ms after saccade target onset also produces a speeding effect on latency when compared to latency obtained with longer overlaps. This facilitation could still be accounted for in terms of a warning signal effect (Ross and Ross, 1980) especially if an offset is perceived before a stimulus onset. It is also compatible with the idea of facilitating the disengagement of attention thus speeding the latency of the saccade (Fischer and Breitmeyer, 1987) if it is assumed that this process takes over 150 ms to complete. Braun and Breitmeyer (1990) performed an experiment to examine the effects of the reappearance of the attended fixation point. They showed that the prior onset of fixation can speed saccade latency if it occurs up to 100 ms before target onset and the reappearance of fixation after target onset produces a slowing on saccade latency. They see the facilitation effect with prior onset as being compatible with either the warning signal effect, or prior attentional disengagement. The onset of fixation after target onset could slow saccade latency by reengaging attention.

Fischer and Breitmeyer (1987) reviewed experiments similar to that of Saslow's, which examine the effects of fixation offset on saccade latency and considered the implications for visual attention. Two main effects are observed in gap conditions the first is the decrease in saccade latency with increasing interval between fixation offset and target onset as noticed by Saslow (1967). The second finding is the presence of a bimodal distribution of saccade latency obtained under gap conditions. Fischer and Boch (1983) and Fischer and Ramsperger (1984, 1986) used a gap paradigm with monkeys and human subjects and showed a bimodal distribution of saccade latencies. The first peak of the distribution showed a group of saccades with extremely short latencies in the order of 100-120 ms in humans, which have been termed 'express saccades', with a second peak of normal latency saccades at 170 ms. The probability of obtaining express saccades increases as the gap between fixation offset and target onset increases. Fischer uses an attentional explanation incorporating Posner's idea of attentional engagement and disengagement to account for the reduction of regular saccade latency in the gap paradigm. When attention is engaged at a location it inhibits the saccadic system, so attention must first be disengaged from fixation, before it can be moved to a new location. Removal of the fixation point before the target appears, enables the system to be in a disengaged state and facilitates saccade generation. This argument is supported in experiments where subjects attended to a peripheral stimulus, while gazing at the centre of a screen. Saccade latency towards a target is reduced when the peripheral stimulus is turned off, even though the central fixation stimulus remained unchanged (Braun and Breitmeyer, 1988; Mayfrank et al., 1986). Directing attention to either a central fixation point, or a peripheral stimulus has been shown to reduce the occurrence of express saccades (Mayfrank, Kimmig and

Fischer, 1987). So directed attention at the moment of target occurrence abolishes express saccades. The reduction in saccade latency by prior removal of an attended stimulus has been replicated in many experiments, however the presence of a separate population of express saccades has been questioned (Wenban-Smith and Findlay, 1991). Fischer and Breitmeyer's model of engaged/disengaged attention can account for the reduction of regular saccade latency in gap situations, but does not explain why there should be a sudden change from the fast regular to the express state.

Tam and Stelmach (in press) further examined the role of attentional disengagement in the gap effect, to see if it had a unique role in the speeding of saccades. This was achieved by comparing saccade latencies obtained with the offset of the peripheral attentional stimulus, with a condition in which the attentional stimulus remained on and the non-attended fixation point went off. If the gap effect depends on the attentional system only, then offset of the non-attended fixation point would not be expected to reduce saccade latency. Saccade latency was however shown to be reduced by prior offset of the attended stimulus and with the offset of the non-attended fixation point. The greatest facilitation effect being produced by the offset of the non-attended fixation stimulus. The possibility that the offset of the non-attended fixation point acts as a warning signal, was examined by measuring manual reaction times under similar conditions. Prior offset of the attended stimulus and non-attended fixation stimulus, both speeded manual RT's suggesting that part of the facilitation effect obtained is due to a warning signal effect. With manual RT's there was no difference in the results for offset of the attended or non-attended stimulus. The conclusion is that the reduction in saccade latency depends on both the attentional and ocular sampling systems. Prior disengagement of both systems speeds saccade latencies.

Reuter-Lorenz et al. (1991), examined the possibility that the facilitation effect of the gap condition could be due to sensory processes (Reulen, 1984), or oculomotor readiness (Kalesnykas and Hallett 1987), or attentional factors (Fischer, 1987). They examined the predictions of Reulen's (1984 ab) facilitation model of saccade latency. This model is based on an accumulation of sensory activity produced by target onset, which increases at a specific 'rise rate'. The rise rate of sensory activity is determined jointly by an accumulation of the signal intensity (brightness of target) and a facilitation factor produced by fixation offset. Brighter targets and greater gaps will have a commutative effect on the rise rate of sensory activity. When the level of activity has exceeded a threshold level the system is put into a fast operating or 'facilitated' state, which gives faster processing of the saccade parameters attributed to enhanced early visual processes. This model predicts that saccade latency should increase with dimmer targets and this increase should be greater with increasing overlaps between fixation and target onset. Reuter-Lorenz measured saccade latency in gap and overlap conditions, with both bright and dim targets to examine predictions from the facilitation model. Results showed that reduction in saccade latency in the gap condition was not additive with the effects of target luminance as would be predicted from Reulen's model.

In a second experiment Reuter-Lorenz examined the attentional and oculomotor

explanations of the gap effect, by comparing manual reaction times; normal saccade latency; as well as saccade latency when the eye is moved in the opposite direction to the target, termed 'antisaccades' (Hallett, 1978). Antisaccades are thought to involve different neural pathways than those required for normal saccades (Guitton et al., 1985), but could still be speeded by disengagement of attention. Fischer's attentional disengagement hypothesis should predict that both normal saccades, and antisaccades should be speeded under gap conditions. Preprogramming explanations of the gap effect on saccades latency (Kalesnykas and Hallett, 1987), predict a speeding of both normal saccades and antisaccade latency. Reuter-Lorenz further suggested that a similar preprogramming explanation could be applied to manual reaction times. The results showed a facilitation effect of the gap effect for normal saccades, and no significant speeding for manual reaction times, or antisaccades. The preprogramming explanation of the gap effect on saccade latency is not supported, nor is the attentional disengagement hypothesis of Fischer. The lack of the gap effect on manual reaction times is used to suggest that the effects of fixation offset are specific to oculomotor processes only. In particular Reuter-Lorenz suggests that the gap effect could affect collicular mechanisms involved in generating saccades, by releasing the system from inhibition produced by active fixation. The collicular mechanisms are not thought to be involved in manual reaction times, or antisaccades. Other workers however have found a facilitation effect of fixation offset on manual reaction times (Ross and Ross, 1981), Reuter-Lorenz suggest that this may be due to subjects making more anticipatory responses in experiments where the fixation offset provides the only warning signal of the impending target.

Recent work by Mackeben and Nakayama (in press) directly examines Fischer's attentional hypothesis of the gap effect by using a visual search task, which involves the deployment of attention to discriminate a target from distractor. Performance curves of the percentage correct responses plotted for various cue to target intervals, showed a significant increase in rise rate with a gap condition compared to a no gap condition. Control experiments showed that this increase in performance was specifically related to fixation point offset and not to a warning signal effect. This result suggests that an attentional movement to the cued location will be speeded in the gap condition due to attention being in a disengaged state following fixation offset.

The use of gap conditions have shown that saccade latency is reduced and target detection facilitated if fixation point offset occurs before target onset. This could be due to oculomotor readiness (Kalesnykas and Hallett, 1987; Saslow, 1967), a warning signal effect (Ross and Ross, 1980; 1981), a facilitation of premotor processes (Reuter-Lorenz et al., 1991), attentional disengagement (Fischer and Breitmeyer, 1987; Mackeben and Nakayama, In press) or an interplay between ocular and attentional processes (Tam and Stelmach, In press). The evidence reviewed strongly implicates the role of attention in producing the gap effect so it is of interest to examine the gap effect in experiments that manipulate attention with other manipulations such as cueing attention by central and peripheral cues.

1.7 Bilateral target presentation: Effects on saccade latency.

The saccadic reaction time experiments mentioned above have used attentional manipulations to examine the effects of attention and/or saccade preprogramming, on saccade latency to single targets. These single targets are placed either to the left, or right, of fixation and saccade latency measured under various attentional and stimulus presentation conditions. However, a small number of experiments have examined the effects of presenting two targets, either both in the same hemifield, or one in each hemifield. The effects produced using this technique are relevant to theoretical models of saccade generation and could also be relevant to an oculomotor explanation of visual attention.

Presenting two targets in the same hemifield has been shown to have little or no effect on saccade latency, but has been shown to affect the amplitude of the resulting saccade (Findlay, 1981; 1983). Saccade amplitude was shown to increase when two targets are presented on the same side away from fixation, with the saccade falling at an intermediate location between the two targets. Coren and Hoenig (1972) termed this overshoot the centre of gravity effect, while Findlay (1982) termed it the global effect. Findlay (1983) showed that the overshoot of the saccade in the global effect is linked to saccade latency, with greater amplitudes occurring with shorter latencies. Longer delays in latency result in saccades being more accurately directed to the target. The effect is explained in terms of a parallel processing model of saccade generation, in which the direction of a saccade is calculated separately from the amplitude. The implication is that short latency saccades have not had sufficient time for the amplitude computation to be completed, before the saccade is initiated, resulting in an averaging of amplitude and overshoot of the saccade. Early visual processes are shown to influence the global effect, as the saccade will fall closer to one of the pairs of targets, if it is larger, or more intense, than the other.

Becker and Jürgens (1979) and Findlay and Harris (1984) examined the programming of saccadic eye movements by presenting targets that moved in a step jump from one position to a second following a time delay. The results to double steps on the same side of fixation showed that a saccade initiated at a time before or up to 80 ms after the second step occurred will be made to the first position, if the saccade is made at long intervals after the second step (180 ms +/-10) then it will be made to the second position. Saccades initiated at intermediate times show a modification of amplitude and fall at intermediate locations between the two eccentricities. Double step targets which crossed the vertical meridian produced saccades to either the first or second location without producing intermediate amplitude saccades. Saccade latency is shown to be increased with stimuli that cross the vertical meridian with especially long latencies associated with saccades made to the second step. Findlay and Harris showed that saccade latency was not increased for double steps which crossed the vertical meridian above the central point. Becker and Jürgens (1979) propose a model of saccade generation in which the amplitude and direction are programmed separately, whereas Findlay and Harris (1984) favour a model in which amplitude and direction are programmed in a more holistic way possibly

in terms of a retinotopic motor map (McIlwain, 1976).

Two targets appearing simultaneously in opposite hemifields do not affect the amplitude of the saccade, but have been shown to produce a significant slowing on saccade latency. Lévy-Schoen (1969) showed that saccade latency was slowed by some 40 ms, with double targets presented in opposite hemifields. Findlay (1983) also showed a comparable slowing of latency with double targets at 2 and 3 degree eccentricities presented simultaneously left and right of fixation. With double targets the subject was free to saccade to either one of the two targets and latency was slowed by some 40 ms. Findlay explains the slowing in terms of a model of saccade generation which is similar to that of Becker and Jürgens (1979). The model incorporates a separate decision mechanism to produce a saccade in a certain direction, and a mechanism to calculate the size of the saccade. Left and right direction 'initiate' components, subject to reciprocal inhibition are incorporated into the model and are used by Findlay to explain the slowing of latency to double targets. Double targets will produce an increase of inhibition for both the left, and right saccade 'initiate' components. The increase in saccade latency reflects the time taken to overcome this extra inhibition. Two targets appearing in the same hemifield will produce an increase of inhibition for the initiate component to make a saccade in the opposite direction leaving saccade latency unaffected.

The slowing of saccade latency for double targets presented on opposite sides of the vertical meridian may be similar to the slowing shown on manual RT experiments when attention crosses the vertical meridian. There could therefore be a link between the slowing shown to double step and bilateral target presentation and the meridian crossing effect shown following central cueing of attention. Also If there is a link between the saccadic eye movement system and the attentional movement system (Rizzolatti et al., 1987; Tassinari et al., 1987), then double target stimulation could be expected to interact with measures of visual attention. The slowing of saccade latency to double targets, could be accommodated into inhibition models of visual attention such as Hughes and Zimba (1987) if it is assumed that attention automatically produces inhibition for the contralateral hemifield, when a stimulus is presented. For this reason both single and double targets will be used in the following experiments measuring saccade latency with attentional manipulations. In particular it is of interest to show if the slowing of saccade latency with double targets in opposite hemifields is still apparent when the subject is instructed to always saccade in one direction. If the slowing is still observed it could reflect an automatic inhibitory effect at a low level of visual processing such as visual attention.

The following chapters describe experiments which measure saccade latency to single and bilateral targets using central cueing procedures to examine the inhibitory affects of double target presentation and the possibility that it is associated with attentional mechanisms.

1.8 A deficit of visual attention: Unilateral spatial neglect.

1.8.1 Introduction

The accounts of visual attention mentioned so far have considered results from experiments which used attentional manipulations on a variety of tasks, in normal subjects. It is also of interest to study occasions in which the attentional system has become impaired following brain damage to develop a greater understanding of the way the attentional system functions in its normal capacity. One commonly studied impairment shown in patients following brain damage, which is often thought of as having an attentional origin (eg Kinsbourne, 1977; Posner et al., 1984; 1987), is **unilateral spatial neglect**. It should be mentioned that neglect is not only viewed as being due to a deficit of attention as it has also been viewed as resulting from a deficit of the internal mental representation of space (eg Bisiach and Luzzatti, 1978; Bisiach et al., 1979; Bisiach and Berti, 1987; DeRenzi, 1982). These two views will be outlined briefly here and discussed in greater detail in Chapter five. The following section will also provide a brief introduction into some of the other issues which are important to understanding the nature of the neglect condition are also introduced here and will be discussed in greater detail in Chapter five.

Unilateral spatial neglect can be seen in a wide variety of everyday tasks and can also be evinced on a range of clinical tests. For example, the neglect patient may leave food on the left side of a plate, bump into things on their left and only dress the left side of their body. When reading single words neglect patients omit, or substitute the initial letters and when reading text can miss out words on the left side of the page.(Riddoch and Humphreys, 1991; Young et al., 1991). When copying a simple line drawing, and more interestingly, when drawing from memory they leave out left sided details. Clinical tasks used to illustrate neglect include crossing out tasks (Albert, 1973) and line bisection. In crossing out tasks patients are asked to cross out lines from a which are randomly presented on a page and fail to cross out lines located on the contralesional side of a page. In line bisection tasks the patient is asked to bisect the centre of a horizontal line and typically deviate towards the ipsilesional end of the line.

1.8.2 The nature of the brain damage associated with neglect.

Neglect occurs more frequently following damage to the right hemisphere resulting in left sided neglect, than following left hemisphere damage (see DeRenzi, 1982: for reviews). This suggests that the right hemisphere has a dominant role in processing spatial information, so that damage to the right hemisphere leaves the patient with a greater impairment on spatial tasks than does damage to the left hemisphere. The presence of neglect in man is typically associated with unilateral lesions in the region of the inferior parietal lobe (Heilman, Watson, Valenstein and Damasio, 1983), although other brain areas, such as the frontal lobe (Heilman and Valenstein, 1972) and basal ganglia (Damasio et al., 1980), have also been implicated. The right hemisphere in man seems to have a greater degree of involvement in attention which

explains why neglect is more common following right brain damage and why patients with left brain damage show a much faster recovery from neglect than do the right brain damaged patients. The specific areas of brain damage and the dominant role of the right hemisphere in producing neglect are discussed in chapter five.

1.8.3 The frames of reference involved in neglect.

A further issue which has been the subject of experimental studies of neglect patients involves the frames of reference that defines the neglected and non neglected areas. Neglect could operate in terms of a viewer centred frame of reference, termed the *egocentric* coordinates, which could represent locations in terms of retinotopic, head centred, or body centred representations. With regard to neglect operating in terms of egocentric coordinates the evidence suggests that it does not operate in terms of retinotopic coordinates (Ladavas, 1987; Karnath and Fischer, 1991)

The second possibility is that neglect operates in terms of representations that are independent of viewpoint which are termed *allocentric* coordinates. The allocentric coordinates could represent locations in terms of environmental locations, or in terms of the locations of objects. Calvanio et al. (1987) decoupled the viewer centred and environmentally centred coordinates by having patients scanning an array when sitting upright and reclined on their sides. The results indicated that patients neglected the left side of the array when upright, but when they were reclined they neglected stimuli to their left and to the left of the array. This suggests that neglect operates in terms of both the viewer and environmental representation. Ladavas (1987) showed that when patients performed a RT task with their heads tilted, they continued to be slower responding to stimuli which would have been left most if their heads had been in the upright position. This suggests that neglect can operate in terms of the left side of a display irrespective of viewpoint in allocentric coordinates.

If neglect operates in terms of an object centred description (allocentric) then patients may neglect the contralesional features of individual objects. If an object centred frame is considered then 'left' is defined as being left of the object's midline and this remains stable with changes in the object's and observer's position. Experimental work by Farah et al. (1990) failed to show evidence of object centred neglect in neglect patients, but more recent work by Driver and Halligan (1991) has provided evidence of neglect operating for the left side of objects in a single patient P.P. When copying a line drawing of two bicycle wheels P.P. missed out left sided features of each wheel, but when these wheels were drawn so that they formed part of a bicycle P.P. copied the whole of the right sided wheel while missed out the left features from the whole bicycle.

It appears that neglect can operate in terms of one or more of these coordinate systems which could account for the many different patterns of neglect shown by different patients. The frames of reference involved in neglect is discussed in greater detail in Chapters five and also in Chapter eight with respect to the proposed model of visual attention.

1.8.4 Neglect dyslexia.

Neglect patients typically show two different types of reading errors which are classed as 'neglect dyslexia' (Ellis et al., 1987). Then first involves the misreading of single words in which the initial left sided letters are either omitted or substituted to give a plausible real word alternative (eg MEND as end, MALT as salt) and the second involves omitting whole words on the left side of a page. The single word reading error are not thought to result from an impairment of the word recognition system, as the patients can read words correctly if they are presented in a vertical orientation. Riddoch et al. (1990) suggest that the single word reading errors could be due to the patient failing to attend appropriately to individual words. It is possible to show neglect patients that make one of these misreading errors which suggests that the whole word and initial letter misreadings arise in a different way (Ellis et al., 1987; Young et al., 1991). Young et al. (1991) showed that misreading errors in single words occurred regardless of left/right spatial position and visual field that the words were presented in. They suggest that these errors could reflect an impairment of the distribution of attention within the word-form system (cf. Costello and Warrington, 1987). Whole word omissions could reflect an impairment in the patients making an overt orienting response involving a large left saccade to locate the start of the subsequent line (Riddoch and Humphreys, 1991; Young et al., 1991). The impaired ability at making a left saccade could have an attentional explanation.

1.9 Accounts of neglect.

1.9.1 Perceptual explanations.

One of the simplest accounts of neglect and also the one which is easiest to discount as a plausible explanation, is that the patient fails to respond to stimuli in the contralesional side of space, due to a visual field defect (Battersby et al., 1956; Denny-Brown et al., 1952). Zarit and Kahn (1974) showed that the degree of neglect shown by the patients they studied, correlated with other impairments such as visual field defects and loss of intellectual capacity. Other workers however, have failed to show any evidence of a loss of intellectual functioning (Lawson, 1962; Ettlinger, Warrington and Zangwill 1957). Hécaen (1962) showed that neglect is associated with a high level of visual field defects as 76% of his patients had a hemianopia recorded. However, visual field defects are not thought to be the cause of neglect for two reasons. The first is that it is possible to find patients without any sensory loss, who do still show neglect (Girotti et al., 1983; Karnath and Hartje, 1987). Conversely patients who do show evidence of having a visual field defects (due to a cortical lesion), do not always show neglect as they make compensatory eye and head movements (Ishail et al., 1987; Meienberg et al., 1981).

1.9.2 Representational explanations.

The representational view of neglect (eg Bisiach and Luzzatti, 1978; DeRenzi, 1982) relies on neglect being due to a higher level deficit than a loss of early visual processing. This explanation proposes that the unilateral damage to the cortex results in a loss of the representation for space contralateral to the lesion site. This loss of the representation results in neglect for contralesional stimuli. The representational view is weakened by studies which have shown that the information in the neglected field is processed to some degree (Karnath and Hartje, 1987; Marshall and Halligan, 1988) suggesting that there is not a complete loss of a representation for that side of space. It has been shown that neglect can be reduced by cueing the patient in the neglected field which the representational account does not readily account for (Posner et al., 1984; Riddoch and Humphreys, 1983).

1.9.3 Attentional explanations.

The third view of neglect which is central to this thesis is that it results from an impairment of visual attention. Kinsbourne (1977) attentional hypothesis accounts for neglect in terms of inter-hemispheric inhibitory processes. Activation of one hemisphere is thought to inhibit activity in the other hemisphere (Kinsbourne, 1970). In addition Kinsbourne suggests that there is a bias towards orienting towards the right side of space in normal subjects which will be countered by a normally functioning right hemisphere. Damage to one hemisphere will result in the loss of inhibition for the intact (contralateral) hemisphere, which will lead to overactivity with the result of attention being directed in the ipsilesional side. Evidence against this particular attentional theory is provided as neglect is not abolished following bilateral lesions (which would be expected to restore the imbalance of inhibitory activity) but can result in bilateral neglect (Segarra and Angelo, 1970).

Heilman (see. Heilman, Watson and Valenstein, 1979) suggested that neglect results from a reduction in arousal levels in the damaged hemisphere. This selective loss of arousal results in neglect for the contralateral side of space. Heilman terms this selective loss of orienting responses 'directional akinesia', as neglect patients have difficulty moving their eyes and limbs in the contralesional direction which is not due to lesions to the motor neurons. The further assumption is made that the left hemisphere controls orienting to the right side only, while the right hemisphere controls orienting to both sides of space. This accounts for the larger number of occurrences of left neglect following right sided lesions. However, the results of Riddoch and Humphreys which show that neglect for the contralateral side of space can be reduced by cueing suggests that the the damaged hemisphere cannot be regarded as being akinetic.

One further attentional explanation is that neglect results from an impairment of visual attentional orienting. Posner et al. (1984) suggested that there are three different components involved in orienting attention which can be illustrated in covert cueing experiments. Initially attention must be disengaged from its current location, then it is moved (oriented) to the cued

location and finally it is engaged at targets at that location. Neglect patients were shown to produce equal RT's to targets in both hemifields following a valid cue to the target location showing that they could engage attention on contralateral targets. The patterns of RT's obtained showed that RT's improved to valid targets as the time interval between cue and target increased with targets on both sides showing that patients have no difficulty in moving attention in either direction. The neglect patients showed a great slowing of RT's for contralateral targets following a cue to the ipsilesional side, which Posner suggests is due to a selective impairment in disengaging attention from an ipsilesional location if a movement in the contralesional direction is required. Posner showed this deficit of disengaging from ipsilesional stimuli in left and right brain damaged patients although the effect was greater in the right hemisphere group. This suggests that each hemisphere controls attention in the contralateral side of space, but the the right hemisphere could control movements in both sides of space, while the left controls movements in the right direction only.

The available evidence has strongly implicated the role that attention plays in producing neglect. The affect of cueing suggests that the attentional explanation is a more parsimonious one than the representational accounts. The attentional accounts are therefore directly relevant to the work performed in this thesis. In Chapter five, the attentional explanation of neglect will be expanded to incorporate more of the experimental data from studies of neglect patients and the attentional hypothesis expanded to take into account some of these findings. In Chapters six and seven, the results from attentional orienting experiments with a single neglect patient B.Q. are discussed in relation to the attentional hypothesis of neglect. In chapter eight the proposed model of attentional orienting is developed to incorporate an explanation of B.Q.'s attentional deficit.

1.10 Overview of thesis.

This thesis is concerned with the orienting of visual attention. In Chapters two, three and four, the orienting of visual attention in normal subjects is examined. Chapters six and seven examine the deficits of attentional orienting in a single patient (B.Q.) with unilateral spatial neglect. In chapter eight a model of visual attention is developed which it is hoped can account for the findings from the normal subjects and also the findings from the single neglect patient.

It is usual for a person to be attending to the stimulus located on the foveal region of the retina, so there is an obvious link between the process of visual attention and the system involved in making a saccade to bring a stimulus onto the fovea. Models have suggested that the same system may be involved in orienting attention as is used to make a saccadic eye movements. The experimental examination of visual attention performed in this thesis has centred on the importance of saccadic eye movements as a way of studying the attentional process in normal subjects and the nature of the deficit observed in a brain damaged patient with unilateral spatial neglect.

The work with normal subjects investigated the link between the eye and attentional systems by measuring the saccadic reaction times (latencies) obtained to stimulus targets, which were presented under conditions of directed visual attention. The first attentional manipulation involved instructing subjects to attend to a spatial location and examining the effect on saccade latency. A second manipulation involved altering the activity of the central fixation point, which is known to affect saccade latency and has been attributed to attentional processes. The third manipulation was to present bilateral saccade targets in both hemifields, which have been shown to slow saccade latency. The use of bilateral targets has implications for the component involved in selecting the direction of the planned saccade.

These attentional manipulations were shown to have both inhibitory and facilitatory effects on saccade latency. Directing attention seems to have a small facilitation effect on saccade latency, and a much greater inhibitory effect for saccades made to the non-attended hemifield. Prior fixation offset had a facilitatory effect on latency, which was independent to the affects of directed visual attention and bilateral target presentation. Bilateral target presentation had both a facilitatory and inhibitory effect on saccade latency, which depended on the time course of the target presentation. These findings are developed in light of models of saccade generation and models of visual attention in Chapters two, three and four. The final aim being to assess the implications of these results (if any) for models that emphasises the close link between the eye and attentional orienting systems.

Recent attentional models of neglect have suggested that neglect results from a deficit of the attentional orienting system. An impairment of attentional orienting could therefore be expected to effect the production of an overt saccadic eye movements. This possibility was examined in Chapters six and seven by a study of saccadic eye movements made by a neglect patient B.Q. The aim being to examine B.Q.'s ability to make a saccade to stimuli presented left and right of fixation. The affects that manipulations of the fixation point and the presentation of bilateral saccade targets, had on B.Q.'s ability to saccade overtly orient and report the target stimuli was assessed. The findings showed how B.Q.'s neglect could be reduced by removal of the attended fixation point. The reduction in B.Q.'s neglect was considered in terms of the attentional orienting hypothesis of the neglect condition. A further aim was to incorporate these findings into the attentional model, proposed to account for the results obtained with normal subjects.

The affects that the attentional manipulations have on saccade latency are incorporated into a model of the attentional/eye orienting system, in Chapter eight. The aim is to emphasise the inhibitory consequences of visual attention, into a model that could be involved in generating a saccadic eye movement. It is hoped that the model could also be used to account for some of the deficits of attentional orienting observed in B.Q., by speculating about damage to specific components of this model.

Chapter 2

The effects of directing visual attention along the horizontal and vertical axis on saccade latency.

2.0 General Introduction.

The following chapter describes five experiments in which subjects were instructed to direct their attention covertly, while eye movement latencies were obtained to unilateral single, and double bilaterally, presented targets. The subject was directed to maintain fixation on a central cross and attend covertly in a certain direction, by a verbal instruction. The direction of attention was either left or right with targets presented on the horizontal axis (experiments 1-4), and up or down with targets on the vertical axis (experiment 5). The task was to make a saccade as quickly as possible, to a target when it appeared. There were two types of targets: single targets which appeared at one of two eccentricities in the attended or non attended direction, or bilaterally presented targets which appeared simultaneously at equal and opposite eccentricities. The targets appeared in an otherwise unstructured visual field in which the target locations were not marked. Targets were presented at two eccentricities in each hemifield, the presentation of which was randomised across trials. Two eccentricities were used to minimise the possibility of preprogramming of the amplitude of the saccade before target onset (Becker and Jürgens, 1979).

The experimental procedure was similar to that used by Hughes and Zimba (1985; 1987) who measured the effects of directing attention on the manual reaction times obtained to targets presented in either hemifield. Hughes and Zimba (1985) suggested that the effect of directing visual attention was to produce a broad area of inhibition for the non attended hemifield. Targets appearing in the non-attended direction are therefore subject to costs, when compared to neutral trials, while targets in the attended direction will not show any benefits on performance. Their work was extended (Hughes and Zimba, 1987), to examine the spatial extent of the inhibitory effect, by directing attention along both the horizontal and vertical axis. The results confirmed that the inhibitory effect operates in the left/right hemifields and also in the upper/lower hemifields. The effects of visual attention are not restricted to left and right spatial coordinates, but also operate on upper and lower spatial representation. The experiments described in this chapter direct attention along the horizontal axis (Exp's 1-4) and the vertical axis (Exp. 5), to examine the inhibitory and facilitatory effect of directing attention above and below fixation, on saccade latency. An uncluttered visual array was used to eliminate increases in costs as observed by Zimba and Hughes (1987) when target locations are marked which affects the subsequent cost/benefit analysis.

Experiment 2. examines the effect of directing attention in a 'gap' paradigm, in which the fixation point went off 100 ms before the target onset using a task otherwise identical to that used in Exp 1. The facilitation effect of fixation point offset observed in gap paradigms (eg.

Saslow, 1967; Ross and Ross, 1980, 1981; Reulen, 1984b) has been attributed to attentional processes (Fischer and Breitmeyer, 1987). The facilitation produced is thought by Fischer, to reflect the time saved in not having to disengage attention from fixation, before it is moved to the target location. This implies a model of attention moving in an analogue way from fixation similar to the spotlight analogy of Posner (1980). An alternative explanation is that active fixation produces a generalised increase of inhibition (within the orienting system) which inhibits a saccadic eye movement in any direction, this could be thought of as being a reluctance to make a saccade produced by active fixation. According to the attentional disengagement explanation the use of a gap paradigm could be expected to produce a greater facilitation effect in neutral trials when the subject is attending to the fixation point, than in valid trials when the subject is covertly attending to the target location. However, if the gap effect reduces the level of inhibition within the saccadic system then the facilitation effect will be equal in both valid and neutral trials.

Experiments 3 and 4 were performed to examine the effects of practice on saccade latency to enable a more detailed discussion of the small benefits obtained on saccade latency by directing attention (exp.'s 1 and 2). The small benefit could have occurred as the neutral blocks were always performed first; so that there were no carry-over affects from attentional, to neutral, blocks. It is possible that the small benefits obtained when comparing the neutral blocks to the attentional blocks in experiments 1 and 2, were due to practice effects which speeded saccade latency over the three blocks of trials. Experiment 3., examined the speeding obtained over three blocks of trials to show if this could explain the benefits obtained. Experiment 4 presented neutral and attentional blocks in a counterbalanced order across subjects so that any facilitation obtained could not be attributed to practice effects.

Experiment 5, examined the effects of directing visual attention vertically in the upper and lower visual fields, with unilateral single and bilateral double targets presented at two eccentricities along the vertical axis. The idea was to examine to spatial distribution of visual attention to show if it is restricted to the horizontal axis, or if it can also be directed along the vertical axis, in two dimensional space. The slowing of saccade latency which has been observed for targets in the lower visual fields (Heywood and Churcher, 1980) was examined by running blocks under both monocular and binocular viewing conditions.

2.1 General Method.

The experiments described in this chapter all use a similar method and procedure, so a general method is described here to avoid repetition. Where there are any differences these are emphasised, or mentioned in the method section of that experiment.

Apparatus.

Stimuli were presented in all experiments, on a Phillips monochrome VDU monitor (P31

phosphor), generated by a BBC Master series microcomputer. Eye movements were recorded using the differential limbus reflection technique, using a binocular infrared system described by Young and Sheena (1975). Experiments 1 and 2, used an ACS model EM130 tracking system, while experiments 3 to 5 used a Skalar IRIS system (described by Reulen et al., 1988). The analogue signal was sampled every 5 ms by a Cambridge Electronic design Alpha computer, with a 502 interface (Exp. 1-2), or an Apple Macintosh II with a Labdriver (Exp's. 3-5). A signal from the BBC microcomputer, synchronised with the raster scan of the display monitor, initiated the analogue to digital conversion as the stimulus appeared on the screen and stopped sampling 500 ms later. The digital records were stored on disc for later examination using a semi-automatic saccade detection programme, described in the appendix section (1). A chin rest was used to limit head movements throughout the test session. Dim background illumination was provided by an Anglepoise lamp, which reduced the effect of the persistence of the stimulus on the display screen, which was apparent due to the slow decay rate of the P31 phosphor.

Timing sequence of stimuli presentation.

The following experiments used either a constantly displayed fixation point (overlap); or, the fixation point went off 100 ms before, the onset of the saccade target (+100 ms gap). The sequence of stimulus presentation in the overlap and +100 gap paradigms is displayed in Figure 1.

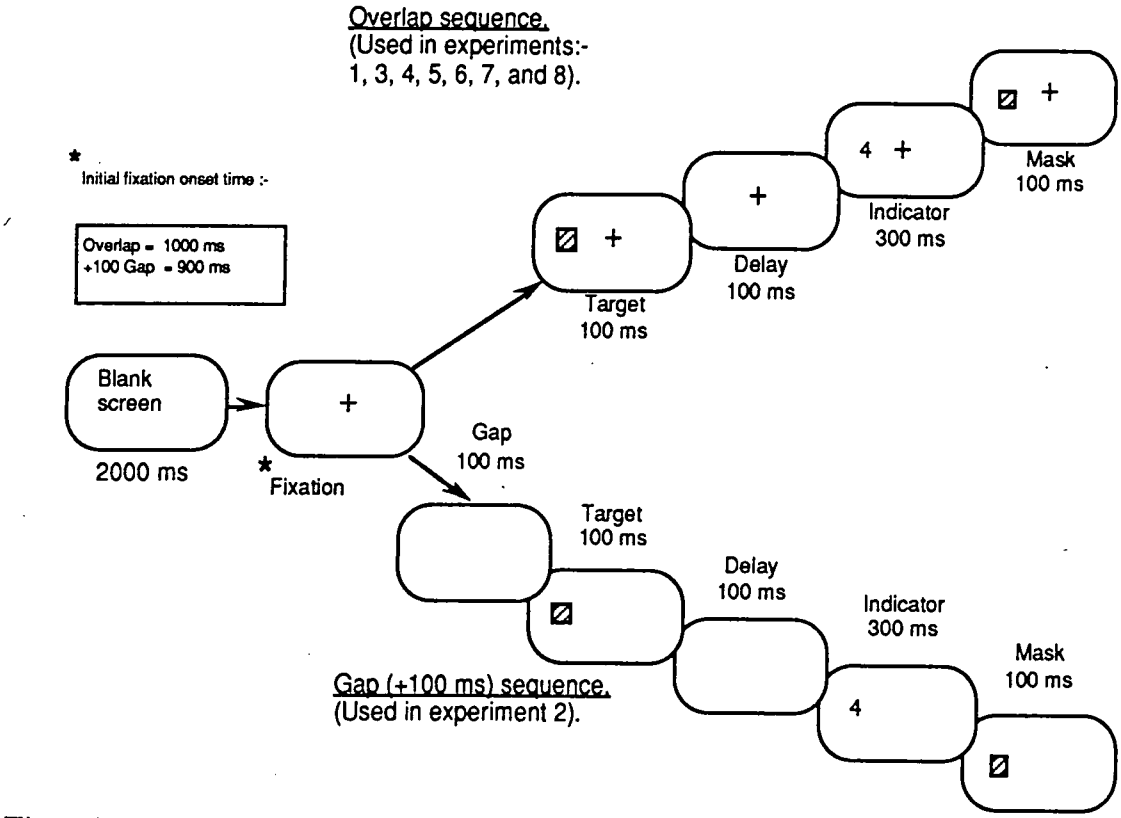


Figure 1.
Sequence of stimulus presentation used in the overlap and +100 ms gap conditions.

At the start of each trial a fixation cross (of 0.57° visual angle), was presented in the centre of the display screen. Saccade targets were squares (sides 0.57° visual angle) presented at 5.5°, or 9.5° eccentricities in both fields. Targets appeared 1 second after initial fixation onset, and were presented for 100 ms. A delay of 100 ms then occurred, during which time the subject would be expected to make a saccadic eye movement. An indicator stimulus consisting of a hollow square (0.57°) containing one or two small dots, was presented at the target location. The dots were sufficiently small so that correct discrimination was only possible under foveal vision. Subjects were required to indicate how many dots they had seen by using a toggle switch. The indicators were presented for 300 ms and were immediately followed by a visual mask consisting of the same stimulus as used for the target, presented for 100 ms.

Procedure.

Subjects were seated 50 cm from the display screen, with their eyes level with the fixation cross and their head supported on the chin rest.

Each subject was tested under three conditions, with 84 trials in each block. Subjects were tested under a neutral condition in which they were instructed to keep their eyes on the central fixation cross while it was presented and to move their eyes as quickly as possible to the target location when the target appeared. The instructions given to the subjects are given below.

General Instructions.

"Please try to keep your head as still as possible on the chin rest and avoid altering your position during eye movement recording. At the start of each trial a small fixation cross will appear at the centre of the screen, following which a small target will appear on the screen. The targets will either be located to the left, or right, of the fixation cross; or, two targets will appear simultaneously left and right of centre. On the double target trials you are free to move your eyes to either target. Your task is to keep your gaze on the central fixation cross and then move your eyes as quickly as possible to the target location when the target appears. You will see one or two small dots inside the target square. Move the switch on the button box left for one dot, and right for two dots. Try to be as accurate as possible a bleep indicates an incorrect response. Return your eyes to the central location ready for the next trial."

Attentional Instructions.

"In the next block of trials you should bias your attention to the left/right sides of the screen. Keep your eyes on the fixation cross as before, but attend to the left/right target location. On bilateral double target trials you will always saccade towards the target in the attended direction."

Subjects were instructed to report the number of indicator dots seen by using a hand held button box on each trial. Accuracy of response was emphasised and an audible bleep occurred if an incorrect response was made. Subjects were also tested under two 'attentional' blocks, in which they were instructed to bias their attention to the left/right side of the screen while maintaining central fixation and then move their eyes to the targets, as before.

Each block of 84 trials contained equal numbers of left single targets, right single targets and bilaterally presented targets, appearing simultaneously at equal and opposite eccentricities. This produces a total of 252 trials per subject which took approximately 40 minutes to complete.

2.2 Experiment 1: Directing attention along the horizontal axis in an overlap paradigm.

2.2.1 Introduction

The following experiment examined the effect of directing attention (by a verbal instruction), with a constantly displayed central fixation point ('overlap' paradigm). The subjects were instructed to attend covertly; either left, or right, of fixation, by a verbal instruction given at the start of each block of trials. The direction of attending remained constant across each block. This procedure was similar to that used by Tassinari et al. (1987) and was used to obtain costs and benefits which were not confounded with the difficulty of interpreting a symbolic cue located at fixation. To obtain an accurate baseline measure of mean saccade latency, subjects were first tested on a 'neutral' block in which there was no instruction to direct attention. Subjects made a saccade to the single target when it appeared, or to either one of the bilateral targets, on double target trials. On bilateral trials in the neutral block, the subject could choose either direction in which to make a saccade. The neutral trials baseline measure, was obtained before the attentional blocks were carried out, so that subjects were not practised at making saccades in a certain direction, which might affect saccade latency to bilaterally presented targets. The use of a neutral block carried out before the experimental blocks is open to the criticism of practice effects facilitating saccade latency, which is important in terms of the cost/benefit analysis of directing attention. It has been shown that the benefits of covertly orienting attention can be small (Hughes and Zimba, 1985, 1987; Rizzolatti et al., 1987; Zimba and Hughes, 1987), so any latency decrease (facilitation) produced by practice effects needs to be examined.

It has been shown that presenting targets bilaterally, produces an increase in saccade latency of some 30-40 ms (Lévy-Schoen, 1969; Findlay, 1983). Two possible reasons for this increase can be suggested. The first is that this increase could be due to the saccadic system having to select a direction in which to make a saccade on bilateral trials, which is not required on single target trials, as saccade direction is defined by the target onset. The attentional instruction should eliminate this response competition for bilateral double targets, as saccade direction will always be in the attentional direction. It would then be expected that the instruction to attend in one direction will reduce the slowing of saccade latency with bilateral target presentation. An alternative explanation to that of conflicting saccade direction, is that a stimulus onset could, as a result of automatic activation of the attentional system, produce an increase of inhibition in the contralateral hemifield, similar to the hemifield inhibition suggested by Hughes and Zimba (1985; 1987). Bilateral target onsets will therefore produce an automatic increase in levels of inhibition for <u>both</u> hemifields with a resulting slowing of saccade initiation (Findlay, 1983). Directing attention in a certain direction will not reduce the inhibitory effect of the contra-attentional bilateral target, so saccade latency would be slowed by an equal amount in both the attentional and neutral trials. The experiments in this chapter all use single and bilaterally presented targets which should enable the two alternative explanations of saccade

direction and attentional inhibition to be compared. The saccade direction explanation predicts that directing attention should reduce the slowing produced by bilateral target presentation. The increase of inhibition explanation predicts no reduction in the cost produced by presenting targets bilaterally.

2.2.2 Method.

Subjects.

The subjects were postgraduate students three female and four male, of ages 24 to 34 years. All were from the Psychology department at Durham, and had normal or corrected to normal vision.

Procedure.

The procedure is similar to that described in the general procedure section. Saccade targets appeared at 5.5° and 9.5° eccentricities, left or right, of fixation. Subjects completed a practice block of some 56 trials, before starting on the neutral (no attentional instruction) block; in which saccade latency was measured to left, right and bilateral double targets. Following the neutral block, subjects completed two attentional blocks in which they were instructed to direct their attention left, or right, of fixation on each trial, and saccades were again recorded to single and bilateral double targets. On all trials the subject responded to the number of dots in the indicator stimulus by using a hand held button box. The next trial occurred after the response had been made, which enabled a 'self pacing' of trials.

2.2.3 Results.

Saccades from each subject were examined by a semi-automatic analysis programme which located the point at which a saccade occurred and calculated the time from target onset, which is the saccade latency. Saccades with latencies less than 70 ms were eliminated as anticipatory, and saccades with latencies over 300 ms were rejected as not being stimulus driven.

The mean saccade latencies obtained to single and double targets, at the two eccentricities, in the neutral, attend left and attend right conditions are shown in Table 1.

Table 1. Mean saccade latencies (in ms) obtained in the neutral and attend left/right conditions, to single and double targets at two eccentricities.

	Bil.9.5°	Bil.5.5°	RSing 9.5°	RSing.5.5°	LSing.9.5°	LSing.5.5°
Neutral	183.7	196.5	167.0	161.1	173.7	170.9
Att. right	176.9	180.2	154.7	159.4	203.3	206.9
Att. left	185.1	180.2	207.9	204.4	162.3	166.1

Bil. = Bilateral double targets.
LSing = Left single targets.
RSing = Right single targets.

It can be seen that the means of saccades made in the left and right direction are similar. The means obtained in the attend left and attend right conditions were combined and collapsed to display the results in terms of the 'attended' and 'non-attended' directions. Mean latency obtained for saccades made to single and bilateral double targets are displayed in Table 2.

Table 2. Mean saccade latencies (in ms.) combining saccades made left and right of fixation, to single and bilateral simultaneous targets at two eccentricities.

	Attended				Neutral				Non-attended	
	Single		Bilateral		Single		Bilateral		Single	
Eccentricity	far	near	far	near	far	near	far	near	far	near
Mean Lat. (ms.)	159	163	181	180	171	166	184	196	206	206
S.Dev.	(30)	(34)	(29)	(32)	(36)	(31)	(33)	(34)	(43)	(43)

Mean saccade latency was found to be unaffected by target eccentricity (see ANOVA below). The means were combined for saccades made to the two targets eccentricities, and are displayed in Figure 2 below, for saccades made in the attended and non-attended direction. The mean latency obtained to bilateral targets is shown for the neutral and attended conditions only, because subjects do not made a contra-attentional movement with bilateral targets.

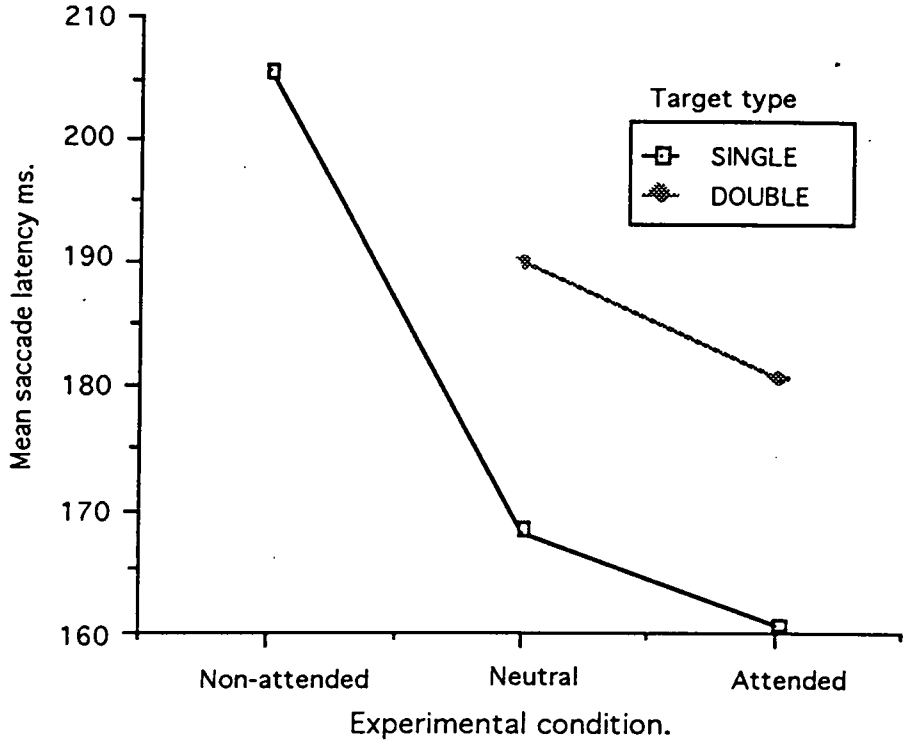


Figure 2.
Mean saccade latency obtained in Experiment 1.
Overlap condition using single and bilateral double targets under attended and neutral conditions.

Figure 2 shows that the mean saccade latency obtained to single targets, in the neutral condition was 168 ms, and the mean obtained in the attentional conditions was 161.1 ms, a small speeding of some 7 ms. The mean latency made to single targets in the non-attended (invalid) direction of 205.6 ms, indicating a substantial slowing of some 38 ms, compared to the neutral mean (161.1 ms). The mean latency obtained to bilateral double targets in the neutral condition was 190.1 ms, a slowing of some 22 ms compared to the mean latency obtained to single targets in the neutral condition. A comparable 19.5 ms slowing was also indicated for the mean obtained to bilateral double targets in the attentional condition.

As there were no invalid bilateral target means, two separate ANOVA's were performed. The first compared the attended, non-attended and neutral, mean latencies obtained with single targets. The second compared the mean latencies obtained to single and bilateral targets in the attended and neutral conditions.

The first (two factor) ANOVA was performed to examine the costs and benefits of directing attention on saccade latency with single targets. The first factor of 'condition' had three levels (attended, neutral and non-attended), while the second factor of 'eccentricity' had two levels (far and near). There was no significant effect of target eccentricity on saccade latency ($F(1,6)= 0.00$ $p < 0.1$). The factor of condition was shown to be significant

(F(2,12)=54.2, p<0.001). A Newman-Keuls post-hoc analysis was performed and showed that the small 7 ms speeding observed in mean latency to single targets, in the attended direction, was not significant. The mean latency obtained to single targets in the non-attended direction was shown to be significantly slower than to single targets in both the neutral (p<0.01) and attended directions (p< 0.01). The increase in mean saccade latency (38 ms cost) shown to single targets in the non-attended direction is therefore significant. The two way interaction between condition, and eccentricity, was not significant (F(2,12)=1.79, p=n.s..).

The second (three factor) ANOVA was performed to examine the effect of bilateral target presentation on saccade latency. The three factors each had two levels: 'condition' (attended and neutral), 'target type': (single and bilateral), and 'eccentricity' (near and far). The main effect of condition was significant (F(1,6)=6.45, p<0.05), so the attended trials mean of 171 ms being significantly faster than the neutral mean of 176 ms. The mean of single targets is 164.5 ms and for bilateral targets 185 ms, which was show to be a highly significant slowing on saccade latency (F(1,6)=61.55, p<0.001). There was no effect of target eccentricity on saccade latency (F(1,6)=0.63 p = n.s..). There was no significant interaction effect between any two factors, but there was a significant three way interaction effect (F(1,6)=15.05, p<0.001). This interaction is seen in Table 2, which shows that a comparable slowing was produced for bilateral targets at both eccentricities, in the attentional condition; but in the neutral condition the near eccentricity bilateral target produced a greater slowing than did the far eccentricity bilateral target.

2.2.4 Discussion.

The results showed that saccadic reaction times to single targets were faster in the attended condition by some 7 ms, a small and non significant benefit, compared to the mean obtained to single targets in the neutral condition. A similar speeding of 9.5 ms is apparent in the mean latency obtained to bilateral targets in the attended condition, compared to the mean obtained to bilateral targets in the neutral condition. The speeding reached significance when means obtained to single and bilateral targets in the attended conditions were compared to those obtained in the neutral condition. Interpretation of this small speeding is difficult as the confounding variable of practice effects cannot be excluded. This arises because the neutral condition was always performed before the attentional condition (see introduction). Experiments 3 and 4 examine this possibility in detail to show if this speeding could arise from practice effects. Alternatively, the small speeding could be interpreted as a small benefit for targets in the attentional direction due to a preprogramming of saccade direction (Findlay, 1983; Rizzolatti et al., 1987). The result could also be explained in terms of the attentional spotlight being oriented to the attended target location by the attentional instruction, which gives a small benefit on saccade latency. This small benefit of performance in the attentional conditions is however, smaller than would be predicted by either of these explanations and is comparable to the small non significant benefit shown in Hughes and Zimba's (1987) manual RT task. The hemifield inhibition model suggested by Hughes and Zimba (1987) does not accommodate for

observed benefits obtained by directing attention so the small benefit obtained in this experiment is worth further examination.

Single targets appearing in the non-attended direction produced a significant costs of some 37 ms on saccade latency. There are various possible explanations to account for this result. The non-attended target will require the saccade direction programme to be modified which could explain the long latencies in a motor response framework without any direct attentional explanation (Findlay, 1983). Alternatively, an attentional explanation could be used to explain the slowing observed on non-attended trials. The attentional instruction is assumed to enable the spotlight to be oriented into the attended hemifield. On invalid trials the spotlight will need to move from its current location, to the target location in the non-attended hemifield. The process of re-orienting the spotlight may take a certain amount of time (Posner, 1980), so targets in the non-attended hemifield will be subject to a cost in terms of saccade latency, due to the re-orienting of attention. Given the close link proposed between the attentional orienting and saccadic orienting system (Shepherd, Findlay and Hockey, 1986) the re-orienting of attention could be expected to increase saccade latency. A second attentional explanation is provided by Hughes and Zimba (1985; 1987) who proposed that visual attention when directed in one hemifield, produces a uniform inhibition for the opposite hemifield. Crossing the vertical meridian will therefore produce an increase in reaction time as this inhibition has to be overcome. The increase in saccade latency for non-attended targets and the small facilitation for attended targets seems to favour the inhibition model of Hughes and Zimba, and the link between the saccade system and attentional system of Rizzolatti et al. (1987).

Bilateral targets in the neutral condition can be seen to have produced slower saccadic reaction times (by 22 ms), compared to single targets. This increase in saccade latency, shown for bilateral targets presented simultaneously in opposing visual fields has been noted previously (Lévy-Schoen, 1969; Findlay, 1983). It is thought that the increase could be due to the requirement of the system to make a decision regarding direction (where system) of the saccade, before a when decision to make the saccade is made. However, the results obtained for bilateral targets in the attentional conditions seems to argue against this view. Saccade latencies for bilateral targets are speeded by 9.5 ms by the attentional instruction (possibly due to practice effects), but are still some 19.5 ms slower than to single targets ($p<0.001$). This is the case even though the subject does not have to make a directional decision, as to which bilateral target they should move to. The subject will in effect always respond to the target in the attended direction. The slowing of mean latency on bilateral target trials could only be explained in terms of a directional conflict for motor planning of the saccade if, the subject does not preprogramme a saccade direction prior to target onset on attentional trials. One reason why the subject may not preprogramme saccade direction on attentional blocks is because a contra-attentional direction movement is required on a third of all trials (to the non-attended single targets). In this experiment it is possible that the saccadic system is not preprogrammed in terms of the direction component, prior to target onset due to non-attended single targets. This possibility is examined in Chapter three by comparing the latencies obtained to single and bilateral double targets without presenting single targets in the non-attended direction.

Findlay's (1983) model of saccade generation includes mutual inhibitory links between the components which initiate a direction computation. Bilateral targets produce bilateral inhibition in the initiate left and initiate right components, which increases saccade latency. If the saccade system and attentional orienting system are closely linked (Shepherd, Findlay and Hockey, 1986), then a similar model may be used to explain the increase in latency for bilateral targets in the attentional conditions. An attentional explanation of the slowing observed on bilateral trials is that the onset of a stimulus in one hemifield produces an increase of inhibition acting on the contralateral orienting system. On bilateral trials there will be an automatic increase in the level of inhibition on both of the movement components, regardless of where the person is already attending. This has the effect of inhibiting an attentional movement to either of the bilateral targets and increases the latency of the resulting saccade. The hemifield inhibition model of Hughes and Zimba does not explain why bilateral targets should increase saccade latency in the attentional condition.

Bilateral targets at the near eccentricity location produced a greater slowing on mean latency than did the far eccentricity bilateral targets (see: Table 2); as indicated by the significant three way interaction effect. This could reflect a greater inhibitory effect being produced by a stimulus being presented in the nasal hemifield, than is produced by a stimulus in the temporal hemifield. It may also be due to a greater inhibitory effect for a target presented close to the fovea than for targets in the periphery. It will be of interest to see if this is a robust effect in the following experiments.

One further factor to consider regarding the slowing produced on bilateral trials, is the target presentation timing sequence. On all trials the indicator stimulus appears at the saccade target location 200 ms, following initial target onset, so on bilateral trials an indicator appeared in both hemifields exactly 200 ms after the initial onset of the saccade target. It is possible that the onset of the indicator is responsible for the increase in saccade latency, possibly by cancelling the programme of a saccade which is about to be made. However, the mean latency observed to single targets was shown to be some 159-171 ms, which suggests that the indicator onset will not produce the extra slowing on bilateral trials. Given that the standard deviation (Table 2) is comparable for single and bilateral target trials this seems to be an unlikely explanation. The possibility that the bilateral target slowing is produced by the late indicator onset is examined in experiment 3.

The next experiment is performed to examine the effect that prior fixation offset has on the latency of saccades made to single and bilateral targets. Prior fixation offset has been shown to speed saccade latency, which has been attributed to attentional factors (Fischer and Breitmeyer, 1987). It is of interest to show if this facilitation effect will speed saccade latency under conditions in which visual attention has been voluntarily oriented as occurred in experiment 1.

2.3 Experiment 2: Directing attention along the horizontal axis in a gap paradigm.

2.3.1 Introduction.

The results obtained in experiment 1 showed that directing visual attention produced a small benefit for targets in the attended hemifield, with a much larger cost for targets in the non-attended hemifield, on saccadic reaction times. A possible explanation of these results centres on the idea that the verbal instruction (cue) enables subjects to orient attention to the target location on attentional trials, but in the neutral trials attention has to be disengaged and then moved from fixation to the target location. A certain amount of time is assumed to be required on neutral trials for disengagement from either fixation and a movement to be made to the target location. On attentional trials attention is assumed to be aligned with the target location resulting in faster saccadic reaction times being made than is the case in neutral trials. The large cost shown for non-attended targets, could be explained within a similar framework. A target appearing in the non-attended hemifield will require attention to be disengaged from the cued location (in the attended hemifield) and then moved to the non-attended target location (in the opposite hemifield). This process of disengagement and movement from one hemifield to another may be responsible for the increase in saccadic reaction time. The disengagement hypothesis of the costs and benefits can be further tested by using a 'gap' procedure that is thought to manipulate attentional disengagement by removal of the central fixation point (eg. Fischer and Breitmeyer, 1987).

The use of prior fixation offset facilitates saccadic reaction times, which it has been suggested could be due to prior attentional disengagement (Fischer and Breitmeyer, 1987). Prior fixation offset should disengage attention only if it is engaged at the fixation point location. If in experiment 1 attention has been oriented to the target location on attentional trials, then fixation offset should not produce any facilitation effect as attention is not coincident with the fixation point. The use of prior fixation offset should therefore produce a differentially speeding effect for the latencies obtained to targets under the neutral and attentional conditions. In the neutral trials the latency to single and bilateral targets should obtain equal facilitation, due to attention being (at least partly) disengaged before the onset of the target. In the attentional trials there should not be any speeding of latency with prior fixation offset when the targets appear in the attended direction, as attention is assumed to have been moved from the fixation point to the target location. Targets appearing in the non-attended direction should also be unaffected by prior fixation offset, as attention has to be disengaged from the attended hemifield location and moved to the non-attended target location.

The present experiment examined this possibility by repeating experiment 1, but in this instance the fixation point was extinguished 100 ms before target onset. Two attentional conditions (attend left, attend right) and a neutral no instruction condition were again used.

2.3.2 Method.

The method, stimuli and apparatus were identical to those described above. The stimulus timing sequence in this experiment is different and is described in the timing sequence section below. Each subject was first tested in the neutral condition and then in the two attentional conditions.

Subjects

The subjects were: seven postgraduate students who had all been used in the overlap experiment and one other (the author RW) who had not been used before.

Timing sequence:-

The fixation point was initially displayed at the start of each trial for 900 ms and was then extinguished (Figure 1). A delay of 100 ms then occurred during which time the screen remained blank following this delay the targets appeared. The time between the initial onset of the fixation point and the onset of the target was 1 second, the same as occurred in experiment 1. The targets were followed by the indicators and mask in the same temporal sequence as in experiment 1. An inter trial delay of 2000 ms was used to reduce the problem of after image persistence of targets, on the VDU screen.

2.3.3 Results.

Saccades of latency of less than 70 ms and of latencies of over 300 ms, were excluded from the analysis. The mean saccade latency obtained to single and bilateral targets at the two target eccentricities, in the neutral and attentional blocks; are shown in Table 3.

Table 3. Mean saccade latency obtained to single and bilateral targets in the neutral and attentional conditions.

	Attended				Neutral				Non-attended	
	Single		Bilateral		Single		Bilateral		Single	
Eccentricity	far	near	far	near	far	near	far	near	far	near
Mean Lat. (ms.)	139	137	164	161	148	148	181	175	178	177
S.Dev.	(28)	(27)	(36)	(33)	(29)	(33)	(45)	(49)	(45)	(42)
Speeding (Exp 1 - Exp 2.)	23 ms		18 ms		20 ms		12 ms		28 ms	

The mean saccade latencies can be seen to be comparable for the two target eccentricities, so the means were combined and displayed graphically in Figure 3.

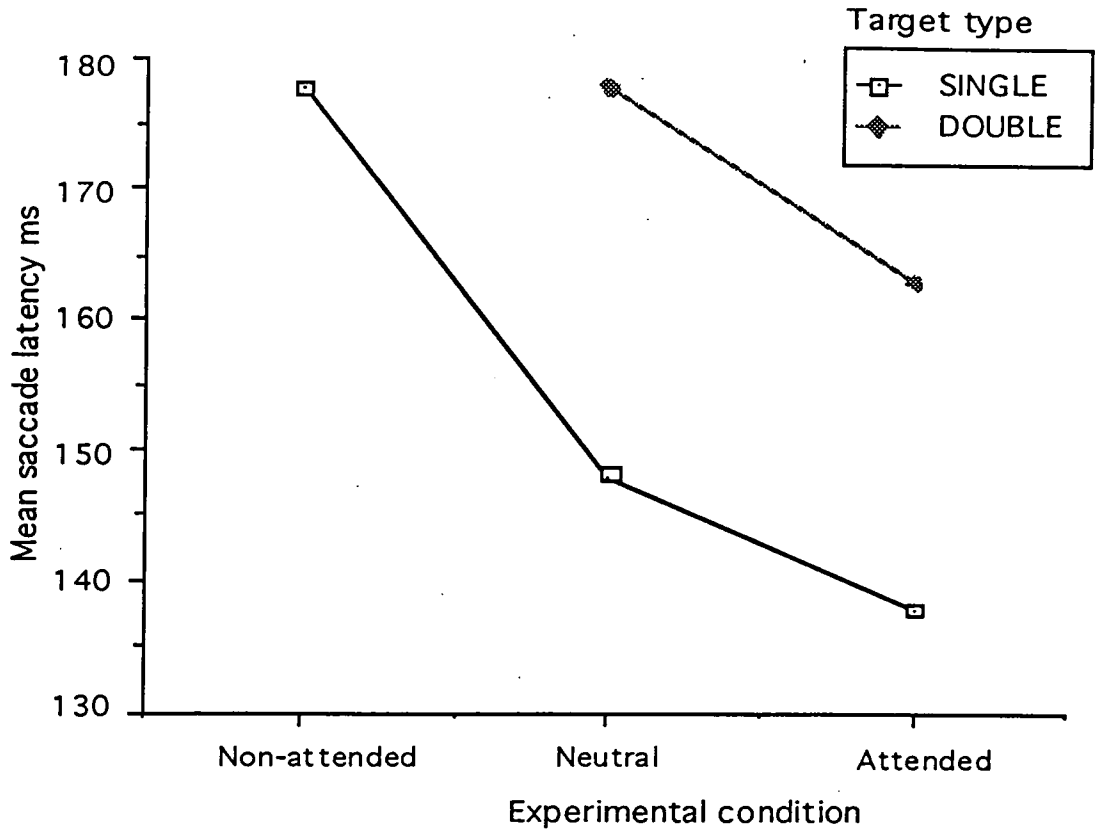


Figure 3.
Mean saccade latency obtained in Experiment 2.
+100 ms gap condition using single and bilateral double targets under attentional and neutral conditions.

Figure 3. shows that a similar pattern of results has been produced in the gap experiment, as was shown in the overlap experiment. The use of the gap paradigm produced faster mean saccade latencies than was observed in experiment 1. A comparison of the means produced in the gap and overlap experiments, shows that single target latencies in the neutral condition are speeded by 20.3 ms, and bilateral targets by 12.4 ms. In the attentional condition single and bilateral targets are speeded by 23 ms and 17.9 ms respectively. The non-attended single target is speeded by 27.7 ms indicating a general facilitation effect of fixation point offset for targets in all conditions.

The mean latencies obtained to single targets at two eccentricities in the neutral and attentional blocks were compared using a (two factor) ANOVA, as performed on the data in experiment 1. The factor of eccentricity was not significant ($F(1,6)=0.19$ p= n.s.), confirming that saccade latency is not affected by target eccentricity. The main effect of condition was significant ($F(2,12)=32.16$ $p<0.001$). Single targets in the attentional direction show a benefit of 10 ms compared to the neutral condition, while single targets in the non attended direction show a cost of 29.5 ms. A Newman-Keuls post-hoc analysis showed that latencies to targets in the attended direction were not significantly faster than made in the neutral condition ($p>0.05$), but mean latencies obtained to targets in the non-attended direction were significantly slower

than saccades made in the neutral condition ($p<0.01$). There was no significant interaction effect between condition and eccentricity ($F(2,12)=0.03$ $p > 0.1$).

The second (three factor) ANOVA was performed to compare the means obtained to single and bilaterally presented targets, at two eccentricities, in the valid and neutral condition. Saccades made to targets in the attentional condition are 12 ms faster than in the neutral condition, this difference was shown not to be significant, although it was approaching significance ($F(1,6)=4.76$ $p=0.072$). There was no effect of target eccentricity ($F(1,6)=0.69$ $p >0.1$). Bilateral targets have produced a significant slowing on saccade latency, the single target mean is 143 ms and the bilateral target mean 170.5 ($F(1,6)=1.18$ $p=0.003$). There were no significant two way interaction, and in contrast to the overlap experiment, the three way interaction was also not significant. In the gap experiment bilateral targets have produced an equal slowing effect in the neutral and attentional condition, which is not effected by target eccentricity.

2.3.4 Discussion.

The use of the gap condition, can be seen to have produced mean saccade latencies which are at the fast end of the distribution of human saccade latencies (Fischer and Breitmeyer, 1987). This is most likely to reflect the use of young well practised subjects and the speeding effect due to the gap interval of 100 ms, used throughout each block, which would enable a 'pacing' of responses. Mean saccade latencies are reduced in the gap condition by 12.4 to 27.7 ms, when compared to those produced in the overlap experiment. The attentional instruction produced a 9.5 ms speeding (benefit) on the latency obtained to single targets in the attended direction and a 15 ms speeding of latency for bilateral targets. This uneven facilitation between single and bilateral targets in the gap condition, could result from ceiling effects. The latency produced to single targets under the gap condition is already very fast (148 ms) and may not obtain much greater benefit from the attentional instruction. The use of the attentional condition has produced a large cost (27.7 ms) for single targets in the non-attended direction, although this cost is slightly reduced when compared to that produced in the overlap experiment.

The gap paradigm produced a speeding effect on saccade latency, for saccades made under the attentional and neutral conditions, compared to the means in the overlap experiment. This generalised speeding effect cautions against a 'spotlight' attentional explanation (Posner, 1980), being applied to explain the costs, and benefits, obtained of directing attention to a cued location. If attention has been 'aligned' with the target location by the attentional instruction, then there should not be any facilitation of saccade latency by prior fixation offset, in the attentional conditions. Only saccades made to targets under the neutral condition should obtain any facilitation of latency, due to attention being disengaged from fixation. Given that an equal size facilitation effect was observed on saccade latency in both the attentional and neutral conditions; the explanation of the costs and benefits in terms of, the 'prior' alignment of

attention with the target location, is weakened.

The finding of a large cost on saccade latency with targets in the non-attended hemifield, with a small non significant benefit on saccade latency to targets in the attended hemifield, supports the 'hemifield inhibition theory' of attention (Hughes and Zimba, 1985; 1987). They proposed that orienting attention to one hemifield produces inhibition within the 'non-attended' visual quadrants. Their model predicts little benefits on RT performance, for targets in the attended direction, but large costs for targets in the non-attended visual quadrants (as was shown in this experiment and experiment 1). If prior fixation point offset affects an early stage of the orienting system, then the gap situation should speed saccade latency, to both single and bilateral targets, in the attentional and neutral conditions, by an equal amount. The assumption is that maintaining active fixation produces an inhibition in the saccade orienting system. Prior fixation point offset reduces this level of inhibition and facilitates the production of the saccade. The attentional instruction used in this experiment produces an increase of inhibition within the attentional orienting system inhibiting a movement into the non-attended hemifield. The inhibition produced by the attentional instruction is independent of that produced by the saccadic system to maintain fixation and gaze on the fixation point. The use of the gap condition will therefore facilitate saccade latency regardless of the state of the attentional system, as was shown in this experiment.

The models of the gap effect suggested by Ross and Ross (1980; 1981), Reulen (1984b), and Reuter-Lorenz (1991), could be applied to the generalised facilitation effect obtained in this experiment for fixation offset. Ross and Ross (1980; 1981) showed that the prior offset and onset of fixation, both speeded saccade latency, but the greatest facilitation was obtained in the offset condition. This suggests that part of the effect of the gap paradigm is due to a warning signal effect that speeds the ongoing saccadic programme. The onset of the fixation point also produces a warning signal, but its effects are reduced by the presence of inhibition produced by the onset of the fixation point. Tam and Stelmach (in press) have also suggested that part of the gap effect is due to a warning signal effect. The reduction in saccade latency is assumed to reflect both disengagement of the attentional system and of an ocular system, that is required to maintain the eyes gaze at the fixation location. Reuter-Lorenz (1991) suggested that the gap effect is produced by a reduction of inhibition produced by active fixation which is specific to the oculomotor system. The conclusion is that fixation point offset appears to effect an early level of processing or programming, which is not influenced by higher level attentional factors such as sustained attentional orienting. The warning signal, and the reduction of ocular sampling explanations, are both compatible with the generalised facilitation effect obtained in neutral and attentional blocks, of this experiment.

Saccades made to bilateral targets under the gap condition, produced a slowing of saccade latency (of 25-30 ms), compared to latency obtained to single targets. The slowing produced on bilateral presentation was reduced by some 15 ms in the attentional trials when compared to bilateral targets in the neutral trials. Prior fixation point offset in the gap condition did not reduce the size of the slowing of saccade latency to bilateral targets. This slowing could

be due to the bilateral targets activating a transient component of attention, which bilaterally increases the level of inhibition within the attentional orienting system. The process of initiating an attentional movement in either direction, will have to overcome this extra level of inhibition which is not apparent for single targets. The premotor theory of attention (Rizzolatti et al., 1987) would predict that bilateral targets would slow saccades, by producing an extra decision making requirement on the oculomotor system. Experiment six examines this possibility by using single targets in the attended direction only. The oculomotor system will therefore always respond in one direction to single and bilateral targets. The increase in latency shown for bilateral targets should be substantially reduced if a premotor conflict of saccade direction explanation, accounted for the slowing of saccades to bilateral targets.

An interesting observation concerns the three way interaction (condition, target type, eccentricity), which was significant in the overlap experiment, but not in the present experiment. The interaction effect occurred in the overlap experiment, due to an extra slowing on mean saccade latency shown for the near eccentricity bilateral targets, which occurred in the neutral condition only. In the gap experiment the mean saccade latency was greatest for the far eccentricity bilateral targets, in the neutral condition, (opposite to that shown in the overlap experiment). This trend was not sufficient in the gap experiment to produce a significant three way interaction. There seems to be little obvious explanation why there should be an extra slowing of saccade latency for the near bilateral target, in the overlap experiment, which is shown in the neutral condition only. It is even more difficult to account for this result given that it was not shown in the gap experiment, which revealed the opposite eccentricity effect. It will be of interest to see if this interaction is apparent when the overlap experiment is replicated in experiment 4.

The next experiment was performed as a control of experiments 1 and 2. It aims to examine the effects that practice has on saccade latency, to see if practice effects could account for the small benefits which were observed in the attentional conditions. The slowing of saccade latency on bilateral target trials is also examined further by presenting targets without any indicators. This is to show that the slowing of latency observed on bilateral target trials, is not due to the onset of the indicator in the opposite hemifield.

2.4 Experiment 3: Practice effects and a further examination of the slowing produced by bilateral target presentation.

2.4.1 Introduction.

This experiment was performed as a control for experiments 1 and 2. In experiments 1 and 2 the neutral (baseline) blocks, were carried out before the two attentional blocks. This was because it was thought that it may not be possible to obtain a true baseline measure of saccade latency on bilateral double target trials, if the subject had already completed an attentional block, (as subjects may always saccade in the direction in which they had previously directed attention). The small speeding of saccade latency shown on the attentional blocks, could therefore be confounded with practice effects. The present experiment was designed to investigate this possibility by examining any speeding of saccade latency that occurs over three blocks of trials. The speeding of saccade latencies over repeated testing sessions has been illustrated by Findlay and Crawford (1983). They showed a reduction in saccade latency over five days of practice sessions for single subjects. Saccade latency was reduced from approximately 180 ms. to 150 ms. by the second session and fell still further to about 140 ms. by the fifth session. It is highly likely that the speeding of some 8 ms between the neutral and two attentional blocks observed in the overlap and gap experiments, could be due to practice effects and not due to attentional orienting.

The second aim of this experiment was to examine the possibility that the slowing of saccade latency on bilateral presentation could be due to the abrupt onset of the indicator stimulus in the contralateral hemifield. The indicator onset occurred 200 ms after the target onset and could disrupt the programme of any saccades not initiated by 200 ms. This was thought to be an unlikely explanation, as the mean latency shown in experiment 2, was approximately 150 ms, and there was no sign of the standard deviations being greater on bilateral double target trials. The present experiment controls for the possibility that saccade latency was increased due to the onset of the indicator on bilateral trials, by presenting saccade targets without any indicators, or masks.

In the present experiment targets were presented for 200 ms (instead of the 100 ms used previously), so that saccades were not made to a blank screen location. This would occur as the time taken to initiate a saccade is over 100 ms, which would result in the saccade being made to a blank screen location in the absence of the indicator onset, a factor which might affect the results obtained. One subject was tested with targets presented for 100 ms to check that increasing the target onset time does not affected the saccade latencies obtained to bilateral double targets.

To examine any speeding on saccade latency, due to practice effects, this experiment used three blocks of 84 trials, under 'neutral' conditions (no attentional instructions). Single and bilateral targets were presented left and right of fixation in an overlap paradigm. There were no

'non-attended' (invalid) single targets as attention was not directed by an attentional instruction. The aim was to show if saccade latency was speeded over three blocks and to show if the slowing of latency observed on bilateral double targets occurred in the absence of an indicator onset.

2.4.2 Method.

Subjects.

Nine postgraduate subjects from the university of Durham were used as subjects. One further subject (RW) was used with targets presented for 100 ms. Four of the subjects had been used in experiments 1 and 2, while five had not been used before. All subjects had normal or corrected to normal vision.

Apparatus, Stimulus and Procedure.

The experiment used a modified version of experiment 1 and used a constantly displayed fixation point (Figure 1). The apparatus used was identical to that described for experiments 1 and 2. The timing sequence of stimuli was as follows: targets were presented 1000 ms after the initial onset of the fixation point and were presented for 200 ms (except RW targets presented for 100 ms). There were no indicators, or mask, following the offset of the target. Subjects were instructed to move their eyes to the target location as quickly as possible, and then to press a button when their eyes were at the target location. Pressing the button initiated the next trial and ensured that the same self pacing of trials occurred as in the previous experiments. A two second delay occurred before the next trial, following the button press. Subjects were tested on three neutral blocks, with 84 trials in each block. These blocks were carried out immediately after each other, as had been the case in experiments 1 and 2.

2.4.3 Results.

Saccades of less than 80 ms were excluded as being anticipatory, saccades over 300 ms were excluded as not being stimulus driven. The mean saccade latencies obtained to single and bilateral targets (combining latency to the two target eccentricities), in the three neutral blocks are shown in Figure 4.

The mean saccade latencies obtained in block 1 were compared to the combined mean latencies from blocks 2 and 3. The means from blocks 2 and 3 were combined, so as to perform a similar test to that carried out in the overlap and gap experiments (ie. one neutral block vs. two attentional blocks). Another reason for combining the means of blocks 2 and 3 is that the mean of these two blocks are highly similar with most of the speeding being observed in block 2, with little further improvement in block 3. The mean saccade latencies in block 2 show a speeding of some 7 ms for single and bilateral targets.

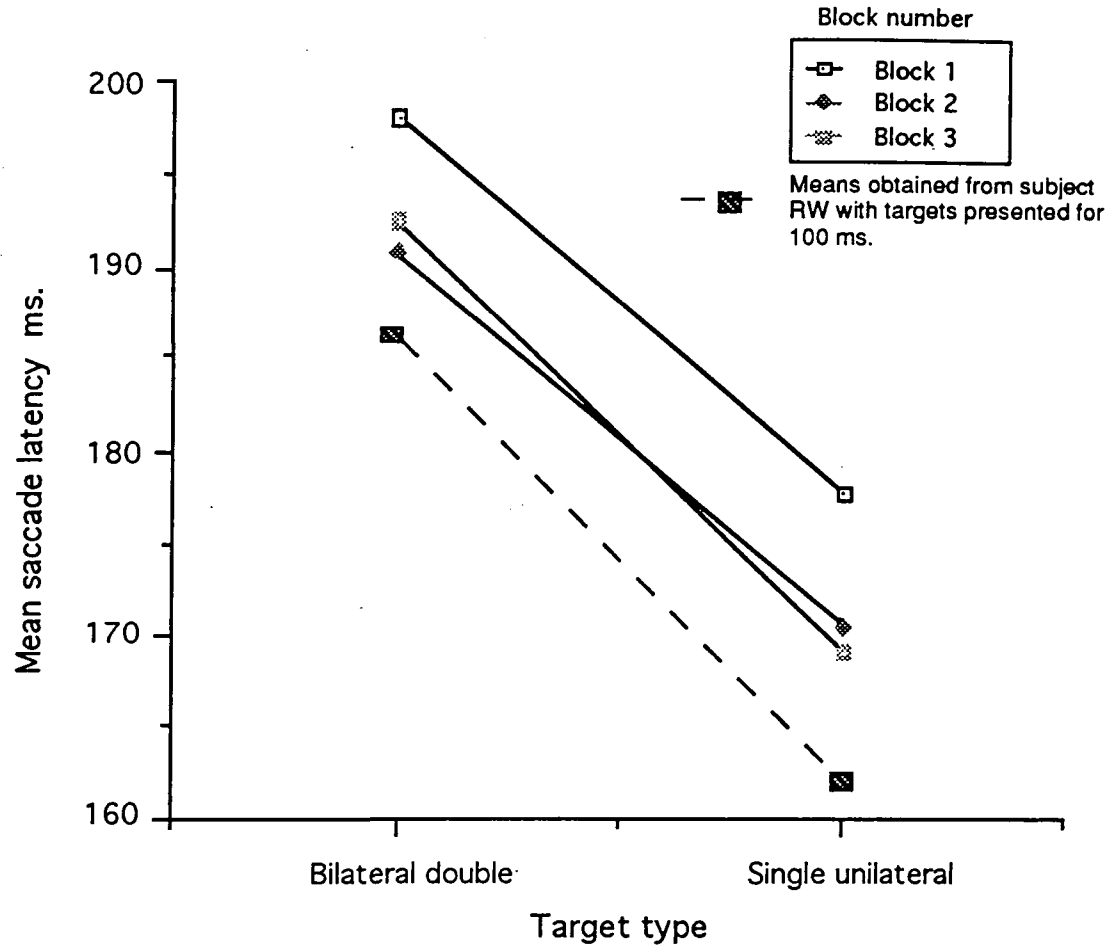


Figure 4.
Mean saccade latency obtained in Experiment 3.
The effect of practice on saccade latency using:
three blocks of trials in an overlap condition.

The results obtained from the single subject (RW) with targets presented for 100 ms are also displayed in Figure 4. The mean latency obtained from this subject confirm that the slowing of latency is observed on bilateral double target trials when targets were presented for 100 ms. This controls for the presentation of targets for 200 ms, with the other subjects.

A (two factor) ANOVA was performed comparing the means obtained to single and bilateral targets in block 1, to those obtained in blocks 2 and 3 combined. The analysis showed that there was no significant effect of block indicating that the 7 ms speeding observed is not significant ($p>0.1$). The small speeding effect observed could be accounted for by practice effects over repeated blocks. The mean to single targets was 173.8 ms and to bilateral targets 194.8 ms, which was shown to be highly significant ($p<0.0001$). There was no significant interaction effect between target and block.

2.4.4 Discussion.

Previous experiments (experiments 1 and 2) have shown a small speeding of mean latency (7 to 10 ms) for saccades made to targets in the attended direction, compared to the means obtained in the neutral block. This speeding could not easily be accommodated in terms of the hemifield inhibition hypothesis of Hughes and Zimba (1987). Inhibition of the non-attended hemifields should produce costs for targets appearing in the non attended field, without any benefits for targets in the attended direction. The present experiment has shown that a 7 ms speeding of mean saccade latency is obtained over three blocks of trials, indicating that practice effects could be sufficient to account for this speeding.

The second aim of this experiment was to show that the onsets of the indicators in the non attended direction were not responsible for the slowing of latency on bilateral trials. The use of saccade targets without any following indicators, or masks, have produced a comparable slowing on mean saccade latency of 25-30 ms on bilateral trials. The slowing of saccade latency does not appear to result from the onset of the indicator (200 ms) after target onset, which could interfere with the programming of saccades not initiated by 200 ms. However, this experiment has not ruled out the possibility that the increase of saccade latency on bilateral double target trials is due to a conflict of saccade direction. The present experiment used targets presented under neutral conditions, so on bilateral target trials a decision must be made to select one of these to make a saccade towards. The previous experiments indicated a similar slowing on saccade latency occurred on bilateral double target trials under the attentional conditions when the direction decision has been reduced. The presence of contralateral (non-attended) single targets on a third of all trials may have resulted in subjects not full programming a saccade direction in the attentional condition. The possibility that the slowing on saccade latency is due to a conflict of saccade direction is further examined in Experiment 6 (Chapter three).

It could be argued that the use of three neutral blocks in this experiment may have affected the subjects response criteria. If subjects use a more conservative response strategy on neutral blocks of trials then it could be expected that a smaller facilitation effect would be observed over three neutral blocks than would be expected in the attentional blocks. The use of well practised subjects could also be criticised as being likely to produce a smaller than expected facilitation effect over repeated blocks. This experiment presented targets for a longer interval, without any indicators, which is a further departure from the original experiments. Given that practice effects are a factor to consider, it is worth repeating the original overlap experiment with a small number of naive subjects, using two neutral and two directed attention blocks of trials and randomising the order of block presentation. Experiment 4 is a replication of experiment 1, using four subjects, with randomised orders of neutral and attentional blocks to examine this possibility.

2.5 Experiment 4: Directing attention along the horizontal axis counterbalancing the order of neutral and attentional blocks.

2.5.1 Introduction.

Experiment three showed that the small speeding effect found for targets in the attentional blocks might be accounted for by practice effects. The present experiment examined this possibility by repeating experiment 1, (using naive subjects) and blocks of neutral and attentional trials, presented in a counterbalanced order. Two neutral and two attentional blocks were used so that the same number of neutral and attentional trials are performed. The presence of a small benefit on saccades in attentional blocks is worth further examination as it is difficult to explain in terms of the hemifield inhibition model of visual attention, which can very conveniently be used to explain the large costs obtained for contra-attentional targets.

2.5.2 Method.

Subjects.

The subjects were two male and two female undergraduates from the Psychology department at Durham of age 21 to 30 years. All had normal or corrected to normal vision. None of the subjects had been used in any eye movement experiments before and were all naive as to the nature of the task.

Procedure.

The apparatus, stimulus presentation sequence and procedure were identical to those used in experiment 1. The fixation point remained on throughout each trial in an overlap paradigm. A Skalar (IRIS) system was used to record the eye movements and an Apple Macintosh II was used to perform the analogue to digital conversion. Subjects were tested on two neutral and two attentional blocks of trials to give equal numbers of trials in each condition. The order of presentation was counterbalanced using an ABBA-ABAB design.

2.5.3 Results.

Table 4 shows the mean latencies obtained to single and bilateral targets at the two eccentricities in the attentional and neutral blocks.

Table 4. Mean saccade latency to single and bilateral targets (two eccentricities) with randomised blocks of attentional and neutral trials.

	Attended				Neutral				Non-attended	
	Single		Bilateral		Single		Bilateral		Single	
Eccentricity	far	near	far	near	far	near	far	near	far	near
Mean Lat. (ms.)	161	164	192	184	175	165	183	187	209	195
S.Dev.	(35)	(38)	(43)	(42)	(45)	(36)	(36)	(42)	(53)	(48)

The combined mean saccade latencies obtained from the four subjects are shown in Figure 5. The graph shows a similar pattern of results to those obtained in experiment 1. Mean saccade latency to single targets in the attended direction, shows a small benefit (of 7.7 ms) compared to the neutral condition mean latency. Mean latency to single targets in the non-attended direction shows a much larger cost (of 32 ms).

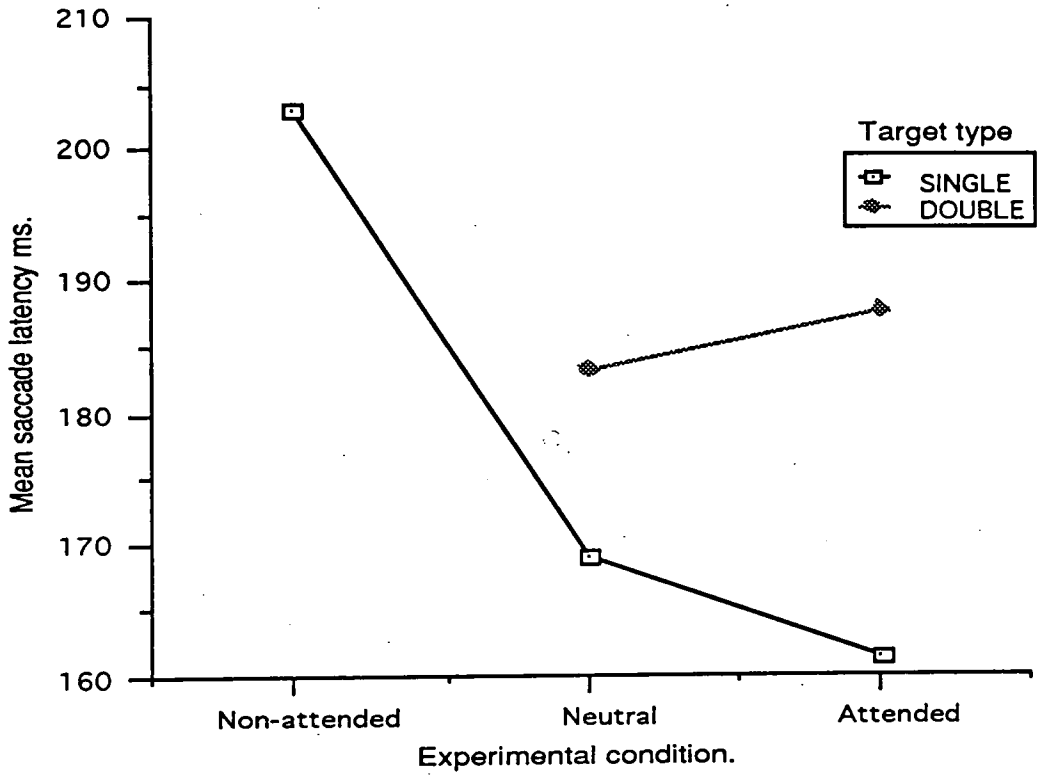


Figure 5.
Mean saccade latency obtained to single and bilateral double targets with randomised blocks of attentional and neutral trials.

A (two factor) ANOVA compared the mean latencies obtained to single targets in the neutral, attended and non-attended conditions and showed a significant effect of attentional condition ($F(2,6)=20.04$ $p < 0.001$). A Newman-Keuls post hoc analysis, confirmed that latencies to targets in the non attended direction were significantly slower than in the neutral condition ($p<0.01$), while latencies to targets in the attended direction were not significantly faster than in the neutral condition ($p>0.05$). There was no significant effect of target eccentricity on saccade latency, and the two way interaction effect was not significant.

A second (three factor) ANOVA was performed to compare the mean latencies obtained to single and bilateral targets, in the neutral and attentional conditions. Bilateral targets were again shown to produce a significant slowing on saccade latency ($F(1,6)=62.92$ $p < 0.001$). There was no significant effect of target eccentricity on saccade latency and in contrast to experiment 1, no significant two or three way interaction effects.

2.5.4 Discussion.

This experiment served as a control for the experiments 1 and 2, which could be criticised on grounds of practice effects. The use of randomised attentional and neutral blocks in this experiment has again produced a similar pattern of results to those shown in the previous experiments. The mean latency of saccades to single targets in the attentional blocks is speeded (by 7 ms) compared to the mean obtained in the neutral blocks, but this is not a significant speeding effect. The reduction in saccade latency in the attentional condition has been shown on all of the previous experiments and cannot now be attributed simply to practice effects. It thus appears that directing visual attention does produce a small facilitation effect on saccade latency. Single targets in the non-attended hemifield are slowed by some 32 ms which is significant compared to the neutral condition. This confirmed the results of the previous experiments, and adds further support to the suggestion that the primary effect of directing attention is to produce inhibition for the non-attended locations.

Mean saccade latency obtained to bilateral double targets was slowed by some 20 ms compared to the latency obtained to single targets, which has also been shown in all of the previous experiments. One difference in the results of this experiment to those obtained in the previous three, is that mean saccade latency obtained to bilateral targets in the attentional condition is slower than the mean saccade latency in the neutral condition. Previous experiments have shown some facilitation was obtained from directing attention with saccades made to bilateral targets. There seems to be no obvious reason why this has not been shown in this experiment. It does however, highlight the strong inhibitory effect that bilateral targets have on saccade latency, which is not reduced in the attentional blocks when the conflict of saccade direction is removed. In the neutral condition of the present experiment there is some evidence of an extra slowing on saccade latency with bilateral targets at the near eccentricity location. This additional slowing is not great enough to produce a significant interaction effect. Furthermore, experiment 1 showed a greater slowing effect of the far eccentricity double target on mean saccade latency than the near eccentricity target. This extra slowing was sufficient to produce a significant three way interaction effect. The failure to replicate this finding in the present experiment (which is a replication of experiment 1) casts doubt on the robustness of this finding.

The inhibitory effect of presenting a target in the contralateral hemifield on saccade latency is worth further investigation and is examined in Chapter three. The next experiment examines the effect of directing attention along the vertical axis in an 'overlap' fixation situation. Single and bilateral targets will again be used to further examine the spatial distribution of directed visual attention.

2.6 Experiment 5: Directing attention along the vertical axis, under binocular and monocular viewing conditions.

2.6.1 Introduction.

In this experiment attention was directed along the vertical axis, with a constantly displayed fixation point (overlap). The previous experiments in this chapter have shown small benefits on saccade latency if attention was voluntarily directed to one side of space on the horizontal axis, with much greater costs on saccade latency when targets appeared in the non-attended hemifield. Saccade latency was also increased when two targets appeared bilaterally and simultaneously left, and right, of fixation. This slowing was also observed in the attention blocks. A possible explanation of the slowing observed with bilateral targets is that they produce an automatic increase of inhibition acting on the contralateral hemifield. This slowing could reflect an automatic inhibitory process of visual attention, rather than an extra programming requirement of selecting the direction to make an overt eye movement. It is of interest to show the spatial nature of the inhibitory effect of directing attention, to see if it operates in terms of the left and right hemifields only, or to show if the same effect is obtained in the upper and lower hemifields.

Rizzolatti et al. (1987) directed attention covertly, along the horizontal and vertical axis, in a manual reaction time experiment. A similar pattern of results was obtained when attention was directed vertically as was shown when it was directed horizontally. A small benefit on RT's was shown for targets in the attended location and a larger cost on RT's for targets in non-attended locations. The cost on RT's was greatest if the non-attended target was in the opposite hemifield, which required an attentional crossing, of either the horizontal, or vertical, meridian. Rizzolatti et al.'s hypothesis was that the extra cost reflected the link between the attentional and oculomotor systems, in what they termed a 'premotor' model of attention. The large costs on RT's shown for crossing a meridian, being due to the cancellation of an oculomotor programme to make a saccade in the attended direction, which is cancelled when the target is in the opposite direction. The costs and benefits obtained by Rizzolatti et al. (1987) are similar to those obtained by Hughes and Zimba (1987). Hughes and Zimba (1987) showed a large cost on RT's when attention crossed either the horizontal, or vertical, meridians. However, they used a different explanation to that of Rizzolatti et al. to explain these findings and concluded that there are two broad inhibitory distributions: one between the left and right hemifields, and one between the upper and lower hemifields.

If visual attention operates on both the left/right, and upper/lower hemifields to an equal extent, as suggested by Hughes and Zimba (1987) then it should produce a similar pattern of costs and benefits on saccade latency, as was shown when attention was directed along the horizontal axis (experiments 1-4). Rizzolatti et al.'s 'premotor' model would also predict a similar pattern of costs and benefits being produced on saccade latency as occurred with manual RT's, but this would be thought to reflect the preprogramming of a saccade on

attentional trials. The present experiment aims to examine the pattern of costs and benefits obtained on saccade latency, but does not aim to separate the 'premotor' and 'hemifield inhibition' explanations.

A confounding factor for measuring vertical saccades is that saccades made in the down direction are often found to be slower than saccades made in the up direction (Heywood and Churcher, 1980; Honda and Findlay, 1992). There is no known anatomical explanation for this slowing of saccades in the lower visual field and it is not shown by all subjects. One possible explanation is that it could reflect an interaction with vergence eye movements. Honda and Findlay (1992) suggested that objects in the lower visual field are usually closer to the observer than objects in the upper visual field. So targets in the lower visual field will require a vergence movement to enable foveation. This mechanism may operate in cases where targets do not differ in real depth for targets in the lower visual field. If this is the case then this slowing should be more noticeable for binocular viewing conditions than monocular conditions. Honda and Findlay (1992) showed that monocular viewing reduced, but did not eliminate the difference between up and down saccade latencies, which does not provide direct support for the vergence hypothesis.

The present experiment aims to measure saccade latency using both monocular and binocular, viewing conditions, to examine the vergence movement hypothesis of the slowing of saccades to targets in the lower visual field. To examine the effect of directing attention along the vertical axis, saccade latency will be measured under three attentional conditions; attention up, attention down, and in a neutral (baseline) condition. The experiment is a modified version of Experiment 1 and uses a constantly displayed fixation point.

2.6.2 Method.

Subjects.

Two female and four male postgraduate students of ages 24-45 years from the psychology department were used as subjects. All had normal or corrected to normal vision. They were not informed of the nature of the experiment.

Apparatus

Stimuli were presented on a VDU monitor which was placed on its side, so that the timing sequence of targets presented in the upper and lower fields were not affected by the screen raster scan. Vertical eye movements were recorded using an infrared binocular eye tracker (Skalar IRIS), and the analogue signal was sampled and digitised every 5 ms by an apple Macintosh II microcomputer.

Stimuli and procedure.

The timing and presentation sequence of targets was as described in the overlap experiment (experiment 1). Each subject was tested under both monocular, and binocular viewing conditions in the three attentional blocks (attend up, attend down and neutral), with 84 trials in each block. The order of each attentional block was counterbalanced across subjects. Three subjects carried out the binocular viewing condition first and three carried out the monocular viewing first. The procedure and instructions were the same as used in the overlap experiment, with the exception that subjects were instructed to direct their attention covertly; above, or below, the central fixation point, on attentional blocks.

2.6.3 Results.

Saccades with latencies less than 100 ms were eliminated as anticipatory, and saccades with latencies over 300 ms were rejected as not being stimulus driven. The means obtained for targets in the upper and lower hemifield were collapsed and are displayed in terms of the attended and non-attended directions.

The mean saccade latency obtained in the binocular and monocular viewing conditions are displayed in Tables 5 and 6

Table 5. Mean saccade latency obtained under binocular viewing conditions.

	Attended				Neutral				Non-attended	
	Single		Bilateral		Single		Bilateral		Single	
Eccentricity	far	near	far	near	far	near	far	near	far	near
Mean Lat. (ms.)	152	150	178	169	164	160	203	186	208	182
S.Dev.	(29)	(24)	(41)	(35)	(31)	(23)	(44)	(35)	(46)	(51)

Table 6. Mean saccade latency obtained under monocular viewing conditions.

	Attended				Neutral				Non-attended	
	Single		Bilateral		Single		Bilateral		Single	
Eccentricity	far	near	far	near	far	near	far	near	far	near
Mean Lat. (ms.)	165	162	185	178	183	182	200	189	214	206
S.Dev.	(31)	(27)	(34)	(36)	(34)	(35)	(36)	(30)	(38)	(38)

The means were combined for the two target eccentricities and are shown for the binocular and monocular viewing conditions in Figures 6 and 7.

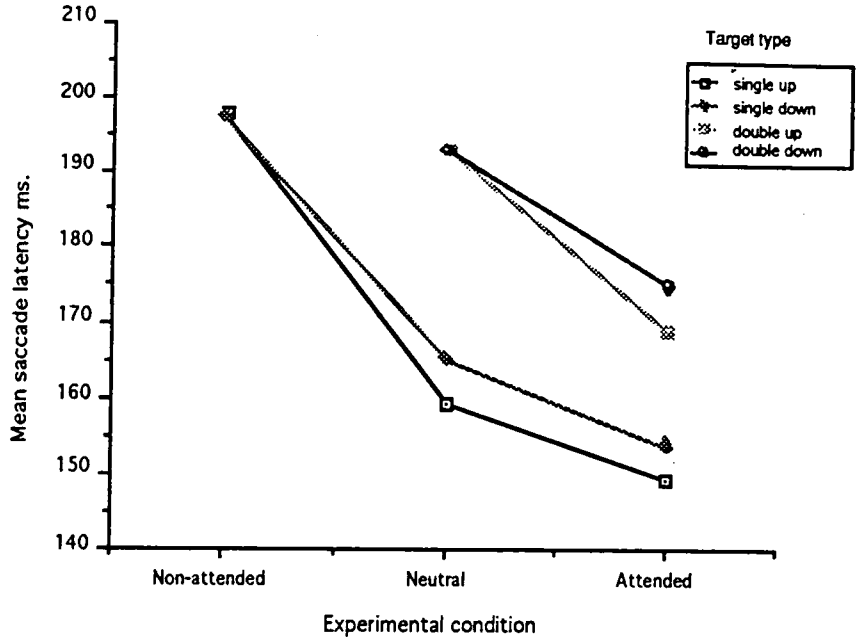


Figure 6.

Mean saccade latency obtained in Experiment 5.

Binocular viewing condition.

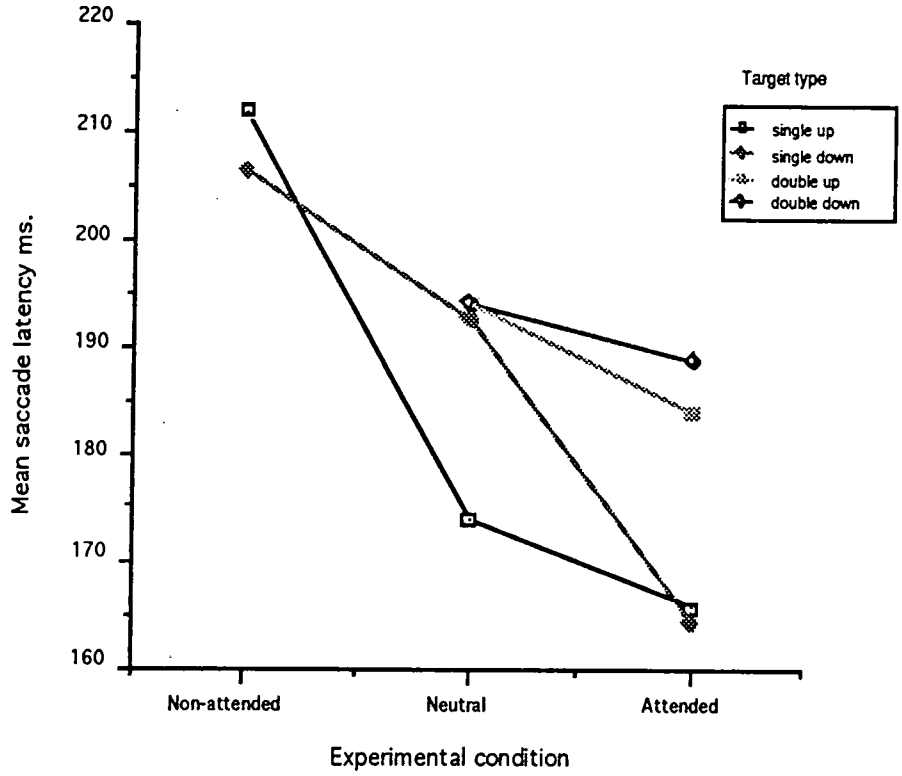


Figure 7.

Mean saccade latency obtained in Experiment 5.

Monocular viewing condition.

Binocular ANOVA.

The first (two factor) ANOVA was performed to examine the slowing of saccade latency to single targets in the lower visual field, using the means from the binocular viewing condition only. The mean latencies obtained to single targets were used, so as to perform a more sensitive test of the slowing of latency for targets in the lower hemifield. The ANOVA had three attentional factors (attended, neutral and non-attended) and two target factors (upper and lower) combined for both target eccentricities. The analysis showed a significant attentional effect ($F(1,5) = 78.4$ $p = 0.000$), but no significant target effect. The mean saccade latency obtained to targets in the lower field (174.4 ms) is slower than to targets presented in the upper hemifield (169.6 ms), but this difference does not reach significance in either the neutral or attentional conditions.

The second (two factor) ANOVA was performed on the Binocular condition results, to examine the effects of directing attention on saccade latency to single targets presented on the vertical axis. The ANOVA had three levels of attention (attentional, neutral and non-attentional) and two levels of target eccentricity (far, near). The main effect of attention was significant ($F(2,10) = 78.4$ $p < 0.000$). Post hoc analysis (Newman-Keuls), showed that the mean latency for targets in the attended direction (151 ms.), was significantly faster than the mean of neutral trials (162 ms $p < 0.05$.), and the mean latency for non-attended targets (195 ms.), was significantly slower than the neutral means ($p < 0.01$). There were no significant effects of target eccentricity on saccade latency ($F(1,5) = 2.4$ $p < 0.1$) and no significant interaction effect.

A third (three factor) ANOVA, compared the difference in mean saccade latency obtained to single and bilateral double targets. This analysis had two attentional factors (attended, neutral), two target factors (single, bilateral), with two levels of eccentricity (far, near). The main affect of attention was significant ($F(1,5) = 6.6$ $p = 0.0499$), showing that mean latency obtained for targets in the attended direction (162 ms) was significantly faster than when made on the neutral trials (178.2 ms). The factor of number of targets was highly significant ($F(1,5) = 76.5$ $p = 0.0003$), showing that bilateral target mean of 183.8 ms is significantly slower than the single target mean of 156.5 ms. There was also a significant two way interaction effect between number of targets and eccentricity ($F(1,5) = 23.1$ $p = 0.0048$). This is due to bilateral targets producing a greater slowing on saccade latency at the far than near eccentricities (means 202.8 and 185.9 ms respectively), while mean latencies to single targets were not affected by target eccentricity. There was no significant three way interaction effect as had been shown in the horizontal condition experiment (exp 1.).

Binocular viewing condition summary:-

1. There was no significant difference between latencies obtained to targets in the upper or lower hemifields.
2. A significant 11 ms benefit was obtained for saccades made to single targets in the attended direction ($p < 0.05$).
3. A large and significant cost (33 ms) was shown for saccades made to single targets in the non-attended direction ($p < 0.01$).
4. Mean saccade latency with single targets was unaffected by target eccentricity.
5. Mean saccade latency was slower by 26.5 ms (significant) on bilateral target trials than on single target trials ($p< 0.01$).
6. Mean saccade latency to bilateral targets at the far eccentricity were slower (not significant) than to bilateral targets at the near eccentricity.

Monocular ANOVA.

A (two factor) ANOVA was performed to show if there was any slowing of saccade latency for single targets in the lower field, under monocular viewing conditions. The ANOVA had three attentional factors (attended, neutral and non-attended) and two target factors (upper and lower) combined for both eccentricities. The analysis produced a significant attentional effect ($F(1,5) = 23.21$ $p = 0.000$), but no significant target effect. The mean saccade latency obtained to targets in the lower field (187.6 ms) can be seen to be slightly slower than to targets presented in the upper hemifield (182.7 ms), but this difference does not reach significance.

A second (two factor) ANOVA was performed on the mean latencies obtained to single targets, under monocular viewing conditions, with three levels of attention (attended, neutral and non-attended) and two levels of target eccentricity. This showed a significant attentional effect ($F(2,10) = 23.4$ $p = 0.0002$). Post-Hoc analysis (Newman-Keuls) showed that mean saccade latency to targets in the attended direction (163.8 ms) was significantly faster ($p < 0.05$) than in the neutral condition (182 ms) and the mean latency of saccades to non-attended targets (209.68 ms) was significantly slower than in the neutral condition ($p < 0.01$). The factor of eccentricity was also significant for single targets ($F(1,5) = 10.1$ $p = 0.025$), showing that the far eccentricity mean (187 ms) was significantly slower than the near eccentricity mean (183 ms).

A (three factor) analysis was performed to examine the slowing produced by bilateral target presentation under monocular viewing conditions. The ANOVA had two levels of attention (attended, neutral), two levels of target (single and bilateral) and two levels of eccentricity (far and near). The factor of attention was not significant ($F(1,5) = 4.1$ $p = 0.09$), showing that there was no benefit obtained when saccades were made to targets in the attended direction. The factor of target type was significant ($F(1,5) = 9.9$ $p = 0.02$) showing that the mean latency to bilateral targets (188 ms) was significantly slower than the mean latency to single targets (173 ms). The target by eccentricity interaction was not significant, although the bilateral target means again showed showed more slowing for far eccentricity (199 ms) than near eccentricity (191 ms) bilateral targets, while single target means were unaffected by target eccentricity (near = 173 : far = 172 ms).

Monocular viewing condition summary:-

1. There was no significant difference between latencies obtained to targets in the upper or lower hemifields.
2. A significant 18 ms benefit was obtained for saccades made to single targets in the attended direction ($p < 0.05$).
3. A large and significant cost (27.65 ms) was shown for saccades made to single targets in the non-attended direction ($p < 0.01$).
4. The mean saccade latency to single targets was affected by target eccentricity.
5. Mean saccade latency was 15 ms slower (significant) on bilateral target trials than on single target trials ($p< 0.05$).
6. Mean saccade latency with bilateral targets at the far eccentricity produced slightly more slowing than did bilateral targets at the near eccentricity (not significant).

2.6.4 Discussion.

The present experiment showed that voluntary directing visual attention along the vertical axis (up and down), had a similar effect on saccade latency as was shown when attention was along the horizontal axis. In this experiment the benefits obtained for saccades made to targets in the attended direction was greater (binocular = 11 ms : monocular = 18 ms) than was shown in the horizontal axis experiments (exp.'s 1-4) and reached significance compared to the mean latency in the neutral condition. Mean saccade latency to targets in the non-attended direction produced large cost compared to latency in the neutral condition (binocular= 33 ms : monocular = 27.6 ms) which is comparable to that shown in the horizontal axis experiments. This confirms that the consequence of directing visual attention is to produce a small benefit and large cost on saccadic reaction times. Presenting bilateral double targets produced an increase of some 26.5 ms on saccade latency under binocular viewing and a 15 ms increase under monocular viewing.

Directing attention along the vertical axis produced a similar pattern of costs and benefits on saccadic reaction time, as has been shown with manual reaction time experiments. Hughes and Zimba (1987) obtained small benefits for the attended hemifield and larger costs for targets in the non-attended hemifield. They concluded that directing visual attention produced a broad area of inhibition, the spatial boundaries of which are restricted in terms of the four visual quadrants. A movement of attention that requires a crossing of the horizontal or vertical meridians increased the costs obtained due to the presence of the inhibition in the opposite quadrant. Rizzolatti et al. (1987) showed a similar cost for an attentional crossing of the horizontal or vertical meridian. They explain this result in terms of their 'premotor' model of visual attention. The assumption is that a movement of attention from one visual halffield to another, requires a change in the direction component of that attentional movement. The same system is thought to be responsible for covertly orienting attention and producing a saccadic motor response. Changing the direction programme of a movement is thought to account for the increase in manual reaction times observed. Rizzolatti's premotor model is described in more detail and investigated further in Chapter four. The theories of Hughes and Zimba (1987) and Rizzolatti et al. (1987) can both provide a plausible account for the large increase in saccadic reaction times, observed for non-attended targets when presented along either the

horizontal or vertical axis.

Mean saccade latency was shown to be slower for targets in the lower visual field, but this slowing was not sufficiently large to be significant under binocular viewing conditions. An examination of the individual subjects results showed that three out of the six subjects produced slower saccades to lower field targets, two subjects showed slower saccade latency for upper field targets and one subjects latency was comparable in both cases. There is no obvious reason why some subjects should have slower latency saccades to lower field targets, while other subjects do not. It is possible that the slowing to targets in the lower field could occur due to some kind of practice affects with certain tasks, for example the three subjects that showed the slowing to down targets are all car drivers, while the three that showed the opposite trend are all non drivers.

2.7 General discussion.

The experiments described in this chapter showed that the primary effect of directing visual attention appears to be inhibitory. Directing attention produced a small (non significant) facilitation on mean latency for saccades made to targets in the attended direction. A greater and significant slowing on mean latency was shown for saccades made to targets in the non-attended direction. A comparable pattern of results was obtained when attention was directed to the left and right hemifields, with targets on the horizontal axis and when attention was directed to the upper and lower hemifields, with targets on the vertical axis. This finding is consistent with Hughes and Zimba's (1985, 1987) hemifield inhibition model of attention and with Rizzolatti et al.'s (1987) premotor model. The implications of these results are described in more detail in relation to a proposed model of visual attention in Chapter eight, which incorporates ideas from both these models.

When saccades were made to targets presented bilaterally and simultaneously the mean latency was significantly slower than when saccades were made to single targets. This slowing of latency was shown to occur when saccades were made to targets in the attended hemifield, when there is a reduction in the conflict of saccade direction. However, the conflict of saccade direction cannot be completely discounted and is examined further in Experiment 6 (Chapter three). The preferred hypothesis is that the slowing of saccade latency, results from an automatic activation of attentional inhibition. The onset of the stimulus is assumed to automatically produce inhibition for the contralateral hemifield which slows the latency of saccades made on bilateral trials. The time course of the slowing of saccade latency with bilateral targets is further investigated in Experiment 7 (Chapter three). The consequences of bilateral target presentation are also discussed in greater detail in relation to the proposed model of visual attention in Chapter eight.

The use of a gap condition produced a generalised speeding of latency for saccades made to single and bilateral targets, under attentional and neutral conditions. It appears that the

facilitation effect of prior fixation point offset must exert its influence on a separate component of the orienting system than does directed visual attention. The facilitation effect observed in the gap condition is consistent with the idea that it could occur due to a warning signal effect (Ross and Ross, 1980; 1981) and to an ocular and attentional disengagement (Tam and Stelmach, in press). The models of Becker and Jürgens (1979) and Findlay (1983) of saccade generation used separate 'when' and 'where' components to trigger a saccade and to compute the coordinates of that saccade. The warning signal effect of prior fixation point offset could enable preprogramming of the 'when' component which reduces saccade latency. The model described in Chapter eight, incorporates the ideas of separate when and where components. The gap effect is discussed further with relation to the proposed model and aims to describe the generalised facilitation effect and to explain why it does not interact with directed visual attention.

Chapter 3
An examination of the slowing of saccade latency produced by bilateral target presentation.

3.1 General Introduction.

The experiments described in Chapter two showed that presenting two targets bilaterally and simultaneously slowed mean saccade latency by some 20-30 ms. A comparable slowing was shown under both the gap, and overlap, fixation conditions; and also when the subjects directed attention to one hemifield following a verbal instruction. The slowing produced by presenting targets bilaterally and simultaneously has been observed by Lévy-Schoen (1969; 1974) and Findlay (1983). However, a different pattern of results is obtained when two targets are presented on the same side of fixation (Aslin and Shea, 1987; Becker and Jürgens, 1979; Findlay and Harris, 1984). Double targets have been presented on the same and opposite side of fixation, in a number of experiments in which saccade amplitude is measured, to gain a greater understanding of the system which generates saccades. Many of these experiments have involved presenting the two targets at varying temporal intervals in what are now termed 'double-step' experiments (Lévy-Schoen, 1969; Becker and Jürgens, 1979; Findlay and Harris, 1984). The main findings of these double step experiments have important implications for models of saccade generation and need to be considered in light of the consistent slowing observed in the previous experiments.

The saccade model of Becker and Jürgens (1979) involves a set of serial and parallel processing stages, which are involved in coding the retinal target location and initiating the final motor output of a saccadic eye movement. The computation involved separate channels to make a decision when to initiate a saccade ('when' system) and the calculation of the saccade amplitude ('where' system). The when system initiates the saccade and is thought to be responsible for the observable differences in mean saccade latency, on double step experiments. When restricted to saccades on the horizontal axis, the model consists of two channels in the decision mechanism, one for each direction. Saccades made to double step targets, in which the second target is located to the opposite side of initial fixation to the first (*pulse overshoot*), show two patterns of saccade responses. At long time intervals between the presentation of the first and second target (Interstimulus intervals =ISI) subjects typically make a 'step by step' response of two saccades, one to each of the target locations. At short interstimulus intervals a 'skip over' response is shown in which the first position is ignored and the saccade is made to the second target location. The implication is that a second target appearing soon after the first, results in the cancellation of the saccade being prepared and the subsequent reprogramming of a new saccade to the second target location, producing the skip over response. At longer ISI's two saccades are programmed in sequential order requiring two separate computations and therefore producing an increase in saccade latency. This idea is supported as saccades made directly to the second target, are executed at a latency of over 260 ms, which is about twice the latency expected for a saccade to a single target. The

amplitudes obtained with double step targets in opposite fields, are shown to be accurately located to either the first or second target locations. This suggests that the saccade programme is prepared and executed to the first location, or the second step causes a completely new programme to be prepared and executed to the second step location, with the resulting increase in saccade latency. Recent models of saccade generation have not all supported the idea that the amplitude and direction of a saccade are computed by a fixed hierarchical programme. It has been suggested that saccade amplitude and direction may be computed in any order (Abrams and Jonides, 1988) and alternatively that amplitude and direction could be computed simultaneously in a holistic way (Abrams and Jonides, 1988; Findlay, 1987).

Aslin and Shea (1987) provided an updated version of Becker and Jürgens (1979) saccadic programming model. The revised model used a global decision making mechanism, not organised into separate directional channels. The decision mechanism is a delay associated with the deployment of attention to an extrafoveal target location and the activation of the subsequent saccadic programme. A comparator is used to constantly monitor and mark the current target location. If a new target appears in the opposite hemifield (as occurs with pulse overshoot target steps) then the comparator aborts the old decision process, and a new one is started. A restarted decision process is assumed to add 50-70 ms on the overall decision time. It is difficult to see how this model could account for the increase of saccade latency, when two targets are presented bilaterally and simultaneously in opposite hemifields, as was shown in Chapter two. The activation of the decision process in this model is stimulus driven and starts when a target appears. In this case the decision mechanism would not be activated, before simultaneous target onset. Given that both targets appeared at equal and opposite eccentricities in each hemifield, it is difficult to decide what triggers the decision mechanism in this model to make a saccade to one of these targets. This model does not have connections with top-down components which would account for a selection of one of the targets in neutral trials and would also be required to explain how the attentional instruction enables the subject to always make a saccade in the attended direction.

Double target steps presented on the same side of fixation show a different pattern of saccadic responses, to those presented in opposite hemifields. In this instance the latency of the saccades is not affected by presenting two targets, but saccade amplitude is affected. The amplitude of the saccade is linked to the time from the second target step and the initiation of the saccade. When the saccade is initiated a short time after the second step, the saccade is made to the first target location. With a long delay between the second step and the initiation of the saccade, the saccade is made to the second target location. However, saccades initiated at intermediate times show a modification of the amplitude of the resulting saccade. Saccades made do not show the clear step by step and skip over responses, but show what has been termed an 'amplitude transition function'. With increasing intervals, the saccade amplitude increases towards the second target step (Becker and Jürgens, 1979; Findlay and Harris, 1984). Becker and Jürgens suggested that the decision process (when) that triggers the saccade is not affected by the second target onset and that the amplitude computation is a separate parallel process. When the decision process is completed the amplitude computation

is sampled and a saccade is executed. Sampling the amplitude computation before the second step will result in a saccade to the first step location, sampling after the second step will result in a saccade to the second step, but sampling during second step presentation results in saccades of intermediate amplitude. Deubel et al., (1984) proposed a saccade model based on a visuomotor map, which can also explain the amplitude transition function obtained by Becker and Jürgens. In this mode the amplitude of the saccade is read out as the centre of activity of the visuomotor map. The activity of the map slowly builds up when a stimulus appears on the corresponding retinal location. When the second target appears the centre of activity begins to move towards the second stimulus location. The amplitude of the saccade depends on the point at which the decision to make a saccade is made, decisions made at increasing intervals after the second step will show amplitudes increasing towards the second target location as a smooth function.

The models outlined above use the notion of a decision mechanism which involves two components for saccades made to the left and right side of fixation. These models are largely based on the findings that double step targets within a hemifield alter the saccade amplitude, while steps over the vertical meridian slow saccade latency. This finding is consistent with modifications to either the amplitude or decision mechanisms of the saccadic system. Not all double step experiments have supported this simple division. Findlay and Harris (1984) used double step targets located off the horizontal axis, to see if the increase in saccade latency observed with double step target is restricted to crossings of the vertical meridian at the fovea. Under one configuration this resulted in steps which crossed over the vertical meridian (into the other hemifield), at a location above the central fixation point. In this instance the amplitudes of saccades made to targets in the opposite direction still showed a smooth amplitude transition function, rather than the expected pattern of a saccade to one location or another, as was shown for crossed double targets located along the horizontal axis. Findlay and Harris (1984) interpreted these results as implying that saccade amplitude and direction are not computed separately, but could be calculated by a 'visuomotor map' of retinal information similar to that suggested by McIlwain (1976) and Deubel et al. (1984). This map could represent two-dimensional decision channels for each retinal location, which would be excited by a stimulus onset corresponding to that retinal location and would trigger a saccade when activity has peaked in this channel. These channels are thought of as having an excitatory centre and an inhibitory surround, so that a second step target falling in an inhibitory region will suppress the on going saccade decision process. Step targets in opposite hemifields along the horizontal meridian are more likely to fall in an inhibitory region, which will result in the bimodal pattern of responses shown to crossed steps. Steps occurring in opposite sides which are located away from the horizontal axis, could both fall within an excitatory area of a single channel, which would result in the unimodal pattern of responses obtained.

Mean saccade latency was increased by some 20-30 ms for bilateral targets, in both the attentional and neutral trials in the experiments described in Chapter two. The slowing of saccade latency on bilateral presentation may be explained in terms of the above framework of a visuomotor map. Two targets located in opposite hemifields along the horizontal axis could fall

in opposite channels of the map. This would result in the production of inhibition within two channels of the visuomotor map, which encode both target locations. This increase of inhibition will require extra time to enable the decision channel to initiate a saccade. So it is possible that the presence of the non-attended bilateral target produces an automatic increase of inhibition within the visuomotor map, that produces the increase in saccade latency.

The first experiment in this chapter examined the slowing observed on bilateral double target trials, under conditions of directed visual attention. A slowing of saccade latency was observed in the experiments of Chapter two under attentional conditions. However, a possible explanation for the slowing which needs to be discounted, is that subjects may not have prepared to make a saccade in the attended direction. This could have been the case due to the presence of a non-attended single target, on a third of all trials. The increase in latency with bilateral double targets in attentional trials, could reflect the conflict on the 'where' system, resulting in extra time to select a direction in which to make a saccade with bilateral targets. This where decision will be required on both the neutral, and attentional blocks, if the subjects did not prepare to move in the attended direction. The next experiment examined this possibility by presenting single and bilateral simultaneous targets, without using any non-attended single targets. In this instance subjects will always saccade in the attended direction, on every trial, so there is no conflict of saccade direction with bilateral targets. If an increase of latency is again observed with bilateral targets, then the idea of inhibition produced automatically by the non-attended bilateral target is further supported.

The second experiment examined the time course of the inhibitory effect of bilateral double target presentation. The non-attended double target was presented at intervals before, after and simultaneously, with the onset of the saccade target. The saccade targets were always presented in the attended hemifield. Equal numbers of single targets were presented to examine the inhibitory effects of the non-attended bilateral target, in relation to the baseline mean latency obtained with single targets. There were no non-attended single targets, so that subjects always saccaded in the attended direction. This enabled the time course of the inhibitory effect that a non-attended bilateral target has on saccade latency, to be plotted.

3.2 Experiment 6: The effect of orienting attention to single and bilateral targets, on saccade latency.

3.2.1 Introduction.

The slowing observed on mean saccade latency with bilateral target presentation could be due to a conflict of saccade direction in the 'where' component of the saccadic system. Selecting a direction on bilateral trials would require an extra direction selection process, which is not required with single targets. This explanation was thought to be unlikely as a comparable slowing was observed, in the attentional blocks of trials in which the subject would always saccade in the attentional direction thus removing the directional conflict on bilateral trials.

However, the previous experiments used single targets presented in the non-attended direction, on one third of all trials. So, it is possible that subjects do not preprogramme the saccade direction on attentional trials and as a result show a slowing of saccade latency, due to the directional decision process, as could be the case on neutral trials. This experiment examines this possibility by presenting targets bilaterally on half of the trials and a single target which is always in the attentional direction on the other trials.

If a slowing of saccade latency is observed in this experiment on bilateral double target trials, then the conflict of saccade direction, explanation is weakened. A different explanation of the slowing on bilateral double target trials incorporates the idea of an automatic increase of attentional inhibition within the attentional orienting system. The appearance of a target in the non-attended hemifield could produce a transient and short lasting inhibition for the opposite hemifield, which would result in an increase of saccade latency. If a target appearing in the non-attended visual field increases inhibition within the contralateral attentional orienting system, then it may increase saccade latency given the suggestion that these systems are closely linked (Shepherd, Findlay and Hockey, 1986; Rizzolatti et al., 1987; Umiltá et al. 1991). If the onset of a non-attended bilateral target increases inhibition within the attentional orienting system, then the increase in mean saccade latency on bilateral target presentation will not be reduced by using a design that does not require a saccadic eye movement in the non-attended direction. If the increase in saccade latency has an oculomotor cause resulting from a conflict of saccade direction (eg. 'where' component of Becker and Jürgens 1979, model), then it should be reduced in this experiment.

The present experiment examined these two plausible explanations by measuring saccade latency to single targets which were always presented in the attended direction, and to bilateral simultaneous targets, under conditions of directed attention. There were equal numbers of single and bilateral double target trials, within each block and no contra-attentional single targets. In this experiment the saccade direction remained constant on every trial. The design, timing and procedure were otherwise identical to that described for Experiment 2 (gap).

3.2.2 Method.

Subjects.

Nine subjects were used in total, seven of the subjects had been used in previous experiments and two naive subjects (VP, KG), were also used to compare individuals performance with those of practised subjects.

Stimulus and procedure.

The stimuli and apparatus used was identical to that used in experiments 4 and 5. Each

block consisted of 96 trials. On half of the trials a single target was presented and on half of the trials bilateral double targets were presented simultaneously at equal eccentricities. Each subject was tested in an attend left and an attend right block (the order being randomised across subjects). Twenty practice trials were used before the main experimental blocks.

The timing of each trial sequence was identical to that used in the gap experiment (Figure 1). It was expected that the use of well practised subjects, with a single saccade direction under gap conditions, should produce fast mean saccade latencies.

3.2.3 Results.

Mean saccade latency was similar in the attend left, attend right conditions and for the two target eccentricities. The means for the two target eccentricities, for the attend left, attend right, conditions were combined for the analysis and are shown for each subject, in Table 7. There appears to be no difference between the results of the practised and unpracticed subjects (shown in bold). There is some variability across subjects, in the amount of slowing produced by bilateral target presentation.

Table 7. Mean saccade latency obtained for each subject.

Subject	Single target mean (ms)	Bilateral target mean (ms).	Increase (ms)
AC	158.4	178.1	+19.7
GD	124.2	133.2	+9.0
KG	**110.4**	**131.1**	**+20.2**
DH	120.9	131.9	+21.5
FN	131.9	148.7	+16.8
FP	151.9	163.8	+11.9
VP	**123.4**	**128.2**	**+4.8**
GW	117.8	144.9	+27.1
RW	107.4	120.3	+12.9
TotalΣ	127.3	142.2	+14.9

The mean latency obtained to single targets is shown to be a very fast 127 ms. This can be attributed to the use of well practised subjects, under attentional conditions (in which saccades were always made in one direction) and the 100 ms gap condition. A fixed time interval (of 1000 ms) was also used between initial fixation onset and target onset, which will have enabled a 'pacing' of responses. This experiment will have enabled all saccade parameters except for the target amplitude, to be preprogrammed before target onset, producing fast mean latency with single targets.

The results of this experiment show that bilateral double simultaneous target presentation has again produced a slowing on mean saccade latency, compared to the mean obtained to single targets. The bilateral double target mean (142 ms) is some 15 ms slower than the mean obtained to single targets (paired t-test 6 df = 6.5 $p<0.01$). This is less than the 25 ms slowing shown for bilateral targets in the gap experiment, but similar to the 19 ms slowing

observed in the overlap experiment.

3.2.4 Discussion.

The present experiment directed attention and measured the saccade latency to single and bilateral double targets with a constant saccade direction. The results showed that the mean latency of saccades made to bilateral targets was significantly slower than the mean latency obtained to single targets. The aim of this experiment was to show that the slowing observed on bilateral trials was not due to the conflict of saccade direction which would occur on bilateral trials. The mean latency obtained to single targets was a very fast 128 ms, which presumably reflected the preprogramming of saccade parameters that can occur covertly, before target onset. Given that bilateral double targets produce a slowing on mean saccade latency (15 ms) under these conditions, it is unlikely that the slowing has a simple oculomotor explanation. In this experiment there is no conflict of saccade direction with bilateral targets, so there is no extra decision for the where component of the saccadic system. It is more likely that the slowing reflects an automatic inhibitory effect, produced by the onset of a stimulus in the non-attended field.

The automatic inhibitory effect of a stimulus onset could be incorporated into a similar model to that proposed by Hughes and Zimba (1987). According to their model covertly orienting attention produces inhibition for the non-attended visual half field. The extra assumption which is required to explain the slowing on bilateral target trials, is that a peripheral transient change, such as the onset of a briefly presented target (or peripheral cue), could activate a transient attentional component which produces a short lasting inhibition for the contralateral field. The presence of this extra inhibition results in a slowing of saccade latency for a saccade made to a target in the contralateral field. Bilateral targets could produce an increase in this inhibition in both hemifields, with the result of slowing saccade latency. The spatial extent of the distributed area is debatable, it could be for the mirror image location (Singer, Zihl and Pöppel, 1977), or for the whole of the contralateral hemifield. These ideas are further developed with reference to the proposed attentional model in Chapter eight.

The next experiment examined the time course of the inhibitory effect that bilateral double targets have on saccade latency. This was performed by varying the temporal interval, between the appearance of the non-attended bilateral target and the onset of the attended bilateral target. The subjects always made a saccade in the attended direction.

3.3 Experiment 7: An examination of the time course of the inhibitory effect of the non-attended bilateral target.

3.3.1 Introduction.

This experiment was performed to investigate the time course of the inhibitory effect of presenting bilateral double targets. The non-attended bilateral targets, were presented simultaneously with the saccade target and also at various temporal intervals before, and after, the appearance of the saccade target. The saccade target always appeared 1000 ms, after initial fixation onset, so that subjects would expect to initiate a saccade at this time enabling a 'self pacing' on repeated trials. The non-attended bilateral target appeared at intervals (of: 0, 20, 40, 80, 160 and 240 ms) *before, simultaneously* with, or *after*, this fixed saccade onset time. This experiment presented single targets in the attended direction to obtain a baseline measure of saccade latency and there were no contra-attentional single targets (as in Experiment 6). Any slowing of saccade latency with bilateral target presentation will be unlikely to reflect oculomotor factors, as subjects will always make a saccade in the attended direction and will be 'cued' by the repetitive timing sequence of the experiment. The fixation point remained on throughout each trial so as to limit the amount of warning signal information available prior to target onset.

It was thought that the appearance of a non-attended target at short time intervals (eg. 20, 40 and 80 ms), before the onset of the saccade target, could produce slower latencies than when both targets appear simultaneously. This could be expected as the inhibition produced automatically by the onset of the non-attended bilateral target, would have time to reach its maximum level, before the onset of the saccade target. However, the appearance of the non-attended target at longer intervals (eg. 160, 240 ms), before the saccade target, may not affect saccade latency, if the level of the inhibition is relatively short lasting and has already decreased in intensity. A second possibility is that a non-attended target appearing at long intervals before the onset of the saccade target, could produce a facilitation effect on saccade latency, due to a warning signal effect (Ross and Ross, 1980, 1981). The early onset may trigger the decision mechanism of the saccade system so that it is ready to initiate a saccade once the saccade target appears. Ross and Ross (1980, 1981) observed a facilitation effect with the prior onset of a fixation point, before the presentation of the saccade target. The prior onset had its maximum effect with an onset 100-300 ms before target onset. This suggests that both a prior onset and offset can facilitate saccade latency. The results of Ross and Ross (1980, 1981) lead to the suggestion that the onset of a non-attended target at short intervals (20-80 ms) after the saccade target onset might also produce a slowing effect on saccade latency. Their results showed that fixation onset *after* target onset slowed saccade latency, with its maximum effect obtained for onsets up to 100 ms after target presentation. This is in contrast to the facilitation effect reported with fixation offsets *after* target onset (Braun and Breitmeyer 1988; Ross and Ross, 1980; 1981; Reulen, 1984; Saslow 1967).

Braun and Breitmeyer (1990) examined the effects of the reappearance of a previously

attended stimulus on saccade latency. In their experiment the initially displayed fixation point went off prior to saccade target onset, but reappeared after an 'off-interval'. The off-interval varied from 0 ms in which the stimulus remained on and overlapped with the target onset, to intervals of 800 ms where it went off before target onset. The target always appeared at a fixed interval of 200 ms, after fixation offset. When the fixation point overlapped continuously with target onset, mean latency was shown to be some 220 ms. Saccade latency decreased sharply when the fixation onset occurred before target onset, reaching a minimum mean latency of 140 ms with onsets of fixation 200 ms, before target onset. When the fixation point onset fell within intervals of 100 ms before, or 150 ms, after the onset of the saccade target, mean saccade latency was shown to be increased. Mean latency again decreases with off-intervals over 150 ms after saccade target onset. Braun and Breitmeyer (1990) account for the initial speeding effect obtained with the offset of the fixation point as being due to attentional disengagement. As saccade latency was slowed if the fixation point re-appeared at intervals 100 ms before to 150 ms after target onset, its re-appearance could 'engage' attention and slow saccade latency. These results are consistent with those of Ross and Ross (1980, 1981) who showed that onset or offset of a visual or auditory warning signal reduces the saccade latency to a target, but only a visual stimulus onset, increased saccade latency. Braun and Breitmeyer (1990) view both the warning signal effect, and disengagement of attention explanations, as providing plausible accounts of the speeding observed with prior fixation offset. The influence of attention in producing the slowing on saccade latency was shown in their control experiment, where only the onset of a previously attended stimulus slowed saccade latency, while the onset of a general distraction did not have this effect.

In light of the findings of Braun and Breitmeyer (1990) and Ross and Ross (1980, 1981), the appearance of the non-attended bilateral target at short intervals (20, 40 and 80 ms) after the onset of the saccade target could be expected to increase saccade latency, compared to when a single target is presented. The first possibility is that the onset of the non-attended bilateral target will serve to cancel the ongoing saccade programme. This will result in an extra time being taken to initiate a saccade as the programme will have to be started from the time of the non-attended target onset. This will result in saccade latency being greater than is observed with simultaneous target onsets. An alternative possibility is that saccade latency will be slowed due to an automatic increase of inhibition in the ipsilateral hemifield, produced by the non-attended target onset. In this instance the slowing may be less than is shown with simultaneous target onset, as the ipsilesional field may not be subject to the maximum level of inhibition, when the target is presented.

A non-attended bilateral target appearing at long time intervals (160, 240 ms) after the saccade target should not affect saccade latency as the saccade should have been executed, or almost fully programmed, by this time. It would therefore be expected that a non-attended target presented 160-240 ms after saccade target onset will not effect saccade latency.

3.3.2 Method.

Subjects.

Five postgraduate students, all of whom had taken part in at least one of the previous experiment and one undergraduate (ZAC) who had not taken part in any similar experiments, were used as subjects. Their ages ranged from 22 to 30 years.

Stimulus and procedure.

The stimuli and apparatus was identical to that used in experiment 6. The saccade target appeared in the right hemifield on every trial and subjects were instructed to: "*attend to the right of fixation on every trial*". The fixation point remained on (overlap) throughout each trial. Each block contained single targets and bilateral double targets in which the non-attended target appeared in the left hemifield, at various time intervals (of: 20, 40, 80, 160 and 240 ms): before, simultaneously, or after, the saccade target. The non-attended targets also appeared simultaneously with the onset of the saccade target (0 ms). The timing sequence of target presentation is shown in Figure 8.

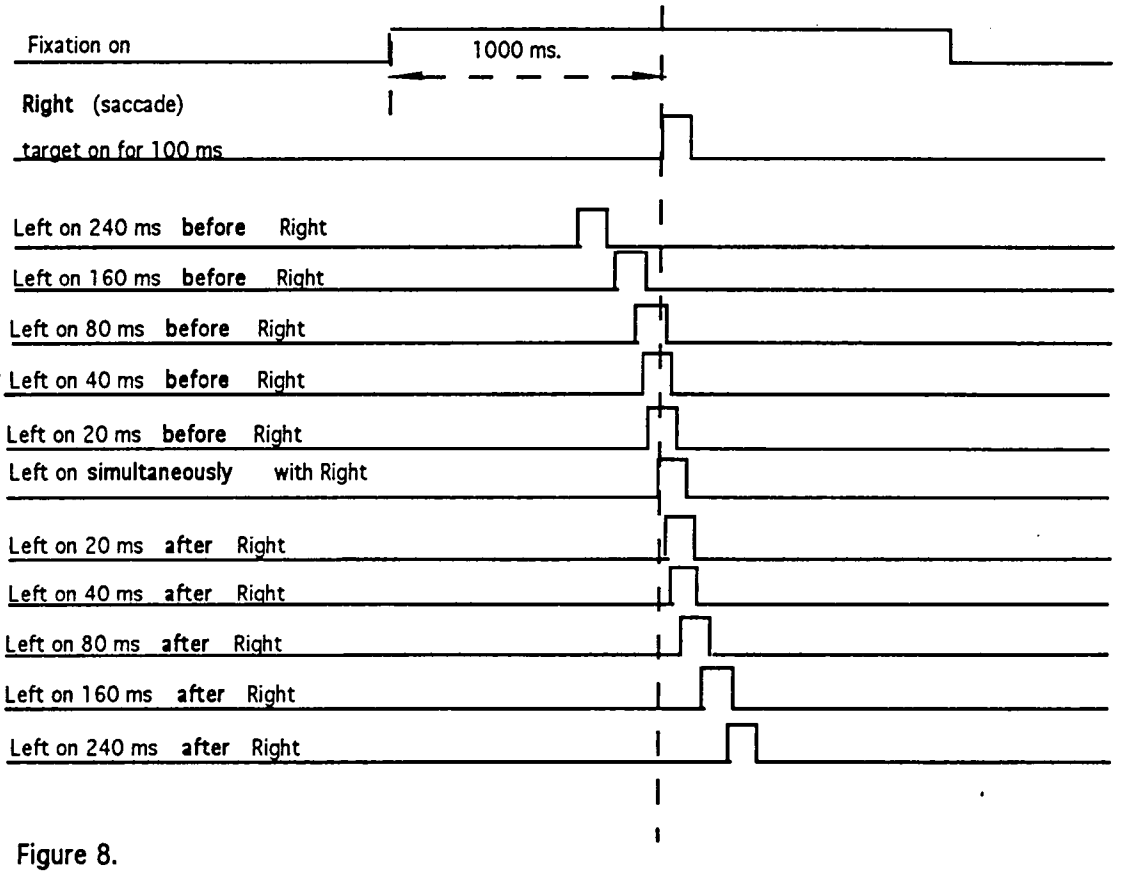


Figure 8.
The timing sequence of stimulus presentation in Experiment 7.

There were 272 trials in each block and subjects were tested on two blocks, to increase the amount of data obtained for each gap interval. Each block consisted of: 80 bilateral trials in which the non-attended target appeared before the saccade target (left before right: **LBR**); 80

trials in which the non-attended target appeared after the saccade target (right before left: **RBL**). Five gap intervals were used in each condition (LBR and RBL) between the onset of the non-attended target and the onset of the saccade target. This produced a total of sixteen trials for each of the bilateral targets with each of the gap intervals, when the latencies from the two blocks were combined. On 16 trials a bilateral targets appeared simultaneously with the saccade target and on 96 trials a single target was presented in the attended hemifield. Single targets were always presented in the right (attended) hemifield and were used to obtain a baseline measure of mean saccade latency. Each block took approximately 45 minutes to complete and were carried out on consecutive days of testing.

Targets appeared at either: 5.0°, or 9.5°, eccentricities, bilateral targets always appeared at equal and opposite eccentricity locations. The saccade target appeared 1000 ms after initial fixation onset, in the right hemifield. An indicator and mask appeared after the saccade target using the same timing sequence as in all the previous experiments (see: Figure 1). Subjects were instructed to, always saccade to the target which appeared in the attended (right) hemifield, and were informed that on some trials a target would appear in the non-attended direction, which should not be saccaded towards. Subjects responded to the number of indicator dots, by using a hand held button box. No further information was given, so the subject was not informed that the non-attended target could appear at various time intervals before, or after, the saccade target.

3.3.3 Results.

A total of 7.7% of records were discarded on the initial analysis as being 'bad data' (ie. record unanalysable due to blinks, saccade initiated before target onset, or two saccades were made to reach final target position). Saccades with latency of less than 80 ms and over 300 ms, were not included in the final analysis. This resulted in a further 1.7 and 7.4% of saccade records being excluded respectively.

The Initial analysis showed that saccade latency was not affected by target eccentricity. The means for the two target eccentricities were combined, to increase the amount of data. Figure 9. shows the individual subjects mean saccade latencies. A similar pattern of results was shown for five of the subjects, but the results from the naive subject (ZAC) show a somewhat different trend. Five subjects produced slower mean saccade latencies, when a non-attended target appeared 20, 40 and 80 ms, *before* (LBR) the saccade target, compared to their 'baseline' mean latency obtained to single targets. The bilateral simultaneous targets produced the slowest mean saccade latency, which was some 15-25 ms slower, than the baseline mean obtained to single targets. When the non-attended target appeared at longer time intervals (160 and 240 ms) *before* the saccade target, mean saccade latency can be seen to have been facilitated compared to that obtained with single targets. When the non-attended double target appeared shortly (20, 40 ms) *after* the saccade target (RBL), mean saccade latency was slower than that obtained to single targets. When the non-attended double appeared some 160/240

ms after the saccade target mean saccade latency is similar to that obtained to single targets.

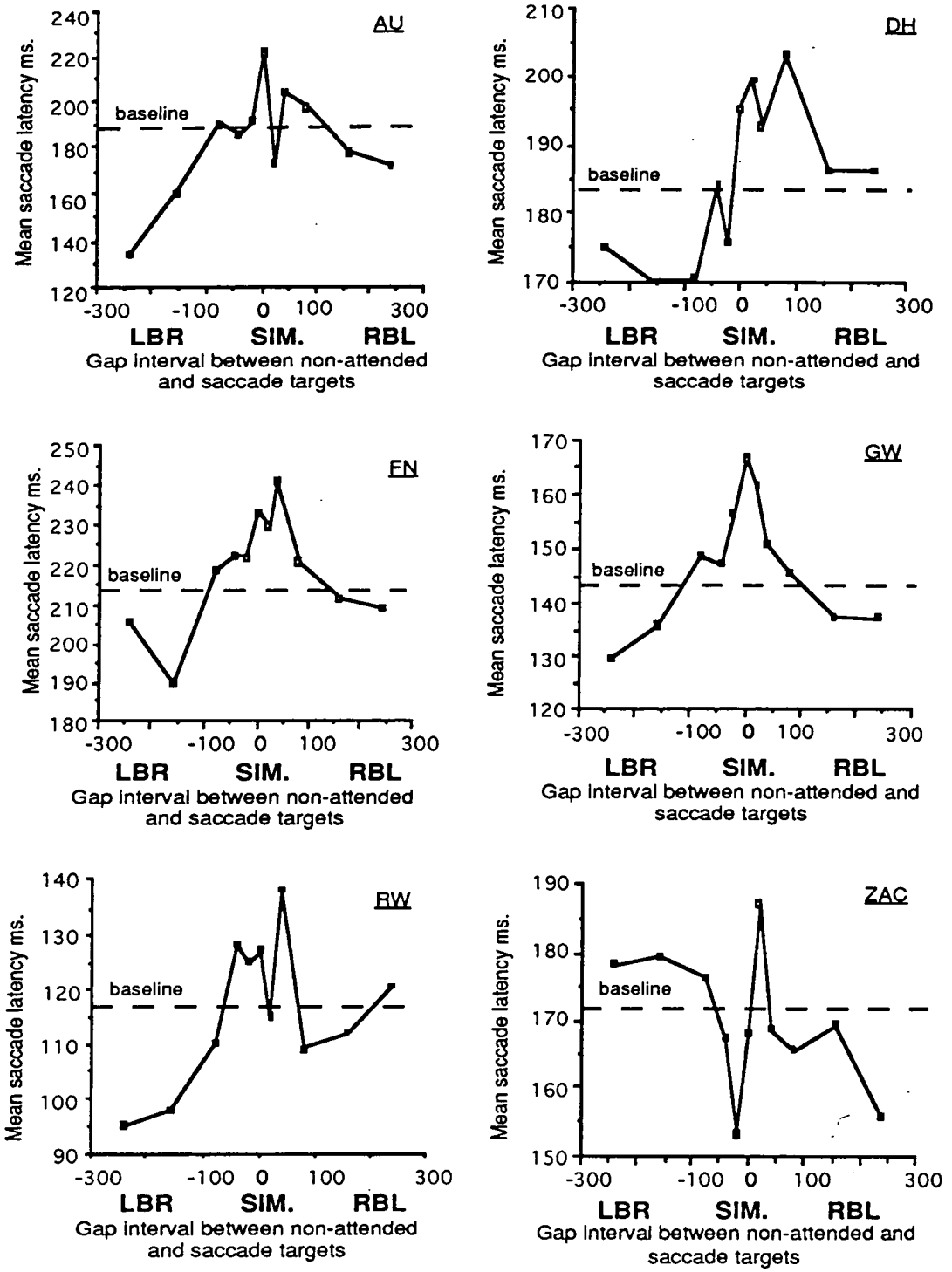


Figure 9.
Mean saccade latency obtained from each individual subject in Experiment 7.

Solid line = latency with double targets for each gap interval.
Dashed line = baseline latency obtained with single targets.

The results from one subject (ZAC) show a different pattern to those obtained from the other five 'experienced' subjects. The onset of a non-attended target at short intervals (20, 40 ms) *before* the saccade target produced a facilitation effect on mean latency. This subject has

not shown the facilitation effect when the non-attended target appeared at longer intervals (80, 160 240 ms) *before* the saccade target as mean saccade latency was slower than obtained to single targets. The greatest slowing of saccade latency was observed when the non-attended target appears 20 ms after the saccade target onset. The simultaneous onset of bilateral targets has not produced the slowest mean saccade latency, as trend which is shown in all previous subjects results in all experiments. Examinations of the results obtained from this subject failed to show any obvious reason for this discrepancy. As the results from this subject are clearly different to those obtained from the other five subjects it is planned to perform two analysis; one on the mean latencies from all six subjects and a second analysis on the mean latencies excluding the results from this subject.

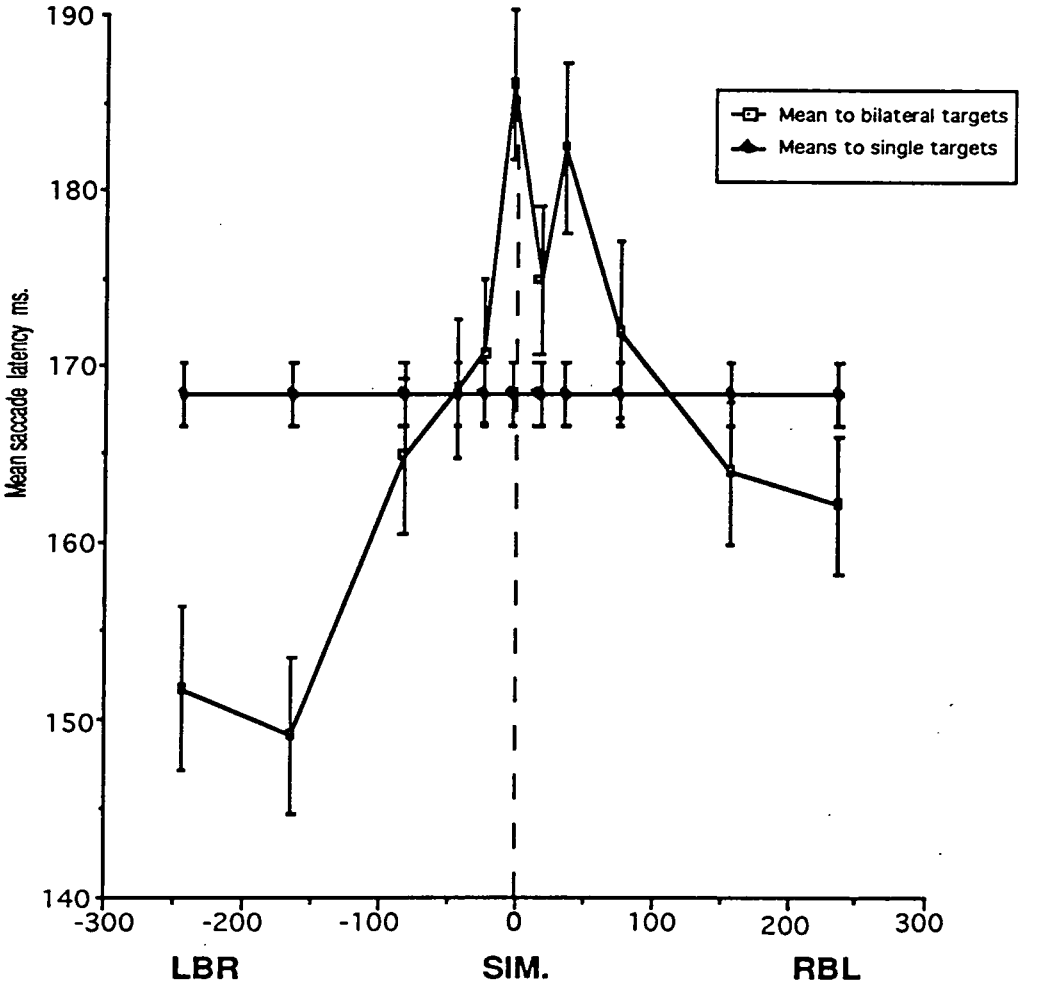


Figure 10.
Mean saccade latency obtained from six subjects in Experiment 7.

The data points show the mean latency obtained to a target in the attended hemifield following an onset in the non-attended hemifield at the gap interval shown.

The combined mean saccade latencies from the six subjects are shown in Figure 10. Mean saccade latency to single targets is shown as a baseline measure. Bilateral and

simultaneous presentation of targets, has produced the greatest slowing of saccade latency by 18 ms, compared to the baseline mean obtained to single targets. A non-attended target appearing a long interval before, the saccade target (LBR 240, 160) produced a large facilitation effect of some 30 ms, compared to bilateral simultaneous target presentation, and of 15 ms, compared to the baseline mean obtained to single targets. The onset of a non-attended target at short intervals (20, 40 ms) before the right target produced mean saccade latencies slower to those obtained to single targets. The onset of a non-attended target at short intervals (20 and 40 ms) after the saccade target, produced a slowing of saccade latency compared to the baseline mean obtained to single targets. Mean latency is slightly faster in trials when a non-attended target appeared 160 and 240 ms after the saccade target. This is an unexpected finding as a saccade should have been initiated by 160/240 ms after the onset of the saccade target and should not be affected by the late onset of the non-attended target. An examination of the individual subjects data shows that this trend is produced by the results from only one subject (ZAC), this is confirmed by an examination of Figure 12 which shows the mean latency obtained from five subjects excluding those from ZAC. In Figure 12 mean latency obtained when the non-attended target appears 160/240 ms after the onset of the saccade target is comparable to the baseline mean.

The latency distributions obtained from the data of all six subjects combined are shown in Figure 11. The latency distributions are plotted separately for single targets and for each of the eleven gap intervals. The peak of the distribution for single targets is shown around 140 ms, while for simultaneous bilateral targets it occurs at 185 ms. The trend shown for the early onset of a non-attended target (LBR 160/240) is to produce many more saccades of short latency. As the time interval between the onset of the non-attended target and saccade target decreases (LBR and RBL 20/40/80) the effect is to produce more saccades of slower latency. When the non-attended target appears at long intervals after the onset of the saccade target (RBL 160/240) the distribution can be seen to be similar to that obtained for single targets. These distributions suggest that the early onset of a non-attended target produces saccades of short latency. Non-attended target onset occurring around them time of the onset of the saccade target produces more saccades of a slower latency. The simultaneous onset of targets producing a definite peak of slow latency saccades.

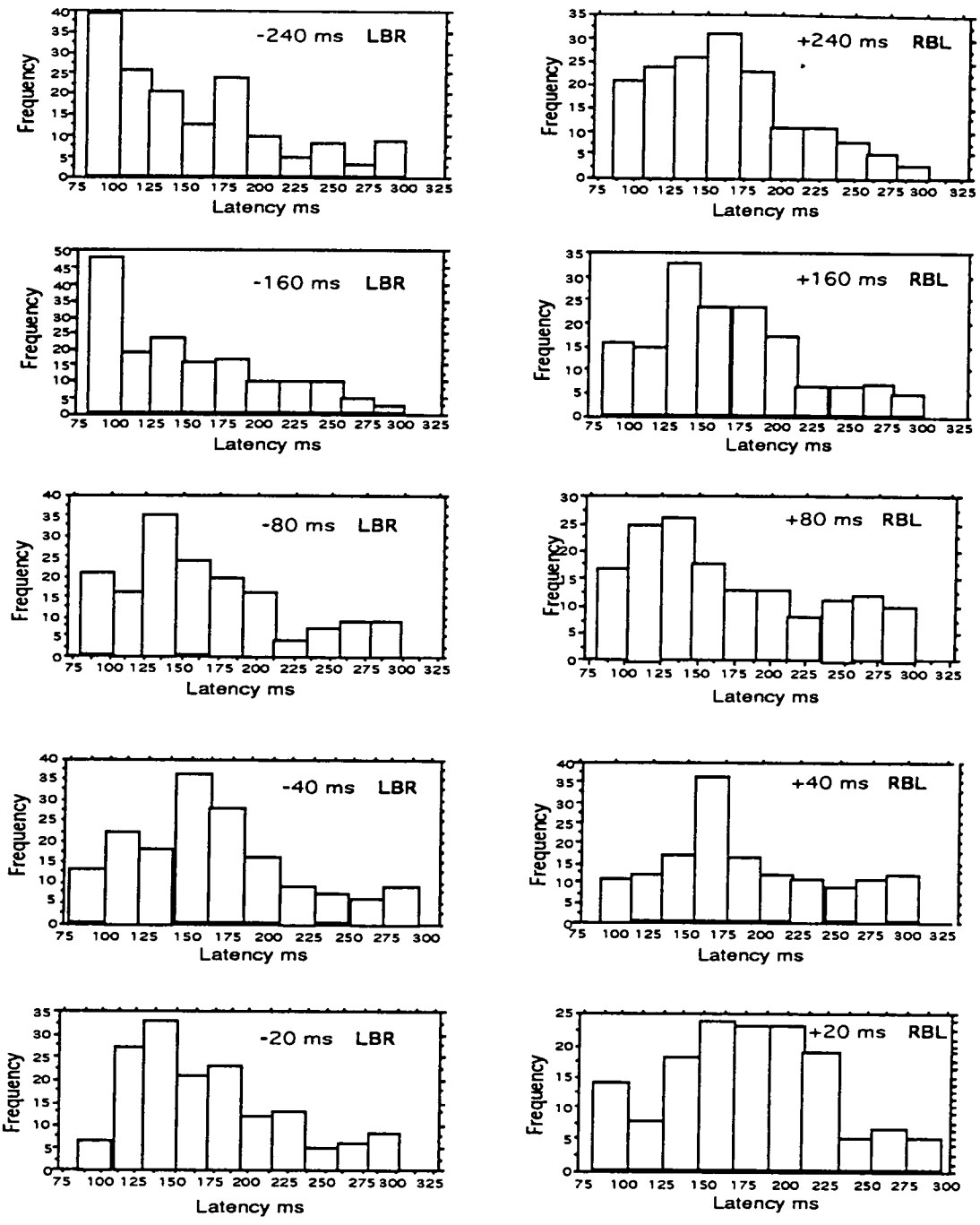


Figure 11.
Latency distribution of saccades made with bilateral targets appearing at gap intervals before and after the saccade target onset.

A (two factor) ANOVA was performed on mean latency for bilateral targets with a factor of gap interval (11 gaps) and target eccentricity (two eccentricities). The factor of eccentricity was not significant ($F(1,5)=4.39$ $p=0.09$) showing no effect of eccentricity of bilateral presentation on saccade latency and there was no interaction effect ($F(10,50)=1.06$ $p>0.4$). The factor of gap was significant ($F(10,50)=4.65$ $p=0.0001$). A Newman-Keuls post hoc analysis was performed and confirmed that the mean latency obtained when a non-attended target appeared at long intervals of 160 and 240 ms (LBR -160/240), before the saccade target are significantly faster than when the bilateral targets appear simultaneously ($p< 0.01$). The mean latency

obtained in the LBR -160/240 condition is also significantly faster ($p<0.05$) than the mean obtained when a non-attended target appeared 20 and 40 ms after the saccade target (RBL +20/40).

A paired t-test was performed to compare the mean latencies obtained to single targets and bilateral targets, to examine the facilitation and inhibitory effects of presenting a non-attended target. The analysis confirmed that the means obtained with bilateral simultaneous presentation and when the non-attended target appeared 40 ms before the saccade target (RBL +40 ms) were significantly slower than to single targets (5 df $p=0.018$ and $p=0.010$ respectively). Mean saccade latency observed when the non-attended target appeared 160 ms before the saccade target (LBR -160 ms) was significantly faster than the mean obtained to single targets (5 df $p= 0.033$). However, the mean obtained when the non-attended target appeared 240 ms (LBR -240 ms) was not faster than when a single target was presented, although the -160 and -240 ms mean saccade latencies are similar. It is thought that this could be due to the results of subject ZAC who showed a slowing of saccade latency under these two conditions of bilateral presentation.

A further examination of the data was performed with the means obtained from ZAC excluded. The mean latency obtained from five subjects excluding those from ZAC are shown in Figure 11. This was thought to be justifiable as this subject's results are clearly different to those produced by the other five subjects (Figure 9). Figure 11 shows a similar pattern of inhibition and facilitation as was shown in the results from all six subjects (in Figure 10).

A (two factor) ANOVA was performed on these mean latencies. There was no significant effect of eccentricity ($F(1,4)=2.72$ $p=0.1745$), and no interaction effect ($F(10,40)=0.5$ $p=0.839$). The factor of gap was shown to be highly significant ($F(10,40)=8.2$ $p=0.000$). A Newman-Keuls post-hoc analysis was performed on the means. A non-attended target appearing 160/240 ms before the onset of the saccade target, produced a mean of some 150 ms. This is significantly faster than the mean obtained with a non-attended targets at all other gap intervals at the $p<0.05$ significance level, and significantly faster than the means obtained with gaps of: -40, -20, 0,+20,+40,+80 ms, at the 0.01 significance level. Bilateral simultaneous target presentation produced a mean latency of 188 ms, which is significantly slower ($p<0.05$) than the mean latencies which are below the single target baseline (ie -240, -160, -80, +160 and +240 ms gaps). There was no significant difference between the means obtained when the non-attended bilateral target appeared simultaneously and when it appeared 20, 40 and 80 ms, before and after, the attended target.

Paired t-tests were again performed on the single and bilateral means to examine the facilitatory and inhibitory effects of the non-attended target. The results of this analysis were the same as shown for the analysis with the six subjects mean latencies. The mean obtained when a non-attended target appeared 160 ms before the onset of the saccade target (LBR -160 ms) is significantly faster than the single target mean. However, the mean obtained in the LBR -240 ms is not significantly faster than the single target mean. A further t-test compared the means

obtained in the -240 and -160 ms conditions and showed that there was no significant difference between these two means. This selective facilitation effect could be due to the variability of the five subjects means. Mean latency obtained with simultaneous target presentation is significantly slower than the single target mean ($p<0.01$). The mean latency obtained when a left target appears 40 ms after the onset of the saccade target (RBL +40 ms) is significantly slower than the single target mean ($p<0.05$).

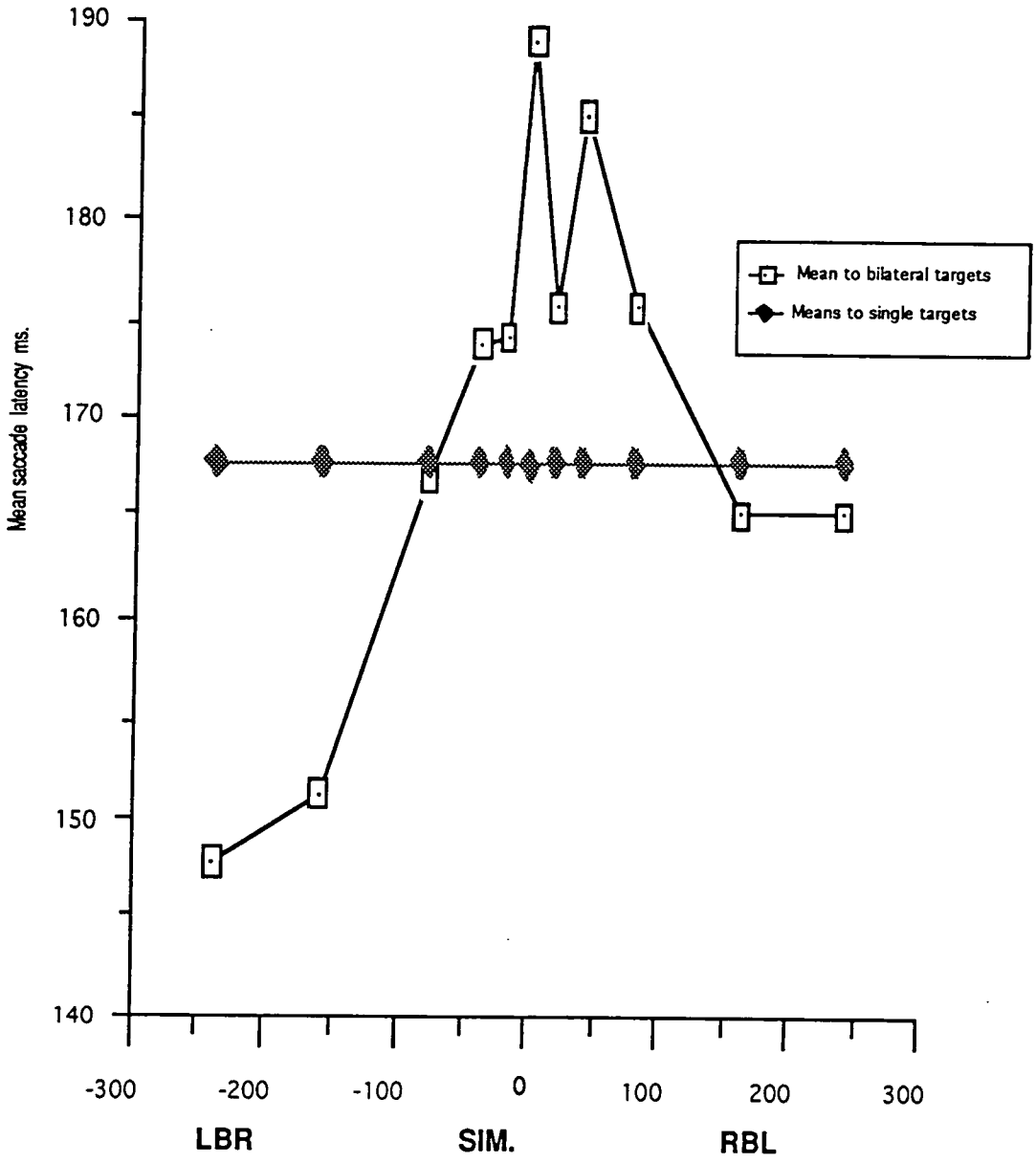


Figure 12.
Mean saccade latency obtained from five subjects (excluding ZAC) in Experiment 7.

The data points show the mean latency obtained to a target in the attended hemifield following an onset in the non-attended hemifield at the gap interval shown.

3.3.4 Discussion.

The results of the present experiment showed that presenting a target in the non-attended hemifield differentially affects saccade latency, depending on the time course of the 'distractor' target presentation. When the non-attended target was presented at long intervals before the saccade target (LBR 160/240 ms) it had a facilitatory effect, speeding saccade latency compared to that obtained with single target presentation. The speeding (of mean latency) reached significance when the non-attended target appeared some 160 ms before the saccade target. This is consistent with the warning signal effect, shown with prior fixation onset by Ross and Ross (1980, 1981). A target appearing in the non-attended hemifield at a long time interval (160/240 ms) after the saccade target has been presented had no effect on saccade latency. This finding is not surprising as the saccade should have been executed, by the time the target appeared in the non-attended hemifield.

When targets appeared bilaterally and simultaneously the mean saccade latency obtained was 185 ms, some 17 ms slower, than that obtained to single targets (168 ms). The mean obtained with simultaneous target onset is slower than obtained in any other bilateral target condition. A non-attended bilateral target appeared at short time intervals (20/40 ms) before, or after, the onset of the saccade target, resulted in the mean saccade latency being slower than was obtained to single targets, but this slowing was not as great as occurred with simultaneous target onset. Mean latency was slowest with simultaneous target presentation and a gradual decrease in latency is shown when the non-attended target appears at short intervals (20, 40 ms) before the saccade target. This is different to one of the predictions made in the introduction, that the onset of a non-attended target at short intervals before target presentation, could produce slower mean latency than simultaneous target onset, as the contralateral attentional inhibition should have had time to reach its maximum level. There are two possible explanations to account for why the mean saccade latency was faster than was observed with simultaneous target onset. The first is simply that the automatic increase of inhibition for the contralateral hemifield is produced very quickly, and has peaked and already started to decrease as early as 20 ms after the initial onset of the non-attended target. This would give a gradual decrease of saccade latency with increasing intervals between non-attended and saccade targets, as was shown. The second possibility is that the level of inhibition is comparable at intervals of 20 and 40 ms to that which is present with simultaneous onset, but there is also some additional benefit obtained by the warning signal effect of early onset of the non-attended target. The small benefit being due to the same warning effect that enables partial preparation of the covert processes required to make a saccade, as has been shown with prior onset of fixation (Braun and Breitmeyer, 1990). When the saccade target appeared the decision when to initiate a saccade could have been partially completed, thus producing faster latency than occurred with simultaneous target onset. The early onset of the non-attended target could produce a small benefit due to a warning signal effect, but with simultaneous target onset there is no advanced warning signal effect, while the level of inhibition in the attended hemifield is the same.

The combination of warning signal and inhibition results in the slowest mean latency being produced in the simultaneous condition (no warning signal, but inhibition) and some speeding being shown with early onset of the non-attended target (warning signal and inhibition). A future experiment could examine this possibility by presenting a non-attended target before and after saccade target onset as used in this experiment, but by reducing the predictive value (and hence the warning signal effect), of the non-attended target. This could be achieved by including a large number of catch trials in which a saccade target was not presented on some trials following the non-attended target onset. This should reduce the warning signal effect and give a rise in the mean latency obtained when a non-attended target is presented before the saccade target.

The prediction that the slowing of saccade latency should be less when the non-attended target appeared after the saccade target was shown in the results obtained. It was suggested that this could be expected as the saccade target occurred before any inhibition has been generated by the onset of the non-attended target. This indeed seems to be the case, as the slowest mean latency was shown with simultaneous target onset and latency then decreased when a non-attended target appeared shortly after the saccade target onset. This suggests that the inhibition is generated quickly and has its maximum effect on saccade latency, when it is produced before saccade target onset. A second possibility is that mean latency is less when a non-attended target appeared after the saccade target, than in the simultaneous onset condition, because of a warning signal effect. The results of previous experiments caution against the idea of a warning signal effect obtained by a stimulus onset which occurs after the onset of the saccade target. The experiments of Ross and Ross (1980, 1981) provided convincing evidence that a stimulus onset appearing after the onset of the saccade target, has a strong interference effect on saccade latency. So the presence of a warning signal cannot account for latency being faster than is the case with simultaneous onset. Instead, the inhibitory effect on saccade latency can be explained by the presence of inhibition produced in the saccade target hemifield by the non-attended contralateral target. The effect of this inhibition should be less pronounced if the saccade target has already appeared.

The pattern of results obtained provides some evidence against the idea that the slowing observed on bilateral trials is due to the cancellation of the saccade programme, which is prepared covertly, prior to target onset. The programme is assumed to have been prepared covertly to make a saccade in the attended direction, but this programme is then 'cancelled' by the onset of a non-attended target, resulting in a delay due to the time taken to restart the decision process (Aslin and Shea, 1987). In the present experiment a non-attended target that appears after the saccade target (RBL) would cancel the previous decision process. The time taken to produce a saccade would then presumably start from the time of the onset of the non-attended target. The earlier the appearance of the non-attended target, then the more time there is to start the covert programming processes and the shorter the saccadic reaction time will be. This would result in faster saccade latencies being produced when the non-attended target onsets occurs soon after the saccade target onset (eg. RBL +20 ms); and slower mean latencies when the saccade is cancelled at a longer interval following saccade target onset (eg.

RBL +80 ms). This trend is not shown in the data of the present experiment. Slower saccade latencies are shown when the non-attended target appears soon after the saccade target (RBL 20 ms) and faster saccades are show when the non-attended target appears at a later interval (RBL 80 ms). The model of Aslin and Shea (1987) was developed to explain the latencies obtained in experiments in which the subject is expecting to make a saccade to two target steps and they would therefore be expecting to have to change the direction of the programmed saccade. The present experiment always required a saccade to be made in one direction. In this instance it appears that the onset of a target in the contralateral direction does not automatically cancel the decision process. This provides further evidence to support the role of attentional factors that need to be incorporated into models of saccade generation.

The facilitation effect observed with the early onset of a target in the non-attended hemifield can be accounted for in the light of previous findings. It has been shown for example, that prior fixation offset and onset, both speed saccade latency, which could be due to a warning signal effect (Ross and Ross, 1980; 1981). However, the onset of fixation before target onset produced a facilitation effect, while the onset of fixation after target presentation, produced an interference effect. A warning signal effect can account for the reduction in saccade latency obtained with the onset of a target in the non-attended hemifield at long intervals (160/240 ms) prior to the onset of the saccade target. The exact nature of this warning signal effect may be explained in terms of the models of saccade generation proposed by Becker and Jürgens (1979) and Aslin and Shea (1987). In this instance the early onset of a target in the non-attended hemifield could trigger the processes involved in saccade initiation. The 'when' signal is triggered by the early onset allowing covert processes to begin with the resulting reduction in saccade latency when the saccade target appears. The facilitation effect observed by the onset of a non-attended target could also be explained in terms of an attentional model of saccade generation. The attentional explanation of the gap effect proposed by Fischer and Breitmeyer (1987) could be applied to the facilitation effect obtained in this experiment, with the prior onset of the non-attended target. In this instance the early appearance of the non-attended target prepares the system to disengage attention from fixation, before the saccade target appeared. This enables the system to be fully disengaged prior to the onset of the saccade target, with the resulting speeding of saccade latency.

The results of this experiment are similar to those obtained by Braun and Breitmeyer (1990) who examined the effects of the reappearance of fixation on saccade latency. They showed that the onset of fixation had its greatest effect, 100 ms before, to 150 ms after, the saccade target onset. The fastest mean latency occurred when the fixation point appeared over 100 ms before target onset. The slowest mean latency was shown when fixation onset occurred at the time of target onset. Latency decreased as the interval between onset of target and fixation increased up to 150 ms, performance then stabilised. They interpreted their results in terms of the findings of Ross and Ross (1980, 1981) where prior onset produces a facilitation due to a warning signal effect and onset after target onset has an interference effect. The facilitation effect observed when fixation onset occurs before target onset being explained either by attentional disengagement, or a warning signal effect.

The patterns of inhibition and facilitation obtained with bilateral presentation of targets at various intervals before, and after, the saccade target in this experiment can be explained in terms of saccade preprogramming and attentional inhibition. The onset of a target in the non-attended visual field can be expected to produce a short lasting inhibition in the contra attentional field. This explains the slowing of saccade latency with simultaneous target presentation and when the non-attended target appeared at short intervals after the saccade target. When the non-attended target appeared before the saccade target there are two processes which could affect saccade latency. The first is the production of the contralateral inhibition, which slows saccade latency at the 20 and 40 ms intervals, but which has decreased by the 80 ms gap intervals. At longer intervals the early onset of the non-attended target acts as a warning signal that a saccade is to be made, thus enabling the decision process to be preprogrammed before target onset. The decision to execute a saccade could be started with early onset of the non-attended target which is reflected in the decrease of latency as the gap intervals increased. This trend is shown in the results with saccade latency dropping with increasing gap intervals, reaching its optimal value with gaps of 160 ms. The saccade system should be ready to generate a saccade after some 160 ms so a target appearing at this time obtains the greatest facilitation effect. When a single saccade target appeared, or when the non-attended target appeared after the saccade target, then there is no preprogramming of the decision mechanism and no facilitation effect. The extra factor of an automatic increase of inhibition in the right hemifield accounts for the slowing observed with simultaneous target presentation and when the non-attended target appeared at short intervals before the saccade target.

3.4 General discussion.

In Experiment 6 single and bilateral double targets were presented while the subjects directed their attention into one hemifield. The bilateral saccade targets appeared simultaneously at equal and opposite eccentricity locations. In the experiments already described in Chapter two, the failure to reduce the slowing observed with bilateral targets on attentional trials, may be explained due to the possibility that the subject did not preprogramme saccade direction because a contralateral single target was presented on one third of all trials. On neutral trials an extra decision process being required to select a direction, to saccade towards on bilateral trials. This same conflict of direction would also occur on attentional trials, if the subject fails to preprogramme the saccade direction, due to the presence of contra-attentional single targets on one third of all trials. Saccade direction remained constant in Experiment 6, but the mean saccade latency obtained to bilateral targets was still significantly slower than was observed with single targets. This suggests that the slowing produced by bilateral target presentation, may not be due to oculomotor factors.

The second possibility is that the onset of a target in the non-attended hemifield cancels the existing saccade programme, resulting in an extra time being required to

programme the saccade following target onset. Models of saccade direction such as that of Becker and Jürgens (1979) and Aslin and Shea (1987) involve separate and parallel processes which control the initiation and the amplitude of the resulting saccade. The model of Becker and Jürgens used two separate decision components, one for movements to the left and one for movements to the right. The onset of two targets would result in a conflict produced by activity in both initiation components. It is not clear in this model how the system will finally select one direction in favour of another with simultaneous presentation. Given that the attentional instruction results in subjects always saccading to the attentional target the system should also have access to higher level cognitive factors to select a direction. The model of Aslin and Shea (1987) used a single decision making component for both directions. The direction in which to make a saccade being indicated by the onset of the target. This location is continuously checked and marked until the saccade is initiated. Again it is not clear how this system could select one bilateral target in favour of another, when they appear simultaneously. The double step experiments used by both Becker and Jürgens and Aslin and Shea, showed that if a decision process is cancelled by an onset in the contralateral hemifield, there will be a time delay while the decision process is again completed. Aslin and Shea suggest that this takes some 50-70 ms, while Becker and Jürgens suggest it takes some 160 ms, the same time as required to generate a saccade to a single target. In Experiment 6, the attentional instruction should have enabled covert processing of the decision process. The onset of the simultaneous targets could cancel this existing decision process, in the same way that a double step target into the opposite hemifield cancels the decision. However, the slowing observed with bilateral targets in Chapters two and three, was in the region of 19-35 ms, which is less than the time thought to be required to compute the decision process. It appears that bilateral target presentation does not slow saccade latency due to the cancellation of any preprogramming of the decision mechanism that could have occurred covertly prior to target onset.

Experiment 7, examined the time course, of the slowing produced by bilateral double target presentation. It provided further evidence against the slowing effect being due to a cancellation of the saccade decision process by the non-attended target onset. If this was the case then an onset in the non-attended hemifield after the onset of the saccade target should produce slower saccades than simultaneous target onset. This is because the decision process will have to be computed from the time that the onset occurs in the non-attended hemifield. However, the results showed that the greatest slowing of saccade latency occurred with simultaneous target onset and this slowing decreased when a non-attended bilateral target appeared after the saccade target.

An examination of the time course of the effect on saccade latency of presenting bilateral targets, showed that when a target appeared at a long interval (160 ms) before the onset of the saccade target a facilitation effect was observed. It was thought that this facilitation effect could reflect a warning signal effect to enable processes of the saccade programme to be performed covertly. This effect is not shown when the non-attended target is presented 80 ms before the onset of the saccade target. Targets presented soon before the saccade target and soon after the saccade target slow saccade latency, with the greatest slowing being shown for

simultaneous target presentation. It appears that the appearance of a target in the non-attended hemifield, has two effects. The first is to produce an automatic inhibition for the contralateral hemifield (attended hemifield) which slows saccade latency. This inhibition is relatively short lasting and has started to dissipate by some 20 ms after the onset of the non-attended target. The second is to provide a warning signal effect to enable preparation of some aspects of the saccade programme. This warning signal can reduce saccade latency if presented at a long interval before the saccade target. When it is presented a short interval before the saccade target, the benefits obtained by the warning signal effect are reduced by the automatic activation of contralateral inhibition. The greatest slowing is observed with simultaneous target presentation due to the lack of the warning signal effect and the maximum amount of inhibition.

The suggestion is that the non-attended target exerts an inhibitory influence on saccade latency due to attentional inhibition. In addition there is a facilitatory effect due to the preprogramming of the saccade decision processes. These possibilities are examined in Chapter 8 where a functional model of visual attention is proposed which can explain the results obtained in the present experiments.

Chapter 4

An investigation into the costs and benefits obtained on saccade latency following the central cueing of visual attention.

4.1 General Introduction.

The aim of this chapter is to investigate the 'premotor' model of visual attention (Rizzolatti et al., 1987; Tassinari et al., 1987; Umiltá et al., 1991). The central argument of the premotor model, is that the system used to orient visual attention covertly, is the same as that involved in producing an overt saccadic eye movement. When attention is oriented covertly, in manual reaction time experiments, a saccade is planned to be made to the cued location, but the final decision to execute the saccade is prevented. It has also been shown that it is not possible to make an eye movement without attention moving to the corresponding location (Shepherd, Findlay and Hockey, 1986). One prediction of the premotor model is that a similar pattern of results should be obtained when attention is cued and saccadic reaction times are measured, as are shown when attention is cued and manual reaction times are measured.

The next section reviews some of the background to the premotor model of visual attention and outlines some of the limitations in terms of current models of saccade generation. The third section is an experimental examination into the premotor model, by replicating the experiment performed by Rizzolatti et al. (1987), but measuring saccadic reaction times instead of manual RT's. There are two primary aims of this replication. The first is to show if the central cueing procedure used by Rizzolatti et al., produces a large costs, and small benefits, on saccade latency as was shown in the experiments described in Chapters two and three. This is important as these experiments used a verbal instruction to direct attention on each block and it is possible that this is not equivalent to directing attention by a symbolic cue (that requires cognitive interpretation on each trial) as is often used in manual RT experiments. The aim is to show if a similar pattern of costs and benefits occur with saccade latencies, as was shown in Rizzolatti et al.'s(1987) manual reaction time experiment. If a similar pattern of results is obtained with saccade latencies as was shown in the initial manual RT experiment then the premotor model gains further support.

4.2 Introduction to the premotor model of visual attention.

Rizzolatti et al. (1987) based their premotor model of visual attention on the findings from a manual reaction time experiment. In this task subjects were required to orient attention covertly, to one of four target locations, following a number cue (at fixation), which indicated the likely target location. The target locations were marked by four stimulus boxes, which were arranged either horizontally, or vertically, above or below the fixation point. Subjects were required to make a manual key press when they detected a target stimulus which appeared at one of the four locations. The experiment was designed to discriminate between the spotlight

and hemifield inhibition theories of visual attention. Two variations of the spotlight theory were considered: attention moves with a constant speed across visual space (Shulman et al., 1979 and Tsal, 1983); or, attention jumps across space in constant time comparable to the way in which a saccadic eye movement occurs (Remington and Pierce, 1984). The predictions from these two variations of the spotlight theory are different in terms of the expected costs and benefits of orienting attention. The constant speed hypothesis predicts that costs will increase with distance from the cued location, while the constant time hypothesis would predict no increase of RT's with greater distances between the cued and target location. The hemifield inhibition model of Hughes and Zimba (1985; 1987) is an alternative to the spotlight models of attention and proposes that directing attention within one hemifield produces a broad area of inhibition for the non-attended hemifield. This was also considered in relation to the results obtained. According to the inhibition account there will be large costs for targets occurring in the non-attended hemifield, regardless of distance from the cued location without any costs for targets appearing at an uncued location in the attended hemifield.

Rizzolatti et al. (1987) found a significant 6 ms benefit on RT's for valid targets, and a large cost of 41 ms for invalid targets in the opposite hemifield. They termed the large costs shown for targets in the opposite hemifield the 'meridian effect' as it occurred for an attentional movement across either the horizontal, or vertical, meridian. A smaller cost of some 18 ms, was shown for mean reaction times to invalid targets presented in the attended hemifield, indicating that the mean on invalid trials was significantly slower than the mean for valid trials. Costs in the opposite hemifield were found to show a graded increase, with increasing distance from the cued location. The presence of costs within the attended hemifield argues against Hughes and Zimba's hemifield inhibition model, which only accounts for costs in the opposite hemifields. The hemifield inhibition model and the constant speed (spotlight) model of attention were not supported by these findings, as both predict similar costs regardless of the distance from the cue to target location. Rizzolatti et al. explain their results in terms of a model of motor planning, which involve the programming of the direction, and exact distance of the motor response independently and in a hierarchical series (Rosenbaum, 1980). Rizzolatti et al. suggest in the premotor model that the same neural system involved in the production of overt saccadic eye movements is also responsible for the covert orienting of attention. A similar model has been suggested by Tassinari et al. (1987). Programming a saccade could involve first specifying the direction in which a movement is required, and then the exact distance to be moved. A large cost in manual RT's and saccade latencies, will be incurred when a target appears in the non cued hemifield and the direction programme has to be cancelled. A smaller cost will be incurred for non cued targets in the attended hemifield which only require the amplitude component to be changed. A small benefit will be obtained for valid targets as the direction programme can be preprogrammed before target onset.

The premotor model of attention was examined by Umiltá et al. (1991) who replicated and extended Rizzolatti et al.'s (1987) experiment to incorporate the use of peripheral cues as well as central cues. Peripheral cues should operate on the 'automatic' attentional orienting component, while the central cueing procedure should operate on the 'voluntary' orienting

system. The central cueing results replicated the findings of Rizzolatti et al. producing significant benefits for targets at the cued location. A small cost was shown for non-attended targets in the attended hemifield and a larger cost was obtained for invalid targets in the non-attended hemifield showing the extra cost incurred for crossing the vertical meridian. However, the peripheral cues produced a different pattern of results. A significant benefit was shown for valid targets, but the costs produced to invalid targets were similar in both the attended and non-attended hemifield, showing that the meridian crossing effect does not occur with peripheral cueing. Umiltá et al. regarded the failure to obtain the meridian effect with peripheral cues as being congruent with the premotor model of attention. They explain the lack of the meridian effect in terms of an 'inhibition of return' operating within the saccade generation system. The peripheral cue is assumed to activate a motor programme for a saccade to be made in the direction of the peripheral cue. This programme is aborted as the subject is instructed not to make an eye movement, which causes a transient bias against making a movement in the same direction. The meridian effect will be reduced as a movement within the cued hemifield will now be inhibited, but a movement in the opposite hemifield will not be affected. This inhibitory component is thought to be different from inhibition of return (Maylor and Hockey, 1985) and may operate at the level of neurons in the superior colliculus. Collicular neurons have been shown to be inhibited by a stimulus which falls outside their receptive field (Rizzolatti et al., 1974) and this inhibition is greater on the side ipsilateral to the attentional stimulus. This would result in a slowing of responses to stimulus located in the cued hemifield.

Crawford and Müller (1992) performed a similar experiment to that of Umiltá et al. (1991) and examined the effects of peripheral cueing on both saccade latencies and manual RT's. They found significant benefits of peripheral cueing on saccade latency for SOA's of 100 ms, but not for SOA's of 500 ms. The benefits of the cue were apparent for the exact cued location only, there was no advantage for saccades made in the same hemifield as the cue, and no advantage for targets sharing the same amplitude as the cue but presented in the opposite hemifield. The meridian crossing effect was not shown in saccade latencies, with invalid latencies being comparable in the cued and non-cued hemifields. Crawford and Müller's failure to find the meridian crossing effect in saccade latencies using peripheral cues, is consistent with Umiltá et al.'s failure to find the meridian effect with manual RT's. However, in contrast to Umiltá et al. (1991), Crawford and Müller did obtain the meridian crossing effect with manual RT's using peripheral cues. In this instance RT's to valid targets were faster than to invalid targets showing a benefit of peripheral cueing at both SOA intervals. Manual RT's to invalid targets in the cued hemifield were faster than to invalid targets in the opposite hemifield indicating a significant effect of cueing direction on manual reaction times which was not shown for saccadic eye movements. Crawford and Müller argue that as the peripheral cueing procedure has produced a different pattern of costs on saccade latencies to those shown with manual RT's, the premotor model of attention is not supported. The findings of Crawford and Müller appear to be convincing in arguing against a similar premotor mechanism being responsible for overt and covert movements of attention, but the conflicting findings of Umiltá et al. weaken this conclusion. As it is questionable if the meridian crossing effect is obtained with peripheral cueing and manual RT's, it may not be surprising that the meridian effect was absent in Crawford

and Müller's saccade latencies following peripheral cueing. The experiments described in Chapter two provide strong evidence to suggest that the meridian effect is obtained with saccade latency following a verbal instruction, so it is of interest to show if the meridian crossing effect is also shown in saccade latencies following the central cueing procedure used by Rizzolatti et al. (1987).

Hughes and Zimba (1987) in contrast to Rizzolatti et al. (1987) found relatively uniform RT performance for invalid (probe) locations within the attended hemifield (although one probe location showed a significant cost) and large costs for all probes in the non-attended hemifields. A large dip was shown in RT performance, for targets appearing at the 'marked', mirror image location, in the non-attended hemifield. A closer examination of Hughes and Zimba's results showed that the pattern of costs obtained in the non-attended hemifields was not equal for all probes and did increase with eccentricity, for example mean RT's to invalid probes at 2^{o} are 227 ms, while probes at the 6^{o} location are 238 ms which indicates that costs on RT's are not equal in the non-attended hemifield. In a further experiment (Hughes and Zimba, 1987 exp. 3) attention was directed along the oblique axis, RT performance was found to be poorest for probes presented in the quadrant diagonally opposite to the attended quadrant. Hughes and Zimba suggest that the spatial effects of directing attention operates in terms of two broad inhibitory distributions, one for the left and right hemifields and one for the upper and lower hemifields.

Zimba and Hughes (1987) claimed that the marker boxes used in many experiments of covert orienting of attention to indicate target locations in the visual array may actually have an additional interference effect on RT performance. They showed that the use of marker boxes produced an extra slowing on RT's when compared to those obtained in an unmarked visual field. This slowing is greatest for invalidly cued locations and increases with eccentricity, but does not affect valid RT's. Zimba and Hughes suggested that this increase of invalid RT's can explain why many experiments of attentional orienting which compare valid RT's to invalid RT's, obtain costs which appear to increase with target eccentricity and may also explain why cost-benefit analysis often reveals a cost within the attended hemifield. In effect they suggest that the use of marker boxes produces an interference effect which increases invalid RT's and produces a pattern of results which is consistent with the spotlight metaphor of attention. If an unmarked visual array is used then the results appear to confirm the hemifield inhibition accounts of attention.

The hemifield inhibition account of attention could be incorporated into the premotor model of attention by assuming that attending in one direction (eg. left) is the equivalent of inhibiting a movement in the opposite direction (eg. right). In terms of saccade generation planning a left saccade is the same as inhibiting the initiation of a saccade to the right. This assumption can be incorporated into models based on experiments in which a cue served to preprogramme parameters of the saccade to the cued location. Invalid targets in the attended hemifield will only be slowed slightly as the amplitude computation alone requires a small change, while a target in the non-attended hemifield will have to be preprogrammed which requires overcoming the attentional inhibition for a movement in that direction, which produces

requires overcoming the attentional inhibition for a movement in that direction, which produces an extra slowing on both saccade latencies and RT's.

Although the premotor model of visual attention is attractive it makes assumptions about the process of saccade generation which are not entirely supported by research into the oculomotor system. Becker and Jürgens (1979) model of saccade generation incorporate the idea that the programming of saccade direction is carried out independently from the programming of its amplitude. This model has been examined in terms of the effects of directing attention on the amplitude and direction component in an experiment by Gorea, Findlay and Lévy-Schoen (unpublished). The attentional manipulation involved varying the probability that targets occurred at 3°, or 15°, locations (left and right of fixation) on the horizontal axis, under conditions in which saccade amplitude was predictable and when saccade amplitude and direction were both predictable. Saccade latency was reduced when the amplitude and direction were predictable compared to when amplitude only was predictable. Saccades in the opposite direction showed the characteristic increase in latency as shown in manual RT experiments. An interaction effect was shown for target eccentricity and target probability: if the near target location was highly probable latency to a target at the far location increased by some 20 ms; in contrast if the far target location was highly probable saccade latency to the near location did not show any increase. This result implies that attending to a point in the periphery involves attending to all intermediate locations from the fovea to that point.

The large increase in saccade latency when a change in the saccade direction programme is required has also been indicated in 'two step tracking' experiments in which a saccade is made to a target which moves in a step jump (Aslin and Shea, 1987; Becker and Jürgens, 1979; Findlay and Harris, 1984). Findlay and Harris (1984) showed that the onset of a second target has an inhibitory effect which makes the release of a saccade less likely. The strength of this inhibitory effect is smallest with a second step on the same side of fixation as the first step and greatest if the second step was in the opposite hemifield to the first. However, they do not interpret their results as supporting the idea that amplitude and direction of a saccade are programmed separately. The 'cancellation' of saccade direction explanation of the extra cost of a step which crosses the vertical meridian is weakened by the finding that the inhibitory effect of the double step into the opposite hemifield was reduced, if the second step crossed the vertical meridian at a point away from the central region. Findlay and Harris suggested that saccade amplitude and direction are programmed jointly in a retinotopic motor map which is involved in performing the motor response required to make an appropriate eye movement similar to the motor map suggested by McIlwain (1976). McIlwain (1986) proposed a collicular model of saccade generation, in which the spatial location of a stimulus falling on the retina is mapped onto a corresponding area of the superior colliculus. The direction and amplitude of the final saccade is controlled by the level of activation within the cells that are connected to the horizontal and vertical pulse generator and therefore control the final motor command. Findlay (1987) proposed a similar motor map model of saccade generation incorporating some of the suggestions of McIlwain (1976; 1986). Findlay's model showed how a retinal input can be transformed into a motor output (saccade) by a transformation of the retinal

a retinal input can be transformed into a motor output (saccade) by a transformation of the retinal position of the stimulus into a pattern of activity which is mapped onto the superior colliculus. The deep layers of the superior colliculus have been shown to be involved in producing a motor output (Robinson, 1972). The sensorimotor transformation is thought to take place by activating the appropriate premotor units for a saccade, this coding being performed by the spatial encoding of the desired saccade size. The models of Findlay (1987) and McIlwain (1986) propose that saccade amplitude and direction are computed in a more holistic way to the separate programming model of Becker and Jürgens (1979).

The experiment described in this chapter performs an examination of the effects of cueing attention to one target location and measuring the effect on saccade latency. The aim is to examine the premotor hypothesis of visual attention, that proposes a strong link between the underlying attentional and saccadic orienting systems. Some of the models of saccade generation should be considered in relation to the results obtained.

4.3 Experiment 8: The effect of amplitude and direction cueing on saccade latency.

4.3.1 Introduction.

This experiment is a direct replication of the experiment performed by Rizzolatti et al. (1987), but instead of measuring manual reaction times will measured the latency of saccades made when attention was oriented using their cueing procedure. Rizzolatti et al. (1987) used a central cueing procedure to orient attention covertly, to target locations and obtained a small benefit for targets at the cued location and much larger costs for targets in the non-attended hemifield. This is similar to the pattern shown in the saccade experiments already described in Chapters two and three. Their procedure also revealed a pattern of costs for targets presented at non-attended locations *within* the attended hemifield. The procedure used in the previous experiments does not enable this detailed examination to be performed, as subjects directed attention in a certain direction and not to a specific target location. According to the premotor model of attention a similar mechanism is used to direct attention covertly, as is involved in moving the eyes overtly. If this is the case a similar pattern of results will be expected to be obtained with saccade latencies when attention is cued using symbolic cues, as was produced with manual RT's. As Crawford and Müller (1991) failed to obtain the meridian crossing effect on saccade latency using peripheral cueing, it is important to examine if the meridian effect can be obtained with central cueing.

Given the findings of Zimba and Hughes (1987) that the use of marker boxes produces an increase in invalid RT's which could account for the graded increase of invalid RT's with increasing eccentricity, conditions with and without marker boxes were run to see if the same effect occurs on saccade latency. A third condition aims to examine the reduction of costs for crossing the vertical meridian shown for saccade tracking when a crossing of the vertical

meridian occurs away from the central region (Findlay and Harris, 1984), to show if the costs incurred are similarly reduced for an attentional movement across the vertical meridian. A final point of interest is to see if the use of the central cueing procedure produces a similar pattern of saccade latency, as was shown in the experiments in Chapter two which used a verbal cueing procedure.

4.3.2 Method.

Subjects

Six postgraduate students from the psychology department acted as subjects. The ages ranged from 22 to 32 years, all had normal or corrected to normal vision. One of the subjects (CV) had not participated in any of the previous experiments.

Apparatus.

The apparatus was identical to that already described for experiments 3, 4 and 5.

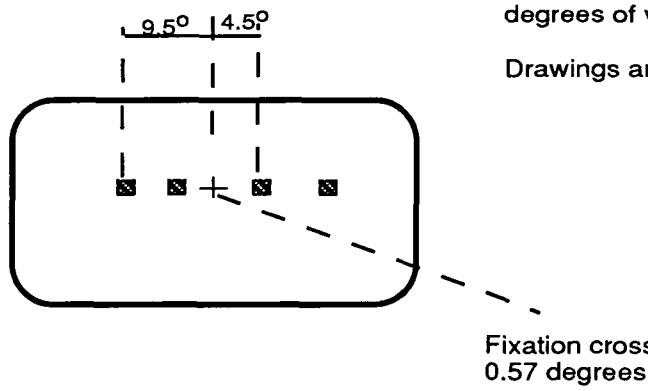
Stimulus display.

Subjects were tested on three display conditions (horizontal axis, horizontal axis with marker boxes and upper horizontal axis) on separate testing sessions carried out over three different days. Figure 13 shows the VDU display for the three conditions. In the 'Horizontal' axis condition the targets appeared 4.5° and 9°, left or right of fixation, on the horizontal axis in an otherwise empty visual array. The 'Horizontal-box' condition was identical except that the four target locations were marked by the presence of an unfilled square (sides 1.5° visual angle). In the 'Upper axis' condition the targets were presented 2.5° above the fixation cross, at the same eccentricities as in the two other conditions, without any marker boxes.

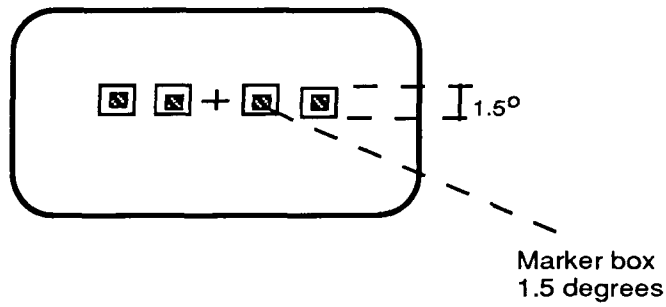
Horizontal axis condition.

All measurements are in degrees of visual angle.

Drawings are not to scale.



Horizontal-Box condition.



Upper axis condition.

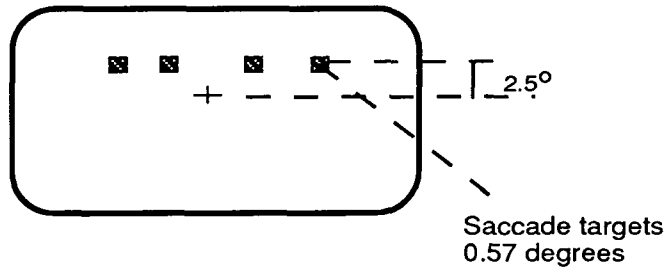


Figure 13.
Stimulus positions used in Experiment 8.

Subjects viewed the VDU at a distance of 50 cm, with their eyes level with the centre fixation cross. A central cross (size 0.6°), was presented for 500 ms, following which a symbolic cue (number digit 0 to 4) of size 0.6° visual angle, was presented for 1 second at the fixation location. The timing sequence after cue offset was identical to that shown in Figure 1. The target (square 0.57°) appeared at one of the four target locations for 100 ms, following the cue offset. A delay of 100 ms occurred, before the indicator stimulus appeared at the target location. The indicators (as used in the previous experiments) were presented for 300 ms, and were immediately followed by a mask for 100 ms. Subjects were required to indicate how many dots were presented by using a hand held button box. Accuracy of report was emphasised and an audible bleep signalled an incorrect response.

4.3.3 Procedure.

On each trial subjects fixated the central cross when it appeared on the screen. Following this a digit 'cue' appeared at the fixation location and indicated the likely target location. Subjects were informed that the target locations were numbered from one to four, starting with the left most location. They were instructed that the central number cue (1 to 4) indicated the location that they should direct their attention towards. On neutral trials a '0' was presented which indicated that the target was equally likely to appear at any of the four locations and subjects were instructed to direct their attention globally to all four locations.

The cue validity was the same as used by Rizzolatti et al. (1987). On 80% of trials a cue appeared (the digits 1 to 4) which indicated the target location with a validity of 70%, on the other 30% of trials the cue was invalid and the target would appear at one of the other three locations (10% each). There were 400 trials in each experimental condition, of which 80 (20%) trials were 'neutral' trials, where a '0' appeared indicating that the target was equally likely to appear at any of the four target locations.

Each subject was tested under all the three experimental conditions. Thirty practice trials were given at the start of each session and the order of testing on the three conditions was counterbalanced across subjects. The testing sessions were completed on separate days and each session lasted approximately 45 minutes.

4.3.4 Results.

Saccades with latencies of less than 90 ms, and over 300 ms, were not included in the analysis. Trials which showed double step saccades, indicating that the subject had initially moved their eyes in the wrong direction, were also removed from the analysis. This resulted in a total of 8% of records being excluded.

The first comparison on the data was to examine the costs and benefits of directing attention. Figure 14 shows the mean saccade latencies obtained for valid, invalid and neutral trials under the three conditions. It is important to realise that in this figure the mean latency for invalid trials, includes non-cued targets in both the attended and non-attended hemifields. The benefits (neutral - valid) for directing attention are shown to be in the range of 7.5 to 12.7 ms., while the costs (neutral - invalid) are in the range of 12 to 13.5 ms. Previous experiments (eg. experiment 1-4) obtained somewhat greater costs for invalid targets than are shown in Figure 16. This is explained by the way in which the data are grouped in terms of the 'valid' and 'invalid' trials. In the previous experiments valid referred to targets in the attended hemifield and invalid to targets in the non-attended hemifield only, and not as shown in Figure 16. in terms of a cued or non cued location. If the data from the present experiment are grouped in terms of valid and invalid hemifields, then the benefits obtained in the horizontal axis condition are 10 ms and the costs 23 ms which are much more like those obtained in previous experiments.

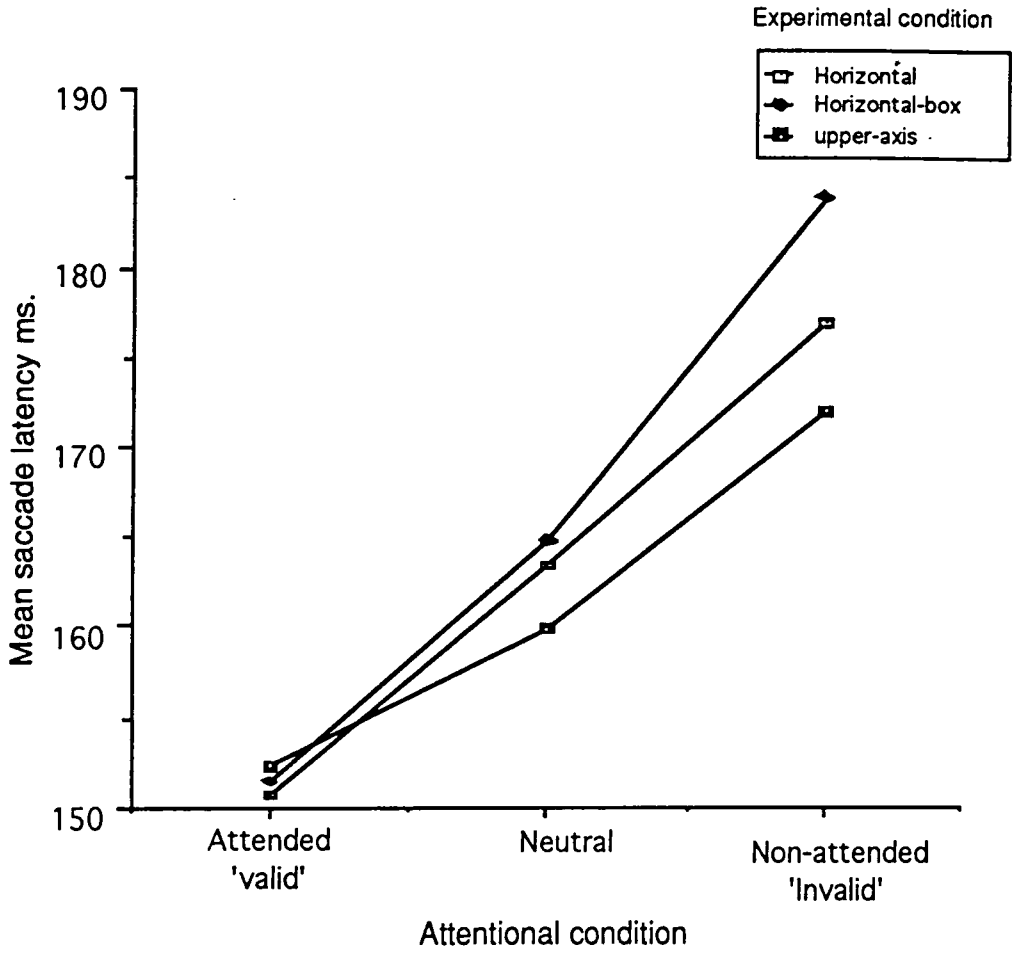


Figure 14.
Mean saccade latencies obtained in Experiment 8.
Saccade targets presented under the three attentional conditions shown

A (two factor) ANOVA was performed on the means shown in Figure 14, to examine the effect of experimental condition (condition 3) and target type (valid, invalid, neutral) on saccade latency. The analysis showed no significant effect of condition ($F(2,10) = 1.1$ $p=0.037$), a significant effect of target type ($F(2,10) = 14.2$ $p=0.001$), and a significant two way interaction between target and condition ($F(4,20)=3.56$ $p<0.024$). Post hoc analysis (Newman-Keuls) confirmed that the valid target mean of 151.5 ms was significantly faster than the neutral mean of 162.7 ms ($p<0.05$) and the invalid mean of 177.64 ms was significantly slower than the neutral ($p<0.05$) and valid ($p<0.01$) means. The significant interaction effect is indicated in Figure 14 which shows that the valid target means are similar in each of the three conditions, but the use of the marker boxes has produced an extra slowing on invalid trials. Post hoc analysis (Newman-Keuls) showed that the upper axis invalid mean (172.1 ms) was comparable to the horizontal axis invalid mean (176.9 ms), but the horizontal axis with marker boxes invalid mean (183.9) was significantly slower than the horizontal axis ($p<0.05$) and upper axis ($p<0.01$) invalid means.

In a second comparison latencies to saccades made in the left and right direction were collapsed, so the results were presented in terms of the attended and non-attended directions. The means obtained are shown in Table 8. and are displayed graphically in Figures 15 (a, b, c) for the three experimental conditions. The data are grouped so that it appears as if the left hemifield was the attended hemifield, and that the cues always indicated either target position 1, or, 2. Target positions 3 and 4 therefore represent the near and far target locations in the non-attended hemifield.

Table 8. Mean saccade latency. Data is collapsed so that the target positions 1 and 2 are 'cued' and positions 3 and 4 are 'uncued'.

	Target location.			
Horizontal-axis condition	1	2	3	4
	Mean (ms)	Mean (ms)	Mean (ms)	Mean (ms)
Cue far location (1)	150.15	153.55	186.8	178.74
Cue near location (2)	162.16	151.2	180.56	191.82
Neutral condition (0)	162.8	164.0	162.8	164.0
Horizontal-box condition				
Cue far location (1)	151.4	156.6	186.5	200.6
Cue near location (2)	166.2	151.5	189.0	203.45
Neutral condition (0)	171.2	158.4	171.2	158.4
Upper-axis condition				
Cue far location (1)	151.3	155.9	170.65	185.5
Cue near location (2)	168.25	153.5	169.45	184.12
Neutral condition (0)	162.8	156.8	162.8	156.8

An examination of Figures 15 (a, b, and c) shows that a similar pattern of results was obtained in each of the three conditions, but this pattern differs (slightly) from those obtained by Rizzolatti et al. (1987). To enable a direct visual comparison the mean manual reaction times obtained by Rizzolatti et al. in their horizontal axis condition, are shown in Figure 16. The pattern of results in the attended hemifield obtained by Rizzolatti, shows that there is a cost for manual RT's made to the near target following a far cue (target at 2: cue at 1) and a comparable cost for a RT to a far target following a near cue (target at 1: cue at 2). The pattern of results shown for targets in the attended hemifield is slightly different to the mean manual RT's. Mean saccade latency to the near targets in the attended hemifield is similar following a cue to either the near or far location (target at 2: cue at 1 or 2). However, mean saccade latency to the far target was slower following a cue to the near location (target at 1: cue at 2) than when the cue indicated the far target location (target at 1: cue at 1).

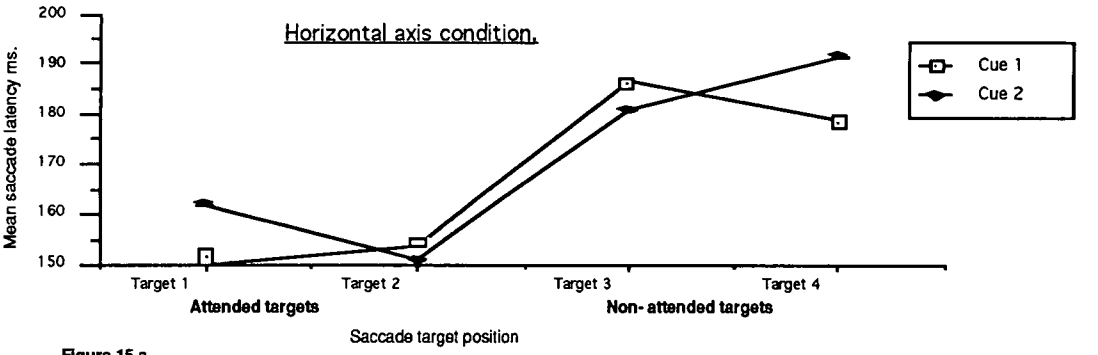


Figure 15 a.
Mean saccade latency obtained in Horizontal axis condition
Experiment 8.

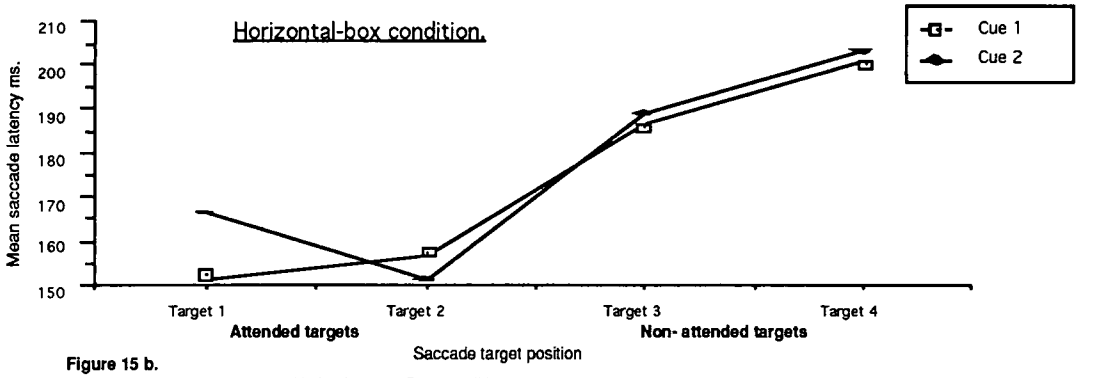


Figure 15 b.
Mean saccade latency obtained in Horizontal - Box condition
Experiment 8.

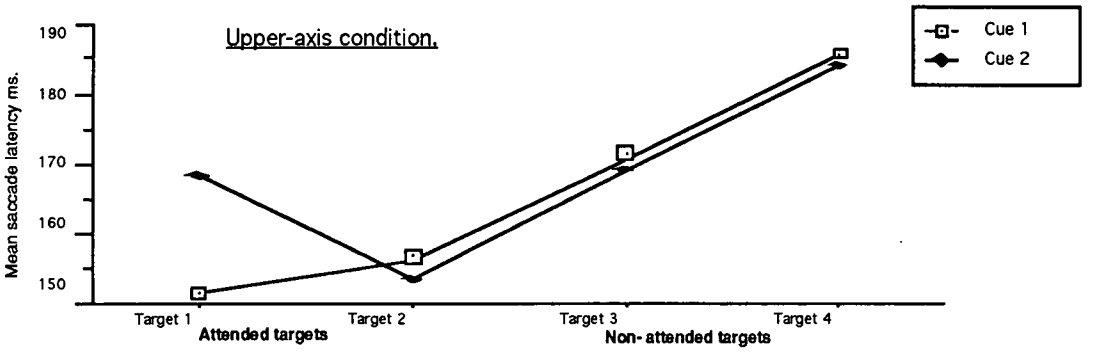


Figure 15 c.
Mean saccade latency obtained in Upper axis condition
Experiment 8.

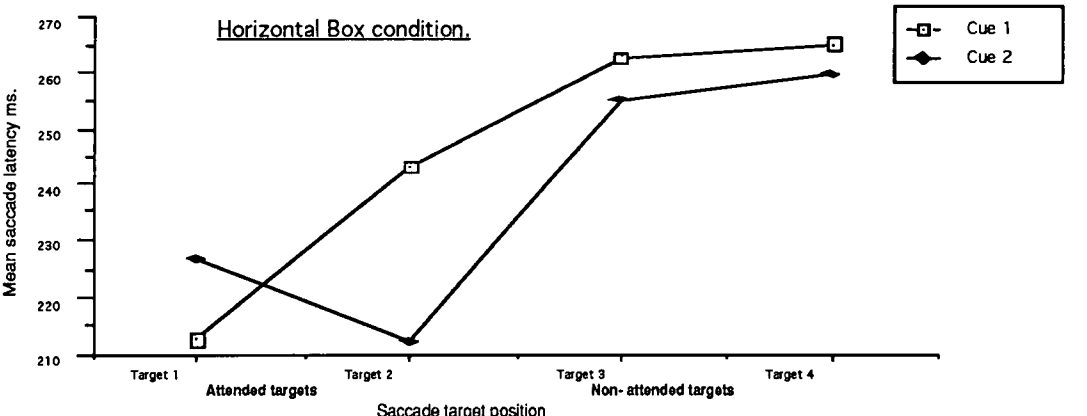


Figure 16.
Manual RT's obtained by Rizzolatti et al. (1987)

The latencies of saccades made to targets in the non-attended hemifield are greater than to invalid targets in the attended hemifield, showing an extra cost for crossings of the vertical meridian on saccade latency. The costs shown for targets in the non-attended hemifield in the horizontal axis condition (Fig 15 a), shows a somewhat surprising result that invalid target saccade latency is affected by the amplitude of the valid cue. This is shown by a reduction in latency for saccades made to invalid far eccentricity targets, following a cue to the far attended location. A similar reduction in invalid saccade latency is shown for invalid near eccentricity targets, made following a cue to the near attended location. This appears to show that the amplitude of a saccade made into the non-attended hemifield is cued by a cue indicating a location in the opposite direction (attended hemifield). In the horizontal-box and upper axis conditions (Fig 15 b and c), the latency to targets in the non-attended hemifield does not depend on the cue eccentricity, but shows an increase with the eccentricity of the target from fixation.

The selective effect that cue eccentricity has on the latency of saccades made to invalid targets in the non-attended hemifield obtained in the horizontal axis condition was examined in a two factor ANOVA. The first factor was target eccentricity (near/far), and the second factor was cue eccentricity (near/far). There was no significant effect of cue eccentricity and no effect of target eccentricity, but a significant interaction effect was shown ($F(1,5) = 9.8$ $p=0.025$). The significant interaction effect confirms that directing attention to a specific location in the attended hemifield facilitates a saccade made to targets at the same amplitude in the opposite (non-attended) hemifield. An examination of the individual results confirmed that this trend was shown in the results from all six of the subjects tested although the magnitude of this amplitude cueing effect varied from subject to subject. This trend was not shown in the horizontal-box, or upper axis conditions.

The results obtained in the horizontal axis condition were compared in two separate ANOVA's to those obtained in the horizontal-box and upper axis condition.

The first (four factor) ANOVA compared mean saccade latency obtained in the horizontal axis condition, to the means obtained in the horizontal-box condition. The four factors were: condition (Horizontal vs. horizontal-box), hemifield (attended vs. non-attended), target eccentricity (far vs. near), and cue eccentricity (far vs. near). The use of marker boxes produced a significant slowing on saccade latency, shown by significant effect of condition ($F(1,5) = 8.8$ $p=0.031$) indicating that the mean latency obtained in the horizontal axis condition (169.4 ms) was significantly faster than in the horizontal-box condition (175.51 ms). Saccades made to targets in the non-attended hemifield were significantly slower than to targets in the attended hemifield (attended= 155.34 non-attended=189.54 ms) ($F(1,5) = 35.01$ $p=0.002$). There was a significant two way interaction effect shown between experimental condition and target eccentricity ($F(1,5)=8.3$ $p=0.034$). The interaction occurred because under the horizontal axis condition saccade latency was comparable for targets at the near and far eccentricities (near=168.0 far=170.7 ms), but in the horizontal-box condition an extra slowing was produced for targets at the far eccentricity (near= 170.63 far=180.4 ms). Post hoc analysis

(Newman-Keuls) confirmed that the mean latency to far targets in the horizontal-box condition is significantly slower than to near and far targets in the horizontal axis condition ($p<0.01$). A second two way interaction effect was shown between target eccentricity and cue eccentricity ($F(1,5)=10.4$ $p<0.023$). This interaction can be explained as follows; saccade latency to the near target location was comparable following a cue to either the near or far locations, but latency obtained to far targets increased if the near location was cued. This result is compatible with the idea of an attentional spotlight with a variable beam size. The beam being broadly distributed following a cue to a far location, covering both the near and far target locations, but is narrowly focused following a cue to a near target location. This explanation would result in the selective slowing on latency obtained for saccades made to targets at the far location following a cue to the near target location.

A second (four factor) ANOVA was performed to compare the means obtained in the horizontal axis condition and those obtained in the upper axis condition. The factors were the same as in the previous ANOVA. The aim was to examine the prediction that costs should be smaller for targets in the non-attended hemifield if targets were presented above the horizontal axis. The main effect of condition was not significant indicating that saccade latency was not affected by presenting targets above the horizontal axis. The effect of attentional direction was highly significant ($F(1,5)=23.9$ $p=0.004$) indicating that latency was slower for targets in the non-attended hemifield. The two way interaction between target eccentricity and cue eccentricity was also significant ($F(1,5)=13.7$ $p=0.014$) which was again accounted for by the slowing of saccade latency to targets at the far eccentricity in the attended direction, following a cue to the near attended location. The mean latency obtained for targets at the near eccentricity in the non-attended direction can be seen to be faster (but not significantly faster) in the upper axis condition than in the horizontal axis condition, which is in line with the prediction of the use of the upper axis reducing the costs obtained when crossing the vertical meridian.

4.3.5 Discussion.

The results of this experiment have shown that the use of the symbolic digit cue produced a similar pattern of results, to those obtained in the previous experiments in Chapters two and three, in which attention was oriented by a verbal instruction. The digit cue in this experiment required cognitive interpretation on every trial, unlike the attentional instruction in the previous experiments which remained constant throughout a block of trials. The digit cue also differed from the verbal instruction, in that it accurately predicted both the target amplitude and target direction on valid trials. The magnitude of the costs and benefits of directing attention with the digit cue in this experiment are comparable to those obtained in Chapters two and three, which cued attention by a verbal instruction. A significant benefit in the region of some 10 ms was obtained for targets in the attended hemifield, and a larger cost of 23 ms occurred for saccades made to targets in the non-attended hemifield, indicating that the meridian effect was again obtained for saccade latency with a central cueing procedure.

The present experiment has shown that a large cost is incurred on saccade latency for movements of attention which cross the vertical meridian. This pattern was not shown by Crawford and Müller (1991) who used a peripheral cueing procedure to direct attention in a similar saccade experiment. Rizzolatti et al. (1987) used central cueing in a manual reaction time experiment and showed a significant benefit for targets presented at the cued location and large costs for targets in the non-attended hemifield which increased with the distance of the cue and target. Rizzolatti's premotor model of attention claims that the same mechanism used to orient attention covertly is the same as is used to make an overt eye movement. The large cost shown for crossing the vertical meridian is essential for this theory, as the meridian effect is explained in terms of reprogramming the direction of a motor programme which takes longer than does alteration to the amplitude programme. Umiltá et al., (1991) replicated the findings of Rizzolatti et al., (1987) using central cueing of horizontal target locations in the upper hemifield. They extended the experiment to incorporate the use of peripheral cues and failed to show the increase in invalid RT's for a crossing of the vertical meridian, in contrast to Crawford and Müller (1991) who did obtain the meridian effect with manual RT's and peripheral cues. Given the results of the present experiment which showed a strong meridian crossing effect on saccade latency with central cues, it appears that Crawford and Müller were premature in rejecting the premotor model of attention on the basis of of a peripheral cueing experiment.

Umiltá et al., (1991) extended the premotor model by considering two alternative models of motor programming. The first is that there is an independent computation of the vertical (up) and horizontal components (left/right) of the movement required to reach the target locations. The second possibility is that the programme requires the computation of a specific diagonal vector (eg. up/left, up/right) to move towards the target location. In both alternatives the meridian effect can be explained by the cancellation of the left/right component, which gives an additional delay to the alteration of the exact distance to be covered. The 'distance effect' which shows that RT's increase as the distance from the cue to target locations increases, is not essential to the premotor model. They suggest that the distance effect in manual RT's is comparable to the effect shown with saccade latency, where an increase of target distance are compensated for by an increase in the velocity of the eye movement, but this compensation does not succeed completely.

The presence of a large increase in saccade latency for invalid targets which cross the vertical meridian is compatible with the hemifield inhibition model of Hughes and Zimba (1985; 1987). However, the pattern of significant benefits shown in the present experiment for targets in the attended hemifield was not shown by Hughes and Zimba and is not easily accounted for in terms of their model, which predicts a large cost for targets in the non-attended hemifield and does not enable an explanation of the benefits obtained from a spatial informative cue. The use of marker boxes to indicate target locations was shown to increase RT's for targets in the non-attended hemifield by Zimba and Hughes (1987). This same finding has been shown to occur with saccade latencies in the present experiment. The finding is of interest in that the use of marker boxes gives the subjective experience that it is easier to direct attention to a marked than unmarked location. The actual finding is that there is no benefit from the use of marker boxes

but that it produces an interference effect and this effect is greatest for targets located further into peripheral vision. This finding highlights the importance of using an unstructured visual array in experiments which compare the costs and benefits of valid and invalid trials to avoid exaggerating the magnitude of the benefits obtained by cueing visual attention. However, under normal everyday viewing conditions the visual field will contain a large amount of structure so the use of the simple visual display in these experiments is a very artificial situation. Visual attention could produce areas of inhibition for structures located in the visual field which serves to prevent an eye movement being made to a stimulus located in the non-attended region of space.

The mean saccade latency obtained to targets in the attended hemifield in this experiment, revealed a subtly different pattern of costs to those obtained by Rizzolatti et al. (1987). Rizzolatti et al.'s results showed a similar cost for invalid targets at the near and far eccentricity locations, following a cue to the adjacent location 4^{o} away. In the present experiment mean latency to the near target location was similar following a cue to the near and far location, but latency to the far target location was slower if the near target location was cued. This pattern of results was also shown by Gorea, Findlay and Lévy-Schoen (unpublished) on a similar saccadic eye movement experiment. They suggested that this result can be explained in terms of an area of activation, which stretches from the fovea to the cued location. If this is the case then cueing the far location will produce a broad area of activation from the fovea spreading to the far target location, so the near target location will be equally facilitated following a near or far cue. If the near location is cued then the beam will spread from the fovea to the near location resulting in the extra cost if a saccade is required to the far location. This difference between the covert and overt orienting experiments is not consistent with the premotor model of attentional orienting, which requires a similar pattern of cueing effects for manual RT's and saccade latencies. An examination of the mean saccade latencies indicates a trend of latency being slower to the near target when the far location is cued, so the results are in the correct direction to those obtained by Rizzolatti et al., but the magnitude is reduced. This difference in the results of this experiment and those of Rizzolatti et al. could reflect the differences of the two separate response modes and highlights the problems of directly comparing the results obtained from different motor systems.

Presenting targets on the horizontal axis in the upper visual field (upper-axis condition) produced a reduction in the magnitude of the costs for targets in the non-attended hemifield, without affecting saccade latency to targets in the attended direction. Although the reduction in costs was not significant it was in the direction of the initial prediction (Findlay and Harris, 1984). In the upper axis condition saccade latency for a far eccentricity target in the attended direction, following a cue to the near attended location was the same as that for the near target in the non-attended hemifield. This reduction in the size of the costs produced in the non-attended direction could be explained in terms of the visuomotor map model of saccade generation (McIlwain, 1986; Findlay, 1987). It could be assumed that the motor map contains areas of activation (channels) and the size of these areas (receptive field size) is smaller for spatial

locations closer to the fovea. Locations away from the fovea fall into a larger areas of activation (channels with larger receptive fields) in the motor map. A target crossing from one hemifield to the other along the horizontal axis will project into different areas (channels) of the motor map. A crossing of the vertical meridian above or below the line of fixation, could however result in targets at the near eccentricity location falling within the same area of the map, thus reducing the cost for targets which cross the vertical meridian (see: diagram below). If this was the case then latency would be expected to be comparable for near eccentricity targets in the attended and non-attended hemifields, which is not the case. One reason for the lack of a significant reduction in the meridian effect in this experiment, could be due to the small vertical displacement used and the distance between the two near target locations. The distance between the near targets in opposite hemifields was approximately 10^{o}, which is much greater than used by Findlay and Harris. Furthermore, it would be unlikely that the two targets 10^{o} apart would share the same area (channel) of activation in a motor map. It would be instructive to repeat this experiment using targets presented level with fixation and above fixation, but with targets at closer eccentricities than those used in this experiment. A near target in opposite hemifields could be represented in the same area of motor activation if a smaller eccentricity was used.

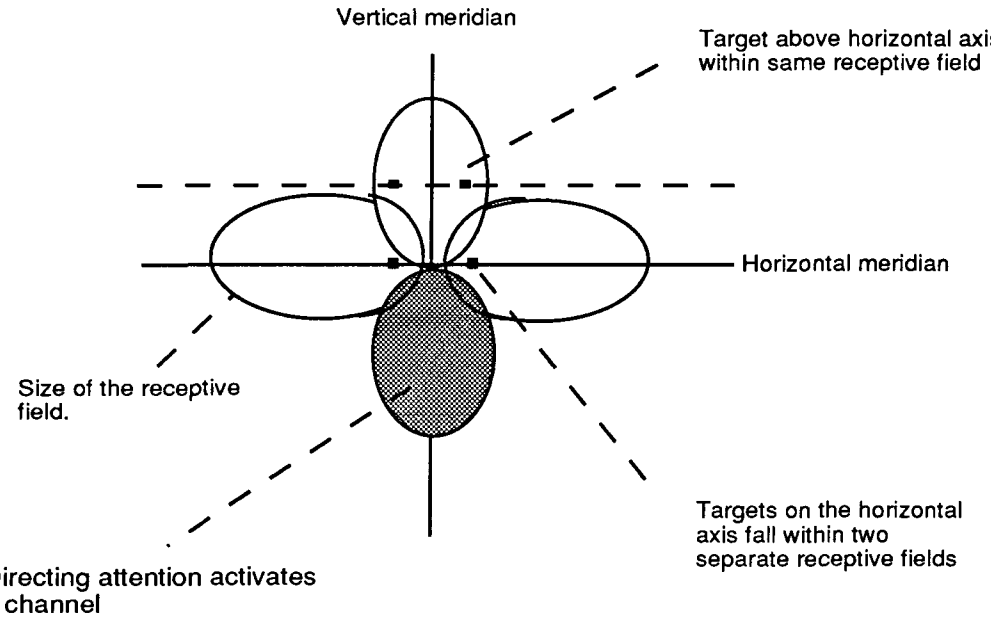


Diagram: to show why a crossing of the vertical meridian in the upper field could have a smaller effect on saccade latency than a crossing located at the fovea (Adapted from McIlwain, 1986).

The basic finding of a large cost for saccades made to a target in the non-attended hemifield can be explained in terms of either the reprogramming of saccade direction (premotor theory), or in terms of inhibition for the non-attended hemifield (Hughes and Zimba, 1987). A further possibility is a combination of both of these theories incorporated into the visuomotor map model of saccade generation (Findlay, 1987; McIlwain, 1986). The physiological evidence suggests that there are two separate maps for generating saccades to the left and right sides of space in the superior colliculi (Sparks, 1986). Directing attention in one side of space could be thought to produce an area of activation in the relevant motor map, and to produce inhibition for

the motor map responsible for movements in the opposite side of space. The exact distribution of the inhibition in the contra-attentional map is not known. The presence of the inhibition on the map of non-attended space would be responsible for the increase in saccade latency for targets that are presented in the non-attended hemifield. This idea is developed further in the proposed model described in Chapter eight.

The idea of a collicular map is in contrast to the models of saccade generation that involve separate programming of saccade amplitude and direction. The results obtained in the horizontal-box and upper-axis conditions do not reveal any evidence of separate amplitude programming. However, there was some evidence to suggest that amplitudes could be programmed separately in the results obtained in the horizontal axis condition. In this case it can be seen that saccades made to targets at the near eccentricity location in the non-attended hemifield, were faster following a cue to the near location in the in the attended hemifield. Saccades made to a far target location in the non-attended hemifield were faster following a cue to the far location in the attended hemifield. This result is surprising as the cue was indicating a location in the opposite direction to that in which the final saccade was made. In terms of models of saccade generation, this finding implies that the amplitude and direction of the saccade are preprogrammed following the cue and that the cancellation of the direction programme for an invalid hemifield target does not result in the cancellation of the amplitude programme. However, given that this pattern of costs was not shown in the horizontal-box, or upper axis condition it is difficult to explain it in terms of saccade generation only. Presumably the same advantage for cueing a saccade amplitude would be expected to occur under all three experimental conditions, so there must be some other explanation. In terms of the known mechanisms of saccade generation it seems unlikely that a saccade direction programme could be cancelled while an amplitude programme remains.

The use of the central cueing procedure in this experiment produced a similar pattern of mean saccade latencies as have been obtained by Rizzolatti et al., (1987) and Umiltá et al., (1991) with manual RT's. The presence of the meridian crossing effect obtained in this experiment is at odds with the finding of Crawford and Müller (1992) who failed to find the extra cost following peripheral cueing of saccades. The reduction in the meridian effect when targets are presented in the upper field is in line with the prediction of Findlay and Harris (1984) and although the result was smaller than expected this could reflect the eccentricities of the targets used. There are two main differences between the present results and those of Rizzolatti et al. and Umiltá et al. Saccades made in the attended direction showed equal facilitation for near targets following both a near and far cue which indicates that a broad area of space is facilitated following a far cue. The results of Rizzolatti et al.'s manual RT experiments obtained benefits which were restricted to the exact cued location. The second anomalous finding is the selective cueing effect shown for targets in the non-attended direction in the horizontal axis condition. This finding suggests that saccade amplitude and direction can be programmed separately and cancelling the direction programme can leave the amplitude programme intact. The generality of the separate programming explanation is abstracted by the failure to find it in the two other conditions.

4.4 Conclusions.

The results provide further support for the premotor model of visual attention. Directing attention to one hemifield produces a large slowing in saccades made in the non-attended hemifield. A similar effect has also been shown in manual RT experiments in which attention was oriented covertly. This suggests that directing visual attention serves to produce a broad area of inhibition for the non-attended hemifield. The large costs for crossing the vertical meridian could be explained by attention producing a broad area of inhibition for the non-attended hemifields/quadrants as suggested by Hughes and Zimba (1985; 1987). This could be included into models of saccade generation by incorporating the idea that the selection of one direction in which to make an eye movement is the equivalent to inhibiting the component responsible for producing a movement in the opposite direction. A change of the direction in which to make a saccade from that which has been planned in advance, will require a reduction of the inhibition acting on one hemifield and activation of inhibition for the other. This could account for the extra time incurred on both saccade latency and manual RT's.

The present experiment showed smaller benefits were obtained for targets in the attended hemifield. The pattern of these results suggests that there could be a broad area of activation in the attended hemifield, which spreads from the fovea, to the cued location, and produces a small speeding of saccade latency. This area of activation could produce the facilitation effect by preprogramming the motor movement required in terms of activating a broad area of a motor map which controls the initiating of the saccade.

The presence of marker boxes was shown to increase saccade latency as had been shown to occur with manual RT's by Zimba and Hughes (1987). This provides some support for their view that marker boxes produce an extra inhibitory effect which may alter the subsequent cost-benefit analysis and exaggerate the apparent benefits of directing attention.

The model of saccade generation thought to provide the best account of the results of the present experiment is similar to the retinotopic map of motor activation suggested by McIlwain (1986) and Findlay (1987) with the extra assumption that crossed inhibition is produced which acts on the opposite map to that which has been selected to make the saccade. The meridian crossing effect shown when a saccade was made to a target in the non-attended hemifield was reduced when the target location was located above the fixation location. This is consistent with the finding of Findlay and Harris (1984) and suggests that the field size of channels of motor activation within the motor map could increase with distance from the fovea. The one result that does not support the visuomotor models of saccade generation is the evidence of amplitude cueing evident in the horizontal axis condition, as has been suggested by Abrams and Jonides (1988). This finding argues that saccade amplitude and direction can be programmed separately which is not possible in models that use a direct translation of target position into a motor output. It is not clear however why this result was not found in the two other conditions so it should be regarded with some degree of caution. These ideas are devoloped further in the proposed model described in Chapter eight.

Chapter 5

Unilateral Spatial Neglect.

5.1 Introduction.

Unilateral spatial neglect is defined as the failure to respond to stimuli located in the contralesional side of space (Heilman, Watson and Valenstein, 1979) and is shown in man following brain damage usually to the right hemisphere (Brain, 1941). Neglect is most severe in the first few weeks/months following the initial lesion and may then show a certain amount of recovery. The patients that do show evidence of recovery tend to neglect a contralateral stimulus only when it is simultaneously paired with a stimulus presented ipsilaterally. This is termed 'visual extinction'. Neglect and visual extinction may both be thought of as reflecting an impairment of visual attention (Karnath, 1988; Young and DeHaan, 1989).

The popular theories of neglect are that it reflects an impairment to an internal mental representation of space (eg. Bisiach and Luzzatti, 1978; DeRenzi, 1982), or an impairment of visual attention (Heilman et al., 1985; Karnath et al., 1991; Kinsbourne, 1977; Mesulam, 1981; Posner et al., 1984). One problem with the impaired representational hypothesis is that it fails to account for studies which have shown that contralateral stimuli are available for some information processing outside of conscious awareness (Volpe et al., 1979; Marshall and Halligan, 1988). If neglect resulted from a failure to construct a representation of contralateral stimuli then it would presumably not be available for any form of covert processing. A second and perhaps more convincing piece of evidence against the representational view is that cueing the patient to the contralateral side of space reduces neglect (Riddoch and Humphreys, 1983; Posner et al., 1984; Posner and Cohen, 1987). If the neglect patient lacked a representation of contralateral space then presumably cueing would not ameliorate the detection of contralateral stimuli. Riddoch and Humphreys (1987) suggested as an alternative that neglect patients may be impaired at scanning one half of an internal spatial representation which accounts for the improvement shown following cueing.

The following chapters are concerned with the attentional explanations of neglect and describe experiments involving an examination of the attentional deficits shown by a single patient (B.Q.) with unilateral spatial neglect. B.Q.'s ability to orient to contralateral and ipsilateral stimuli has been examined using an overt orienting experiment under conditions which manipulate visual attention by controlling the timing sequence of stimuli onset and offset. These experiments have used single and bilateral simultaneous target presentation (similar to the experiments described in Chapters two, three and four) to examine the degree of neglect and visual extinction. Experiments are also described which examine the role of an attentional deficit in producing patterns of left sided word omissions when reading text. For this reason the accounts of neglect discussed in the rest of this introduction will largely be concerned with the attentional explanation of neglect and will also outline the experimental evidence supporting this view. The frames of reference which may be involved in the distribution of attention in

neglect are described in some detail as they have implications for the understanding of some of the results obtained. An outline of the nature of the brain damage associated with neglect is described initially.

5.2 Nature of the brain damage associated with neglect.

Neglect in man typically, although not exclusively, occurs following unilateral damage to the right hemisphere (Brain, 1941) in the region of the *parietal lobe* . DeRenzi (1982) reviews the evidence that neglect results more commonly following damage to the right hemisphere, than to the left hemisphere, showing that this asymmetry does not simply reflect a sampling bias of the patients examined for neglect following a stroke. Such a sampling bias could result for example from excluding left brain damaged patients who show aphasia, who would be more difficult to assess for neglect than right brain damaged patients. DeRenzi reviewed studies which have examined large numbers of left and right hemisphere damaged patients which have adequately controlled for a sampling bias, which clearly show that neglect is more common following right brain damage than left brain damage (eg. Chedru, 1976; DeRenzi, Faglioni and Scotti, 1970). It is still possible that incidence of neglect following left brain damage remain unnoticed as the patient are not tested early enough after the initial lesion, or because the measures of neglect are not sensitive enough to indicate its presence before a degree of recovery of function has occurred. This view was supported by DeRenzi, Faglioni and Scotti (1970) who showed that left and right hemisphere groups had increased search times for contralateral stimuli in a tactile maze. The overall conclusion is that neglect is more frequently associated with right hemisphere lesions and this results in neglect of greater severity than after left brain damage. The right hemisphere appears therefore to have a more important role in directing attention in man than does the left. Damage to the left hemisphere can also produce neglect but this can be compensated by for by the intact functioning right hemisphere. The left hemisphere does not appear to be able to completely take over the functions of the damaged right hemisphere.

Although neglect is most commonly associated with damage to the parietal lobe it is not the only brain area which is implicated. Studies which have examined specific lesion sites of large numbers of neglect patients have shown the presence of lesions encroaching upon the parito-occipital and parieto-temporal-occipital junction (Bisiach, Luzzatti and Perani, 1979; Hécaen and Angelergues, 1963). The parietal lobe receives inputs from, and is connected to, a wide range of cortical and subcortical areas (see: Mountcastle, 1978). This is compatible with the view that its role is to integrate multisensory information and control the motor mechanisms involved in making a saccade to stimuli in the visual periphery which summon attention (Yin and Mountcastle, 1977), although there are other areas which can also serve this function (Schiller, True and Conway, 1980).. Damasio et al. (1980) examined five cases of neglect resulting from non parietal lesions. Three of the patients had lesions in the frontal lobe cortex which affected the supplementary motor area (SMA) and anterior cingulate complex, the two other cases had lesions in the basal ganglia (striatum) which is connected to the other two areas. All of these areas are interconnected with the parietal lobe. DeRenzi (1982) suggests that these structures

could be a part of a hierarchical system, with the parietal lobe controlling visual orienting, while it is commanded in turn by a higher level executor involving the SMA/anterior cingulate complex. DeRenzi (1982) provides a review of cases of neglect resulting from lesions to the frontal lobe (eg. Heilman and Valenstein, 1972; Jenker and Kutschera, 1965) and notes that in these cases neglect is less severe, and recovery much quicker, than following parietal damage. The role of the frontal lobe in sustaining attention may be easily compensated for by other structures.

The role of the right parietal lobe in man seems to be greater than in Primates indicated by the failure to produce the full pattern of left neglect in animal lesion studies (Schwartz and Eidelberg, 1968; Lynch and McLaren, 1989). Lynch and McLaren (1989) showed that unilateral lesions to the parietal lobes of monkeys increases saccade latencies and produces an extinction effect for contralateral targets in bilateral presentation, but not neglect of contralateral targets presented singly. A further lesion in the opposite hemisphere was shown to reverse the direction of the extinction effect. Parietal lesions produced neglect in one monkey for single contralateral targets, but only when coupled with a lesion to the frontal eye fields. The monkeys ability to saccade to single targets provides further support that extinction does not result from a visual field defect, or an impairment to the oculomotor system. The results are taken to support the idea that parietal lesions produce a deficit of the capacity to attend to stimuli located contralateral to the lesion and that the alterations are milder in monkeys than in man.

A further method of studying the functions of the parietal lobe has been to measure the firing patterns of single cells. Lynch et al. (1977) classified three types of cells within the parietal lobe of alert monkeys. *Visual fixation neurons*, fired when an object of interest was within arms reach, but were mostly suppressed before or during a saccade towards a new target. *Visual tracking neurons*, which were active before and during smooth pursuit movements of a target and were suppressed before and during a saccade. *Saccade neurons*, discharged before and during a visually evoked saccade providing the stimulus had some motivational value. Lynch et al. viewed these neurons as being involved in the process of combining visual information about objects and signalling commands to direct and maintain gaze to these objects. Robinson, Goldberg and Stanton (1978) questioned the view that parietal cells are command cells for motor commands. They showed that cells which fired in association with fixation, smooth pursuit and saccades, also fired on the presentation of a sensory stimuli which were not followed by an eye movement. They conclude that the parietal lobe neurons serve to signal the presence of a stimulus and do not necessarily signal a command to move towards it. The cells of the parietal lobe are unspecific and respond to a wide range of stimulus properties and the firing rate can be enhanced in monkeys when the stimulus has a motivational significance. Once an event is detected its presence can be signalled to the motor areas which move the eyes to novel stimuli. As such the parietal lobe can be thought of as being an attentional mechanism which orients attention to the contralateral field and can command oculomotor responses.

Recording experiments have also served to establish how the parietal cortex represents visual space. Anderson (1989) suggests that the posterior parietal lobe could contain non-retinotopic representations of visual space. Anderson et al. (1985) showed that

the retinal receptive fields of parietal lobe neurons in monkeys moved with the eyes (retinotopic coordinates), but the responsiveness of the visual fields varied as the eyes moved. This interaction produces cells with receptive fields that are dependent on the position of the eye and of the target in head centred space. The suggestion is that the parietal lobe is the area involved in the sensorimotor integration. The incoming sensory signal is transformed to a spatial and motor coordinate frame for the guidance of motor responses.

The parietal lobe appears to be the crucial structure involved in cases of neglect. The frontal lobe is also implicated, but its role does not seem to be as important as that of the parietal lobe and its functions appear to be more readily compensated for once damaged. The parietal lobes functions are related to orient attention in the contralateral field and appears to be involved in directing the oculomotor mechanisms to stimuli of interest.

5.3 Accounts of neglect.

One account of neglect is that it results from a deficit of the internal representation of space. The 'representational' hypothesis suggests that neglect is not restricted to a deficit of perception, but can be observed for the contralesional side of the patients mental representation of space (Bisiach and Luzzatti, 1978; Bisiach, Luzzatti and Perani, 1979). The representational hypothesis shows that neglect affects many different cognitive processes, including mental imagery. However, the theories of neglect which are most directly related to the experiments described in the following chapters are the 'attentional' theories of neglect. There are several theories of neglect which suggest that it results from an impairment of attention. Early attentional theories of neglect have suggested that it is produced by an imbalance of activity between the two hemispheres of the brain (Kinsbourne, 1978) or that it is due to a deficit of arousal resulting from damage to the cortico-limbic-reticular loop (eg. Heilman, Watson and Valenstein, 1979). More recent attentional theories have suggested that neglect results from a deficit in specific processes involved in the covert orienting visual attention (Posner et al., 1984; Posner and Cohen, 1987). A large amount of experimental work has been carried out which supports the impaired covert attentional orienting hypothesis of neglect. This section aims to provide a brief outline of the early attentional accounts of neglect with a more detailed section reviewing the evidence for the covert attentional orienting hypothesis.

5.3.1 Hemispheric attentional activity hypothesis.

Kinsbourne (1978) suggested that neglect arises due to a loss of inter-hemispheric activity, which results in a bias of movements of the head and eyes in the ipsilesional direction (for a recent statement, see: Kinsbourne, 1987). The theory is based on the idea that activation of one hemisphere of the brain is assumed to inhibit activity in the opposite hemisphere. In normal operation there is thought to be an innate bias for attending more to the right than to the left. The left hemisphere of the brain is thought to produce a strong tendency to move to the right, while the right hemisphere has a weaker left orienting response. Damage to the right

hemisphere reduces the inhibition acting on the left hemisphere, leaving it over-activated and thus producing a strong orienting tendency to the right. Damage to the left hemisphere however, does not produce severe right neglect, because the turning tendencies of the two hemispheres are not equal, with the right hemisphere orienting response being weaker.

Kinsbourne's hypothesis is useful in explaining why neglect patients can be impaired at responding to stimuli located to the left of other stimuli, in the right side of space. For example, Ladavas et al. (1990) showed that reaction times to left stimuli were slower than to right stimuli, even when both were located entirely within the neglect patients intact right visual field. Neglect patients appear to focus attention at the right relative position and also showed evidence of increased processing efficiency for targets at that location, suggesting that there is a small attentional focus at the right position.

One problem with Kinsbourne's view is that patients with damage to the right hemisphere would be expected to perform better (or at least normally), with stimuli located in the right side of space, due to the reduction of inhibition from the left hemisphere. Heilman and Watson (1977) provide evidence against this idea by showed that patients with left neglect following RBD (right brain damaged) made more errors with stimuli in the right ipsilesional side of space, than do LBD (left brain damaged) patients with left sided stimuli.

5.3.2 Attentional akinesia hypothesis.

Heilman (eg. Heilman and Valenstein, 1972) suggested that sensory and perceptual explanations alone are inadequate in accounting for neglect and put forward what they termed an 'attention-arousal' hypothesis. The theory is useful in that it can account for neglect following damage to many different parts of the brain. This occurs due to damage to pathways (cortico-limbic reticular formation loop) involved in mediating attention and arousal (see: Heilman, Watson and Valenstein, 1979). Neglect is thought to be due to a unilateral decrease of arousal, which produces the selective loss of the orienting response towards contralateral hemispace; which is termed 'directional akinesia'. According to his view the left hemisphere can only control orienting to the right side, while the right hemisphere controls orienting to both the left and right sides. Damage to the right hemisphere leaves the left hemisphere capable of producing motor responses into the right side of space and neglect for the left side of space then occurs. This accounts for the prevalence of neglect for the left side of space as a patient with left brain damage is able to control orienting responses in both directions with the intact right hemisphere. Heilman et al. (1985) showed that patients with right brain damage were slower at initiating a motor response using the ipsilesional limb, than were left brain damage patients. This suggests that the right hemisphere has a dominant role in the preparation of a motor response. Furthermore, the right hemisphere lesion group showed slower reaction times from a left starting point, than from a right starting point, on the motor task. Normals subjects and left lesion group showed no such directional slowing. This delay in initiating a movement in the contralesional direction (shown by the right lesioned group), strongly supports the directional

hypokinesia hypothesis of neglect.

The directional hypokinesia hypothesis gains support in a study of a group of neglect patients completing a crossing out task under normal, and mirror viewing conditions (Tegnér and Levander, 1991). In the mirror viewing condition the direction of the motor response is the opposite direction to that of attention. Four of the patients cancelled lines in right hemispace in the normal and mirror viewing condition, which suggests that they can move attention leftwards, but cannot make a motor response in that direction. The results of these four patients is consistent with the directional hypokinesia hypothesis. Ten patients crossed out lines in the right under normal viewing, but crossed out lines to the left in mirror viewing. This suggests that they directed attention to the right side under both viewing conditions, but could make a motor response to the left or right. The second group of patients results conform to an impaired attentional explanation of neglect. The possibility that the second group have an impairment in moving their eyes to the left cannot be ruled out in this experiment, so they could be demonstrating hypokinesia of the oculomotor system. These results illustrate the fractionation of the neglect syndrome, in that it could reflect an impairment to make a motor response in the contralateral direction, or to move attention contralaterally.

An implication of the akinesia hypothesis, is that the degree of neglect should not be reduced by cueing the patient to the left side of space when there is a competing stimulus in the right side of space, as the left hemisphere will bias attention towards the right sided stimuli. Heilman and Valenstein (1972) showed that cueing did not improve line bisection performance but other workers have shown that neglect is reduced by cueing. Riddoch and Humphreys (1983) showed that cueing reduced neglect in a line bisection task, similar to Heilman and Valenstein's (1972). This suggests that neglect patients should not be thought of as being completely 'akinetic'.

5.3.3 Attentional orienting hypothesis.

A third attentional view of neglect put forward by Posner et al. (1982, 1984, 1987), is that neglect patients are impaired at covertly orienting attention. Posner et al. (1984) suggested that there are three processes involved in each shift of visual attention: initially attention must be *disengaged from a target,* then it is *moved or oriented* to a new target, and finally it is *engaged* at the new location. In Posner et al.'s (1984) experiment, patients were instructed to covertly orient their attention to either a left, or right side target location, following a central arrow cue, or peripheral box brightening. Reaction times were shown to be facilitated for left and right valid targets with increasing SOA's, indicating that the cues were effective at orienting attention in either direction. Right sided targets invalidly cued (by a left cue) produced a slight slowing on RT's, but left targets invalidly cued (by a right cue) produced a much greater slowing with very long RT's being produced. Posner et al. compare the very long RT's produced to left targets following an ipsilateral side cue, to 'visual extinction'. The extinction effect was shown by LBD patients but to a lesser extent than the RBD group. Posner et al.

explained the 'extinction' effect in their RT experiment in terms of their three component model of visual attention. Patients can covertly orient attention to the left and right direction, following a valid cue, indicating that they are not impaired at covertly moving and engaging attention. The extinction effect for contralesional stimuli shows that parietal damage produces a selective impairment to the disengagement procedure, whereby patients cannot disengage attention from an ipsilesional location, if a contralesional movement is required. The poorer performance of the RBD group suggest that the each hemisphere controls shifts of attention in the contralateral side of space, but the left hemisphere only controls shifts to the right while the right hemisphere can control shifts in both the left and right direction. Posner et al.'s impaired covert orienting hypothesis is also useful in explaining how cueing can reduce contralateral neglect by facilitating the disengagement process.

5.4 Further studies of the attentional orienting hypothesis.

There have been many recent studies of neglect based on the impaired attentional orienting hypothesis involving similar experimental paradigms to those used by Posner et al. (1984). The following section aims to outline some of these and to describe the main findings from them.

Morrow and Ratcliff (1988) replicated Posner et al.'s (1984) experiment using a larger group of left and right brain damaged patients, and tested them sooner after the initial lesion, so that the severity of neglect was greater than in Posner et al.'s patients. The results confirmed those of Posner et al., with long RT's shown by the RBD group for contralesional stimuli following an ipsilesional cue, suggesting an impairment of disengagement. The LBD group did not show the extinction like effect. This suggests that the right hemisphere lesion causes a greater deficit than the left hemisphere lesion. The results unlike those of Posner et al.'s also suggested that RBD impairs the patients ability to move attention contralesionally, suggesting that the movement component can also be impaired. This was indicated by the RBD patients showing slower RT's for validly cued contralesional targets, than validly cued ipsilesional targets. It is possible that this deficit in the movement component shows faster recovery than does the disengagement deficit, which is why it was not found by Posner et al. This deficit of contralesional movement has also been suggested by Karnath (1988) in his model of neglect.

Petersen et al. (1989) used a similar covert orienting, cued manual RT experiment to Posner et al.'s (1984), with parietal, frontal lobe and temporal lobe patients. Performance was compared to that of normal subjects. In addition to the use of a valid peripheral cue, a procedure of brightening the whole screen (diffuse cue) was also used. The diffuse cue was initially thought to be equivalent to a neutral cue condition, but provided some interesting results with the parietal patients. The parietal patients results with peripheral cues confirmed the patterns found by Posner et al. (1984). Targets in both field were responded to equally well following a valid cue, but responses to contralesional targets were very slow following an ipsilesional cue. In the opposite situation responses to ipsilateral targets were not slowed following a

contralateral cue. The left parietal patients produced a similar pattern of results to those of the right parietal group with a cost for contralesional orienting after an ipsilesional cue, but the deficit was less strong. Neither the frontal, or temporal patients, showed the extinction effect on invalid trials. Frontal lobe damage produced a generalised slowing of RT's for all cue conditions which is interpreted as showing that the frontal lobe has a role in the preparation of an attentional movement.

The diffuse cue produced a dramatic slowing in RT's made to targets located in both the ipsilesional and contralesional visual fields, the size of which was greater than that shown in the extinction effect. The explanation put forward by Petersen to explain the effect the diffuse cue has on parietal patients, is that the screen brightening could freeze attention possibly at the central fixation location. This could explain the increase in RT's shown to a lesser extent in normal subjects following diffuse cues. Alternatively with parietal patients the diffuse cue could drive attention to the extreme ipsilesional location. If attention is moved to the extreme ipsilateral side of the screen, then targets appearing in the ipsilateral field will also require a contralesional movement of attention, from the far ipsilesional location. This explanation is consistent with Posner et al.'s (1987) finding that parietal patients are impaired at moving attention in the contralesional direction irrespective of which visual field the movement of attention is made in. The long RT's produced following a diffuse cue, to targets in both the ipsilateral and contralateral visual fields, can be explained in the same framework that Posner et al. (1984, 1987) used to explain the 'extinction' effect, in that neglect patients cannot disengage attention for a contralesional movement. Models of neglect should therefore account for a bias of orienting attention in the right direction, following right parietal damage.

The finding of Posner et al.'s (1984) that parietal patients are impaired at moving attention contralaterally, following an ipsilateral visual cue, could reflect an impairment of visual attention, or of a system which controls attention in various modalities such as visual, auditory and tactile attention. Farah et al. (1989) examined the nature of the disengagement deficit to show if it was a visual phenomena, or if it reflected a deficit of a 'supra-modal' attentional system. Eight right hemisphere patients with signs of left neglect, were tested on a direct replication of Posner et al.'s (1984) covert orienting manual RT experiment, and in a variation in which attention was cued by an auditory cueing procedure. The results showed that the patients were impaired at disengaging attention in order to attend to a contralateral visual stimulus, following both a visual and auditory ipsilateral cue. This suggests that the parietal lobe attentional system is not modality specific, but that it contains a representation of space from more than one modality in a supramodal representation of space.

A study of patients with either extinction or neglect by Karnath (1988) used bilateral and unilateral stimulus presentation to examine the attentional nature of both deficits. In this experiment the patients could report left and right stimuli when presented singly, but neglected the left stimuli on bilateral presentation. Two patients showed reduced performance on contralateral stimuli when tested with unilateral presentation with short exposure times in the acute stages of neglect. This confirms Posner et al's. finding of a reduction of performance for

contralesional stimuli. Patients could report the extinguished (LVF) stimuli if instructed to attend to the left and to ignore the right stimuli. Under forced choice naming of bilaterally presented stimuli the patients typically reported the right stimulus before the left. Asking the patients to report the LVF stimulus first during bilateral presentation, lead to an improvement of performance and a reduction of extinction for contralesional stimuli, although patients were still poorer than controls. This suggests that there is a general reduction in the patients information processing capacity. Karnath suggested that three processes are impaired in these patients which in its most severe form produces neglect. Patients cannot: orient attention contralesionally (component A), show a bias of orienting in the ipsilesional direction (component B), and show a general reduction in information processing ability (component C). During the acute stages the patients cannot orient attention in the contralesional direction, while in the less severe stages of extinction this deficit has shown some recovery, but the bias of ipsilesional orienting remains producing visual extinction on bilateral presentation. Karnath's three process model explains the problems RBD patients have responding to contralesional stimuli and also emphasises the close link between neglect and visual extinction.

5.5 Between and within hemifield attentional orienting

The experiments described in the preceding section, have shown that neglect patients are impaired at orienting attention to the contralateral visual field and that they appear to bias attention into the ipsilateral visual field. Baynes et al. (1986) examined the ability of left and right hemisphere damaged patients at orienting attention within the left and right visual fields. Two target locations vertically positioned in either the left or right visual field were used in a cued manual RT task. Right parietal damage was found to produce a generalised slowing of responses to visual stimuli and also produced a selective deficit of orienting attention within the left visual field. Left hemisphere patients also showed a generalised slowing compared to controls, but showed no difference in responses made in either hemifield. The right parietal patients appear to be impaired at covertly orienting attention vertically, in the contralateral field regardless of whether the shift is within, or across, visual fields.

Posner et al. (1987) examined right and left parietal patients ability to orient attention on the horizontal axis in the contralateral and ipsilateral visual fields. This experiment involved a variation of the basic covert orienting manual RT experiment described above. The stimuli were arranged so that when a target was invalidly cued within a hemifield this required an attentional movement in either the ipsilateral or contralateral direction. The first finding was an advantage for ipsilesional visual field events and the second finding was an advantage for movements of attention in the ipsilesional direction. The advantage for movements of attention in the ipsilesional direction was apparent in both visual fields. The disengagement deficit apparent following right parietal damage selectively impairs the patients ability to orient attention in the contralesional direction.

5.6 Studies of overt orienting in neglect patients.

The experiments described so far have involved covertly orienting attention while the patients eyes remain fixed. Attempts have also been made to examine the patterns of overt eye movements produced by neglect patients. Girotti et al. (1983) examined the eye movements made by patients with left and right parietal lesions to predictive targets presented in both visual fields. The right hemisphere group contained patients without signs of neglect (3 with left hemianopia) and seven patients with signs of neglect (6 with left hemianopia, 1 non hemianopic). The right parietal patients with neglect failed to make an eye movement on a quarter of all trials and also showed an increase of saccade latency for targets on the left side. Neglect patients tended to perform large numbers of small amplitude saccades in the contralateral hemifield and had a large time interval between making these saccades. This pattern of multiple search saccades does not appear to be dependent on a visual field defect, as the single neglect patient without a hemianopia showed a similar pattern. Furthermore a hemianopic patient without signs of neglect, showed a normal pattern of eye movements. The hemianopic patients without neglect appeared to be able to make use of the predictive position of the target to compensate for the hemianopia, while the neglect patients failed to use this information. Neglect patient's showed an increase in saccade latency, with an increase in the tendency to perform multiple saccades of small amplitude. The patients often failed to initiate any saccade at all. These results suggest that they have a deficit of overt orienting which is not a result of a visual field defect.

The compensatory patterns of eye movements used by hemianopic patients, which are not shown by neglect patients have been indicated by Ishial et al. (1987). In this study hemianopic patients with and without neglect viewed simple line drawings with free viewing while their eye movements were recorded. The patients with hemianopias without neglect viewed the hemianopic side of a pattern for longer than the other side and showed patterns of search movements (step saccades) in the hemianopic side. This is thought to reflect a strategy used to compensate for a visual field defect (Meienberg et al., 1980). In contrast patients with left hemianopias and neglect failed to show these compensatory patterns, they did not spend longer looking at the left side and did not show evidence of search patterns of saccades. It appears that patients with a hemianopia can use compensatory strategies to overcome the sensory deficit, but patients with neglect fail to use these strategies and spend very little time scanning the hemianopic area of the pattern. This according to Ishail leads to the patients failing to notice the left sides of the patterns and renders the neglect patients hypokinetic as they fail to make eye movements into the hemianopic region.

A recent study by Karnath et al. (1991) has shown that neglect patients cannot be regarded as being completely hypokinetic, as they do make contralesional eye movements under certain situations. Karnath et al. replicated the basic finding of Girotti et al. (1983) and showed that right parietal patients without hemianopias made saccades of slower latencies to targets presented in the LVF than targets presented in the RVF. However, the patients

produced saccades of normal latency when their body trunk was tuned to the left, so the LVF stimuli now fell to the right side of trunk space. Karnath suggests that the impaired ability to voluntarily orient attention operates with respect of the contralesional side of the body midline. The issue of the coordinates involved in neglect is discussed further in the next section.

Rizzo and Hurtig (1992) examined the saccadic eye movements of five neglect patients to random and predictable saccade targets presented in the left and right visual fields. They also measured eye movements to smooth pursuit targets and while the patients were scanning a photograph of a face and a line drawing of a scene. In the saccade target condition the appearance of one target was simultaneous with the offset of the previous target. This was designed to ensure that attention was 'disengaged' from the current target location (Mayfrank et al. 1986). With contralateral saccade targets the patients failed to make saccades of normal amplitude and latency and also failed to make a return eye movement from positions in the ipsilateral field. In contrast neglect patients could track a smoothly moving target in both the left and right direction. The failure of the patients to make a left saccade in the condition in which the previous target went off simultaneously in a 'zero gap' condition is taken by Rizzo as disproving Posner et al.'s (1984, 1987) 'disengagement' hypothesis of neglect. The simultaneous offset should disengage attention allowing a contralesional movement to be made. The ability of the patients to track a smoothly moving target was taken as evidence that neglect patients can use top-down information to search for stimuli in the neglected side of space. The non predictive saccade targets provide bottom up information only which may not be sufficient to overcome the left neglect. A possible problem with Rizzo's conclusion is whether a zero gap leaves sufficient time for the attentional system to be completely disengaged. It is possible that the system is not disengaged instantly once a target is removed, this would result in the left stimulus failing to orient attention as it is not fully disengaged. The patients' eye movements were also recorded while they scanned a line drawing (Cookie theft picture from Boston Diagnostic Aphasia Exam) and the face of president Nixon. The patients were found to make fewer left saccades than right saccades on both stimuli. However, they showed three times more searches into the neglected side with the face, than with the scene. This reduction in neglect for a face than for a scene, is taken as showing an interaction between top-down and bottom-up factors. A face forms a single object while a scene represents many objects located in space. As neglect patients can recognise faces they can presumably group together the features to recognise it as being a face, this knowledge could be used to initiate a scan into the neglected side. The location of the objects in the scene is available from bottom-up information only that may not be sufficient to enable the patient to scan the contralesional side of space.

The use of top-down knowledge to produce a contralesional scanning strategy has also been shown by a neglect patient while reading lines of text. Huber et al (1988) found that the return leftward saccades made to locate the start of a line fell short at a position close to the middle of the next line. This was followed by backward reading until a plausible continuation of the previous line was found even if this was still not the start of the next line. This impairment at locating line starts was not due to an impaired oculomotor system as the patients could make

saccades to single targets at the eccentricities required to locate line starts on request.

5.7 Frames of reference involved in neglect.

An issue relevant to the attentional account of neglect involves the coordinates involved in determining the neglected from the non neglected sides of space. It is possible that combinations of more than one frame of reference could be involved in the impaired distribution of attention. The issue of frames of reference is a complicated one and has received some experimental examination with regard to spatial neglect. The results are often inconclusive and complicated by the problems of experimentally dissociating all of the possible frames that may be involved. The evidence available is reviewed and discussed in this section.

Neglect could impair the allocation of attention within either a viewer centred, environmental centred, or object centred, frame of reference. A viewer centred (egocentric) frame separates the left and right sides of space with respect to the position of the person. The viewer centred representation could be specified in terms of left and right visual fields, left and right side of the viewers head midline, or left and right of the body trunk midline The environmental (allocentric) frame is viewer independent, left and right being specified with respect to the environmental midline regardless of where the person is situated. In an object centred frame of reference the left and right sides of a particular object are specified by the intrinsic midline of the object regardless of the position of the viewer or of the object in the environment.

The first possibility is that neglect operates in terms of a viewer centred reference frame. In this instance left and right could be defined in terms of the two visual half fields separated by the vertical meridian, or they could be defined with respect to the midline of the patients head, or with respect of the midline of the body trunk. Many experimental examinations of neglect confound all three of these possible coordinate systems as the patient are typically sitting upright, with their eyes fixed on a central fixation point. In this instance it is not possible to decide if neglect operates in terms of the contralesional visual half field, or in terms of the contralesional side of either the patients head, or body trunk. There is some evidence to suggest that neglect does not operate in terms of the two visual hemifields. Bisiach and Luzzatti (1978) demonstrated that neglect patients failed to report the buildings located to the left side of a mental image, when asked to imagine they were in the Piazza del Duomo in Milan. This suggests that neglect can impair higher level representations that are not based on retinal coordinates.

Evidence that neglect does not only affect the deployment of attention to the contralesional hemifield has been provided by showing that the deficit in attentional orienting is also present in the ipsilesional hemifield. Posner et al. (1987) performed a cued manual RT experiment with parietal damaged patients in which the cue and target stimuli were presented within the same hemifield. The results showed that neglect patients were impaired at making

movements of attention in the contralesional direction following an invalid cue, with stimuli presented in both the contralesional and ipsilesional visual fields. Ladavas, Petronio and Umiltá (1990) also examined the deployment of attention to stimuli located in the ipsilateral field in a similar experiment to that performed by Posner et al., (1987). In this case simple (uncued) reaction times were obtained to targets positioned at left and right relative positions within the right visual field of neglect patients. Neglect patients were faster at responding to stimuli presented at the right most position, even though the right stimulus position was further from fixation than the left stimulus position. Neglect does not appear to simply affect shifts of attention into the contralateral visual field but also affects the deployment of attention to stimuli at a left relative position regardless of the visual field. Furthermore, neglect patients responded faster to the target at the right relative position than did control subjects. This result suggests that the neglect patients attention is directed to the right position, while the control subjects attention is distributed more evenly over the entire field which has to be focused at the right target location.

Ladavas (1987) performed an experimental examination of patients with right parietal lobe damage (who showed visual extinction, but not neglect) specifically aimed to investigate if the viewer centred representation involved in neglect is in retinal or head centred coordinates. Patients performed a simple manual reaction time experiment with left and right target positions presented on the horizontal axis, above a fixation point. To dissociate the retinal from gravitational coordinates the patients tilted their heads to the left and then to the right, so that the left and right target stimuli fell entirely within one visual field. The results showed that RT's were slower for the target which were presented in the left position in both the left and right visual fields. RT's were also found to be slower overall when the stimuli were presented in the left visual field (right head tilt) than when presented in the right visual field (left head tilt). Ladavas concludes that the impaired shift of attention involves both the retinal and gravitational coordinates. The impairment results in slower shifts of attention to stimuli located in the left visual field and to stimuli which occupy the left most position in either visual field. The use of the head tilting procedure fails to address the possibility that the viewer centred representation involved could be centred on the patients body trunk midline. If the representation of space is represented in retinal and a body centred framework then tilting the head does not remove the possibility that the impairment of shifting attention is for stimuli presented to the left side of the patients body space.

The possibility that the viewer centred frame of reference involves the body trunk midline has been illustrated by Karnath et al. (1991). Neglect patients saccadic reaction times were obtained to targets presented in the left and right visual fields. This was performed when the patients body trunk, head and visual field midline all coinciding and also when either the head or body was rotated to the left or right while keeping the retinal projection constant. When the head, body and visual fields were all aligned the saccadic RT's were significantly slower for stimuli presented in the left visual field. Rotating the head to the left or right while the eyes maintained central gaze did not affect the saccade RT's. However, rotating the patients body trunk to the left while the head and eyes remained parallel reduced the saccadic RT to left visual

field stimuli. When the patients body was rotated to the left both the left and right visual fields are projected to the ipsilateral (right) side of the patients body midline. Thus it appears that it is the body midline which is involved in the viewer centred frame of reference. Left and right could be defined in terms of the left and right sides of body trunk space and not in terms of left and right visual fields.

The possibility that neglect impairs the allocation of attention in both a viewer centred and environmental centred reference frame has been provided by Calvanio et al. (1987) and Farah et al. (1990). Calvanio et al. examined the ability of neglect patients to name pictures and words displayed in all four visual quadrants while sitting upright and reclined on their sides. When upright they omitted more stimuli in the left than right quadrants. When reclined they omitted stimuli located to their left and also more stimuli located to the left side of the screen. This finding suggests that neglect operates in terms of the viewer centred representation and the environmental centred representation. Farah et al. used a similar paradigm in which parietal patients searched for letters scattered pseudorandomly in the four visual quadrants. A further factor in this experiment was that the search task was performed within the boundary of a simple line drawing of an object. Patients again performed the task sitting upright and reclined on their sides to decouple the viewer centred and environmental frames of reference. In addition the object was also rotated so as to decouple the environmental frame from the object frame of reference. The results showed that fewer letters were reported located to the left side of the viewer and to the left side of the environment which supports the findings of Calvanio et al. that neglect operates in terms of both the viewer centred and environmental reference frame. There was no effect of rotating the object which suggests that neglect does not operate in terms of an object centred frame of reference.

Farah et al. point out in their discussion that the failure to find evidence of neglect operating in the object centred reference frame may be due to the types of objects used (line drawings of animals) which had no relationship to the nature of the search task for letters. Driver and Halligan (1991) directly address this issue by using nonsense shapes with missing left and right features. A neglect patient P.P. was asked to discriminate between two of these shapes which were presented upright or rotated 45^{o} to the left or right. P.P. neglected the left sides of the shapes when they were upright and also when they were rotated so that the critical left sided features of the object fell within the right side of the sagittal midline. This result is consistent with the idea that neglect can operate for the left sides of a representation of an object.

The results of Posner et al. (1987) and Ladavas, Petronio and Umiltá (1990) which showed slower RT's for the left stimuli presented in the ipsilesional visual field, could be interpreted as showing that the coordinates involved in the impaired orientation of attention are not retinal. There is some reason to caution against the conclusion given the finding of Driver and Halligan that an attentional impairment at the level of the object representation is also possible. If as the evidence suggests neglect can affect the deployment of attention at many

different levels of representation, then the patients could be impaired at deploying attention to both the contralesional side of space (possibly in retinal coordinates). and also to the contralesional side of an object based representation. In terms of the experiments of Posner et al. and Ladavas the increase in RT's for targets occupying the left position may well be due to an impairment in deploying attention to the contralesional side of an object. The object in this case could either be the combined representation of the marker boxes presented on the VDU screen or at a larger spatial scale the whole of the VDU itself.

There is some evidence from the work of Young et al. (1992) with B.Q, that an object centred deficit may not be entirely responsible for neglect within the intact hemifield. B.Q. was shown to be severely impaired at identifying the left side of a chimaeric object or face under free viewing conditions. Chimaeric faces were also presented briefly by a tachistoscope so that they fell entirely into B.Q.'s ipsilesional hemifield. B.Q. was again shown to be unable to identify the left sided features. The possibility that B.Q. neglected the left side of chimaeric faces due to an object centred representation was examined by testing her ability to recognise chimaeric rotated 180^{o}. B.Q. still failed to recognise the face located to her left (which formed the right side of the chimaeric), which favours the view that her neglect was in terms of a viewer centred representation.

In conclusion it can be stated that neglect appears to involve more than one frame of reference. Firstly the viewer centred frame of reference based on the midline of the visual fields and also on the body/head midline, appears to have a role in the defective deployment of attention. The experimental evidence does not rule out the possibility that part of the deficit may involve an impairment at the level of a retinal reference frame. For example although Ladavas (1987) showed that longer RT's were associated with the left most stimulus regardless of visual field, the greatest RT's were always shown for stimuli presented in the left visual field. This is consistent with the idea of a deployment in allocating attention into the left visual field (retinal coordinate) and to the left side of an object (screen or grouped structure). In the head tilted condition RT's were slower in the left than right visual field. Karnath et al. (1991) showed that the viewer centred frame could be centred on the side of contralesional side of the body midline. The work of Calvanio et al. (1987) and Farah et al. (1990) have shown that both the viewer and environmental frames of reference may both be involved in neglect. The recent work by Driver and Halligan showed that the object centred frame may also be involved in some patients. Given the possibility that different lesion locations could disrupt one or combinations of more than one of the attentional orienting systems operating at a difference reference frame, it should be expected that patients may well neglect the contralesional parts of some stimuli but not of others.

5.8 Neglect Dyslexia.

Neglect Dyslexia is a condition in which a neglect patient makes errors in reading. It takes several forms patients may omit the beginnings of single words and may also omit whole words located from the left side of a line when reading text. Neglect dyslexia is not thought to result from an impairment of the lexicon, as some neglect patients can often read single words correctly and are aware of the meaning of these words. Presenting words written vertically has also been shown to eliminate neglect dyslexia errors in some patients (Young et al 1991). Neglect dyslexia is therefore thought to result from an impairment of early visual processing rather than being a defect to the more central reading process.

Ellis, Flude and Young (1987) classified neglect dyslexia errors as being those made for the left (contralesional) letters of the word. These differ from simple misidentifications in that all the letters to the right of the 'neglect point' are identified correctly, while none of the letters to the left of the neglect point are correctly identified. Kinsbourne and Warrington (1962) showed a detailed examination of the single word reading errors made by neglect patients. There are two types of single word errors: left sided letter deletions (eg. PEAR read as 'ear'); left letter substitutions and additions (eg. PEAR read as 'gear' and PEAR read as 'spear'). Interestingly substitutions produce real word alternatives, often with a similar number of letters being substituted as was deleted, this implies an intact representation of word length (Ellis et al 1987). The second type of reading error is the omission of whole words located on the left side of the page. This second type of error has been studied less extensively than has the misreading of single words.

Patients show dissociations between whole word omission errors and single word misreadings. Some patients show intact text reading, but make single word misreading errors (Riddoch et al., 1990), while others show intact single word reading with word omissions in text reading (Kartsounis and Warrington, 1989). Young et al. (1991), suggested on the basis of errors made by S.P., that these two forms of neglect dyslexia are due to different deficits produced by the neglect condition. S.P.'s word omissions were shown to depend on the left-right spatial position of the words. Whole words were missed from the left side of a page and never from the right side, while single word misreading errors occurred equally often for words located at both the left and right side of the page. Misreading errors also occurred if the word was presented entirely within the patients right visual field following a digit located to the left of the initial letter position. The implication is that misreading errors are not dependent on a visual field defect, but on a higher level deficit in the word recognition system.

Young et al. (1991), provide a possible explanation of these dissociable impairments in reading. Initial letter reading omissions occur even with tachistoscopic presentation in which an eye movement is not required and could reflect an impairment in central mechanisms involved in word recognition. Costello and Warrington (1987), suggest that an abnormal distribution of attention in the visual word form recognition system could produce the deficit in word

recognition. It is further suggested that whole word omissions could result from an impairment of saccadic eye movements which are required to locate the left hand edge of the text (Young et al. 1991). An impairment in making a left saccade would result in the start of the next line not being located and words being omitted. There is some evidence to support the claim of impaired left saccades producing whole word deficits, but this form of neglect dyslexia has been studied much less extensively than has single word dyslexia. Huber et al. (1988) measured the eye movements of a neglect patient while reading. The left saccades made to locate the start of lines were shown to fall in the middle of the next line, not as shown in normal readers near to the start. A strategy of backward reading, involving small left saccades to locate a word which provided a plausible continuation was shown to be used by this patient. The impaired performance at making left saccades when reading was not due to an oculomotor impairment as the patient was able to produce large left saccades from the left to right border when instructed to do so. The production of small left saccades does not seem to depend on the retinal border between LVF and RVF, as they were made from the far right end of the screen (last word of a line). It thus appears that they were influenced by the midline of the body head/trunk which is coincident with the centre of the screen.

Riddoch and Humphreys (1991), suggested that both the word omissions and misreading errors in neglect dyslexia, could result from an impaired eye/attentional system. They emphasise that there is likely to be a strong link between eye movements and the visual attentional system (Rizzolatti, 1987; Fischer and Breitmeyer, 1987). Single word misreadings may be accounted for in relation to the location of the eye within a word. O'Regan and Lévy-Schoen (1987) showed that recognition of a word is faster when it is fixated slightly to the left of centre at what they term the 'optimal viewing position' (OVP). They also showed that during normal reading there is a tendency for fixations to fall close to the O.V.P. Riddoch et al. (1990) accounted for initial letter misreadings by suggesting a shift in the OVP further to the right of centre in right brain damaged patients. Normal subjects have been shown to have a greater attentional span to the right than to the left (McConkie and Rayner 1975). A shift to the right of the OVP produced by right brain damage would account for initial letter misreadings and will produce a greater deficit in reading performance, than would a shift of the OVP to the left.

Riddoch and Humphreys (1991) further suggest how an impaired eye/attentional system could cause word omissions during text reading in neglect dyslexia. During normal text reading a person will make a series of fixations on individual words, separated by saccades across a page. Finally once the last word of a line has been read, a large amplitude left saccade, will be required to locate the start of the next line. The disorder in text reading could arise if the patient fails to direct their eyes/attention far enough into the contralesional field to detect the left sided text/margin boundary. This failure to orient sufficiently far enough leftwards along the next line results in the words located on the left side of the page being missed out.

5.9 Outline of the experimental investigation into the attentional deficits shown by a neglect patient.

The following chapters describe experiments in which manipulations of attention which have been used in normal subjects, are used in experiments with a single neglect patient (B.Q.). In Chapter six the effect of presenting single and bilateral targets for an overt eye movement, under conditions in which the central fixation point either remains on when the target appears (overlap condition), or is removed prior to target onset (gap condition) are described. The use of gap and overlap techniques have been shown to effect saccade latency in man and monkeys and may have an attentional explanation (Fischer and Breitmeyer, 1987; Mayfrank et al. 1986) are used with the patient to examine the nature of the orienting deficit. The examination aims to investigate the patients ability to report stimuli presented left and right of central fixation and to measure the saccade latencies made to these targets, under various gap and overlap conditions. The patients ability to report and saccade to double simultaneously presented targets is examined to investigate the extinction effect shown in the stages of recovery following lesion and to see if there is a bias towards ipsilesional orienting on bilateral presentation. The possibility that double targets slow saccade latency due to an attentional inhibitory effect is also examined by an examination of saccade latencies obtained with bilateral simultaneous target presentation.

In Chapter seven, B.Q.'s reading ability is examined. B.Q. is of particular interest as she shows little evidence of neglect dyslexia when reading single words, but when reading a page of text neglects the words located on the left side of the page. The hypothesis that these word omissions reflect an attentional deficit which prevents a large left saccade being made is examined in this chapter. B.Q.'s word omissions are examined by presenting lines of text onto a VDU screen and also by presenting single lines on the screen with various temporal intervals (gaps) between line presentation. It is thought that the gap intervals could enable attention to move further to the left to locate the start of subsequent lines. In further experiments the use of a stimulus flash presented at the position of the start of each line is examined to see if cueing can reduce the amount of left sided word omissions.

Chapter 6
An examination of the overt attentional orienting in a neglect patient, under gap and overlap fixation conditions.

6.1 Introduction.

Neglect is characterised by a failure of patients to respond to and report stimuli located in the contralateral side of space to the lesion, which could result from a deficit of attention (eg. Heilman and Valenstein, 1972; Karnath, 1988; Kinsbourne, 1978; Posner et al., 1984, 1987). Posner et al. (1984) suggested that the covert orienting of visual attention consisted of three separate processes: disengagement, movement and engagement on the basis of cued manual reaction time experiments. Posner et al. (1984) showed that left and right brain damaged patients show very long RT's to contralesional stimuli when attention has been cued in the ipsilesional direction. These long RT's were termed the 'extinction effect' to compare them to visual extinction which is seen when patients neglect an ipsilesional stimulus when it is paired with a contralesional stimulus. According to Posner's view neglect patients are impaired at disengaging attention from an ipsilesional stimulus to orient towards a contralesional stimulus. Morrow and Ratcliff (1988) supported the view that right brain damage produces a selective deficit of disengagement of attention which is not shown following left brain damage. They showed that the size of the extinction effect was greatest in the patients who showed the greatest degree of neglect, suggesting that the disengagement deficit is directly related to neglect and does not reflect a separate phenomenon following left or right parietal damage. Their results, in contrast to Posner's, also provide some evidence that neglect patients are impaired at moving attention in the contralesional direction as they were slower to orient attention contralesionally following a contralesional (valid) cue.

Some recent theories of visual attention have emphasised the link between the system which orients attention and the saccadic eye movement system (Fischer and Breitmeyer, 1987; Tassinari et al., 1987; Rizzolatti et al., 1987; Shepherd, Findlay and Hockey, 1986). If the attentional and eye movement systems do interact, or involve the same underlying system, then factors which manipulate covert attentional measures, could also be expected to affect overt measures of attention. Posner et al.'s three component view of attention has been adopted by Fischer and Breitmeyer (1987) to explain the results that attentive fixation has on saccadic eye movement latencies (saccadic reaction times). Saslow (1967) showed that normal subjects saccade latencies were reduced if the central fixation point was removed prior to target onset (gap paradigm). The fastest saccadic reaction times were made if fixation was removed 200 ms or more, before target onset, and the slowest SRT's were produced if the fixation point and target overlapped by 100 ms or more. Fischer and Ramsperger (1984, 1986) showed that under gap conditions distributions of saccadic reaction times showed two distinct peaks, the first at 70 ms and the second at 140 ms. The first peak was taken as indicating a separate population of saccades to the fast regular saccades, which they termed express saccades. Express saccades were also produced in overlap conditions if the subjects were instructed to

gaze at the fixation point without attending to it. Directing attention towards a peripheral stimulus with eyes gazing at central fixation has been shown to inhibit the presence of express saccades (Mayfrank et al., 1986). Fischer and Breitmeyer (1987) suggest that in order for a saccade to be generated the attentional system must first be in a disengaged state. Attentional engagement being thought to inhibit the initiation of a saccadic eye movement. With a sufficiently long gap interval attention is in a disengaged state which facilitates SRT's by saving the amount of time which would otherwise be required to complete this process. When attention is disengaged the system is able to generate express saccades which would otherwise be inhibited by attentional engagement.

The following experiments aimed to examine the ability of a patient with profound neglect to report overtly and saccade towards, single left and right field stimuli and when two targets are presented bilaterally in the left and right fields. The attentional disengagement hypothesis was examined by the use of a gap and overlap paradigm. In the basic experiment the patient (B.Q.) was required to move her eyes from a central fixation point to locate stimuli in either the left or right visual field, when the fixation stays on (**overlap**), is extinguished simultaneously with target onset (**zero-gap**), or is extinguished 100 ms before target onset (**+100 gap**). The effects of the gap and overlap condition on the patients ability to report left and right stimuli were examined and the effect on saccade latency measured by recording eye movements. Bilateral targets were used to provide a measure of extinction given that neglect often recovers in the months following initial lesion. (A published version of these experiments has already been reported: Walker et al., 1991) Bilateral targets can also be useful in examining the hypothesis of Karnath (1988) that neglect patients show a bias towards ipsilateral orienting. The appearance of a stimulus in the contralesional field which is paired with the ipsilesional stimulus could produce an increase in orienting to the ipsilesional (RVF) stimulus.

The following experiments involved testing a single neglect patient B.Q. under various conditions of reporting stimuli presented in the left and right visual fields. The patients case history is as follows:-

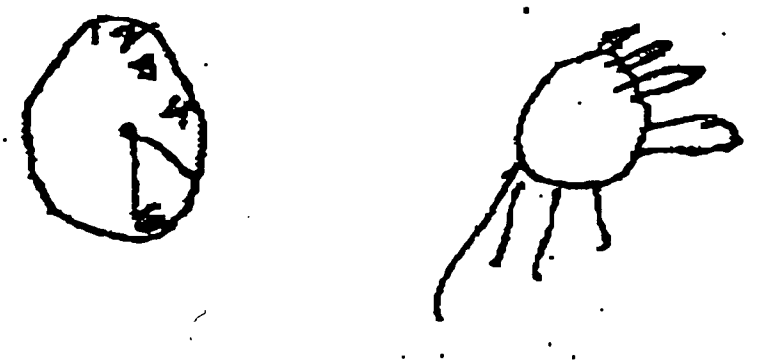
6.1.1 B.Q. Case History.

B.Q. (D.o.b 1925) is a right handed retired professional woman who was admitted to hospital in August 1989 with a suspected stroke. Neurological examination revealed normal cranial nerves and fundi, deviation of the eyes and tongue to the left, and left sided facial weakness. She also showed persistent unilateral neglect for the left side of space (See: Figure 17). On conventional perimetric testing she showed a left hemianopia with macular sparing (the issue of the apparent hemianopia is questioned in the following experiments).

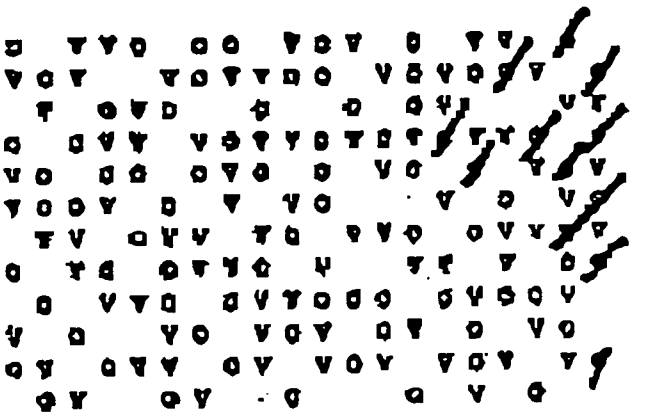
C.T. scan revealed an extensive, low density lesion surrounding the right parietal region and moderate midline displacement. The appearances were consistent with an infarct in the territory of the right middle cerebral artery.

Psychological examination in September 1989 showed no significant impairment of verbal recall (logical memory passage from the Wechsler Memory Scale: immediate 10, 10; after 40 Min. delay 10, 10), or immediate digit span (7 forward and 4 backwards). There was no evidence of a speech or verbal comprehension deficit during conversation, but the pro-rated IQ score of 97 (NART Nelson 1982) was below that expected given her previous career. B.Q. showed signs of unilateral spatial neglect on line bisection with lines over 12.5 cm long (40-50% shifts from midpoint), letter cancellation and when copying drawings. When reading she missed out words on the left side of the page. Visual copying tasks and visual short term memory were severely impaired. The block design test of the Wechsler was impossible. Occupational therapy revealed problems of body image and spatial relations and also indicated dressing dyspraxia.

Visual field testing was again performed in July 1990, using screen perimetery and again confirmed the presence of a left hemianopia with macular sparing. Near visual acuity ('Curpax' test Curry and Paxton) was shown to be good (approximately 6/4) and contrast sensitivity was shown to be in the normal range (Vistech VCTS 6000).



B.Q.'s drawing of a clock and daisy from memory: both show missing left features.



Crossing out task: B.Q. has omitted to cross out the O's on the left side of the page.

Figure 17.

Illustrations of B.Q.'s unilateral spatial neglect:-

6.2 Experiment N1: The effects of prior fixation point offset on B.Q.'s ability to overtly report left and right stimuli.

6.2.1 Introduction

The following experiment examined B.Q.'s ability to overtly report a single left, or single right, sided stimulus (digit), and to report a stimulus when two digits were presented bilaterally and simultaneously left and right of fixation. There were three different testing conditions: overlap:- the fixation point *stayed on* throughout each trial; zero-gap the fixation point offset was *simultaneous* with target onset and +100 gap where the fixation point offset occurred 100 ms *before* the onset of the target.

6.2.2 Method.

Apparatus.

Stimuli were presented onto a VDU screen, generated by a BBC master series microcomputer (See: General method section Chapter 2), using the mode 1 character set.

Procedure.

B.Q. was seated centrally 50 cm from a VDU screen which was mounted at eye level. A central fixation cross was displayed, which coincided with B.Q.'s head/body midline, so that the LVF and RVF were also coincident with the left and right sides of head/body space. Head movements were not constrained during testing.

The stimuli timing sequence used in the three conditions (overlap, 0-gap and +100 gap) is shown in Figure 18. In the overlap condition the targets appeared 1000 ms after initial fixation onset and the fixation point remained on throughout the whole trial sequence. In the zero gap condition the fixation point was displayed for 800 ms, its offset was simultaneous with the onset of the target. In the +100 gap condition the fixation point was displayed for 900 ms and went off 100 ms before, the onset of the target stimulus.

After an inter trial delay of 500 ms, during which time the screen remained blank, a fixation cross (size 0.57°) appeared in the centre of the screen. An audible bleep occurred simultaneously with fixation onset as a warning signal to indicate the start of the next trial. The target was a uniformly illuminated square (sides 0.57°) which was presented for 100 ms. A delay of 100 ms then occurred, during which time it would be expected that an eye movement would be made. An indicator then appeared at the target location displayed for 300 ms, which B.Q. had to report. The indicators were digits 1 to 4 (size 0.57°) which were presented in a random sequence. The digit was then replaced by a mask (same square stimulus as constituted the target) presented for 100 ms. The timing sequence and size of the stimuli was such that it was only possible for the indicators to be reported if an accurate eye movement was made to the target location within some 200-300 ms from the time of initial target onset.

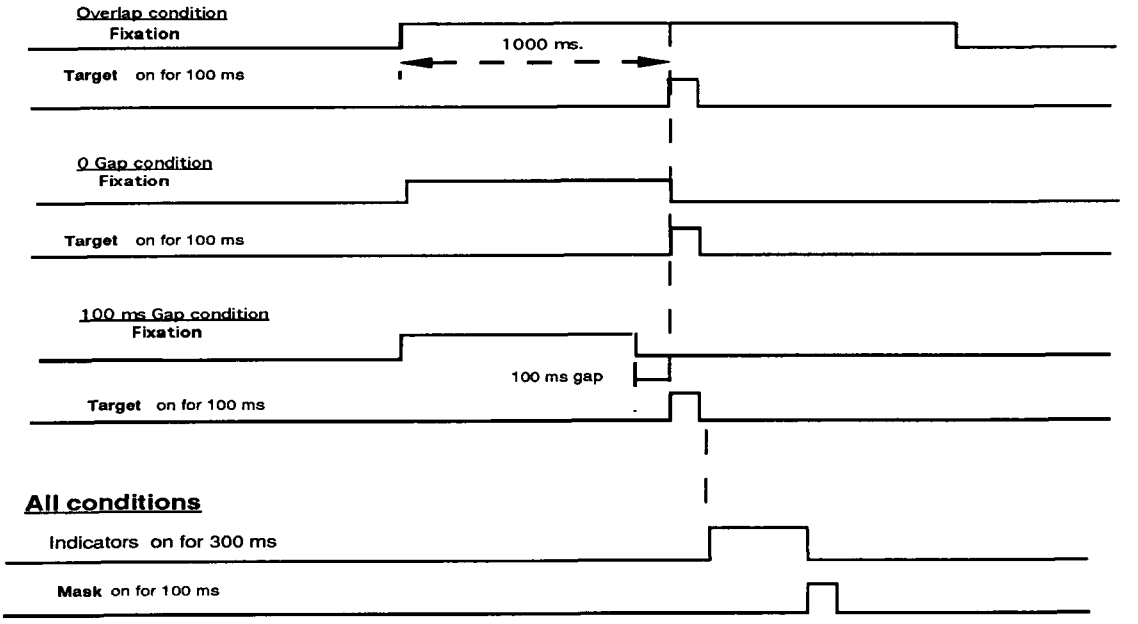


Figure 18.
Timing sequence of st imulus presented in the overlap, 0 gap and 100 ms gap conditions.

The target eccentricities were; 2.5, 5.0 and 7.5 degrees, left and right of the horizontal midline, on the horizontal and principal oblique axes. This produced nine left and nine right visual field target locations. The stimulus locations are displayed in Figure 19. Each condition contained 144 trials with equal numbers of single and double target trials in each block. There were 72 single target trials, where a target appeared at one of the 18 possible target locations. There were also 72 double target trials, in which two targets appeared simultaneously at equal and opposite locations, on either the horizontal axis, or diametrically opposed on one of the principal oblique axes. The order of presenting single and double targets was randomised throughout each block of trials.

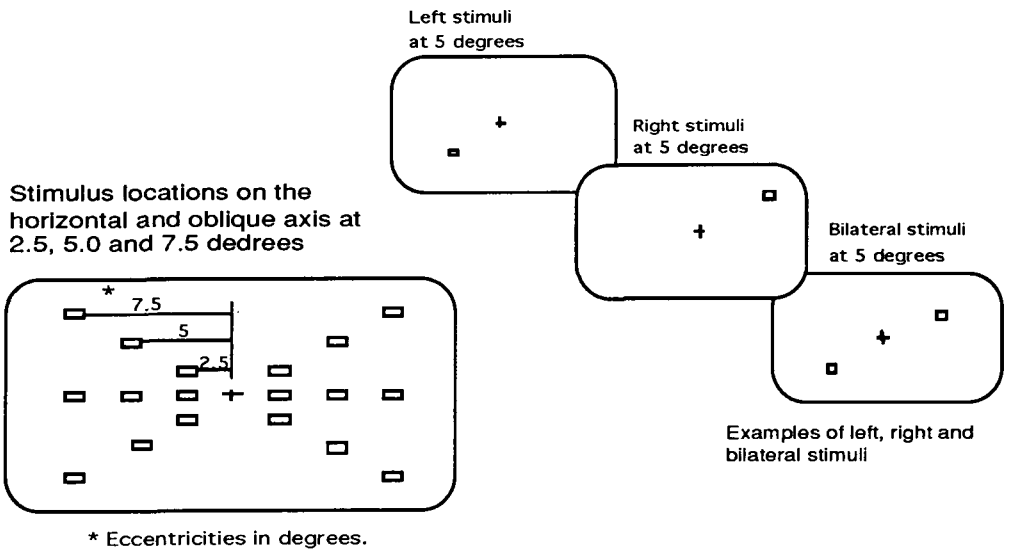


Figure 19.
Stimulus locations used in Experiment N1.
(Not to scale)

B.Q. verbally report the presence of the fixation cross when it was detected by saying 'cross'. This indicated that central fixation had been achieved on the start of each trial. This verbal reporting of fixation had occurred spontaneously on a previous testing sessions and was encouraged by the experimenter. B.Q. was instructed to move her eyes to the location of a 'flash' on the screen and to report the number which appeared at that location. The experimenter keyed these responses into the computer for later analysis. The overlap and +100 ms gap conditions were carried out on the same day of testing, the zero gap condition was carried out some two weeks earlier. Each testing session lasted approximately 30 minutes.

6.2.3 Results.

The percentage of single and bilateral LVF and RVF stimuli reported by B.Q. under the three fixation conditions, are shown in Figure 20. The percentage of single stimuli reported for each of the stimulus eccentricities, are shown in Figure 21.

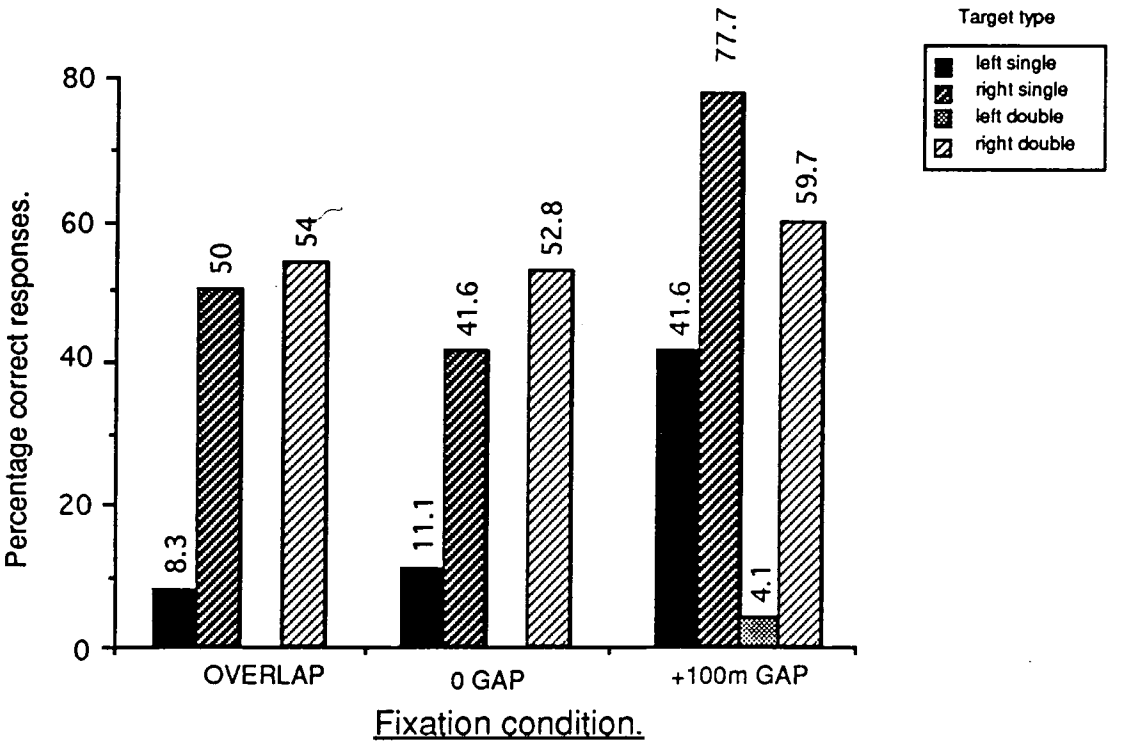


Figure 20.
Percentages of stimuli reported by B.Q. in Experiment N1, in the overlap, 0 gap and 100 ms gap conditions.

The percentages of responses that B.Q. made under the overlap and zero gap conditions were highly similar. B.Q. reported right single targets on 50% of overlap trials and on 52.8% of zero-gap trials. Stimuli were reported from all nine RVF target locations, from all three eccentricity locations. The amount of left single targets which B.Q. reported was drastically reduced. B.Q. reported 8% of left stimuli in the overlap condition and 11% of left stimuli in the zero-gap condition. All but one of these left responses were for stimuli at the nearest 2.5°

eccentricity location, the one other was located 5° left of fixation. On bilateral double simultaneous target trials B.Q. always reported the RVF stimulus (never the LVF stimulus), and reported some 52.8 - 54% of right stimuli on these trials. A chi-square comparison confirmed that there was no difference between the numbers of stimuli reported in the overlap and zero gap conditions ($p>0.05$).

In the +100 ms gap condition the amount of stimuli reported by B.Q. has increased for both left and right stimuli. Right stimuli were reported on 77.7% of single target trials and right double targets were reported on 59.7% of trials. The most dramatic improvement was shown for left single stimuli which were reported on 41.6% of trials. B.Q. also reported one LVF target under bilateral simultaneous target presentation. B.Q. reported left stimuli from all target eccentricities with single target presentation, as can be seen in Figure 21.

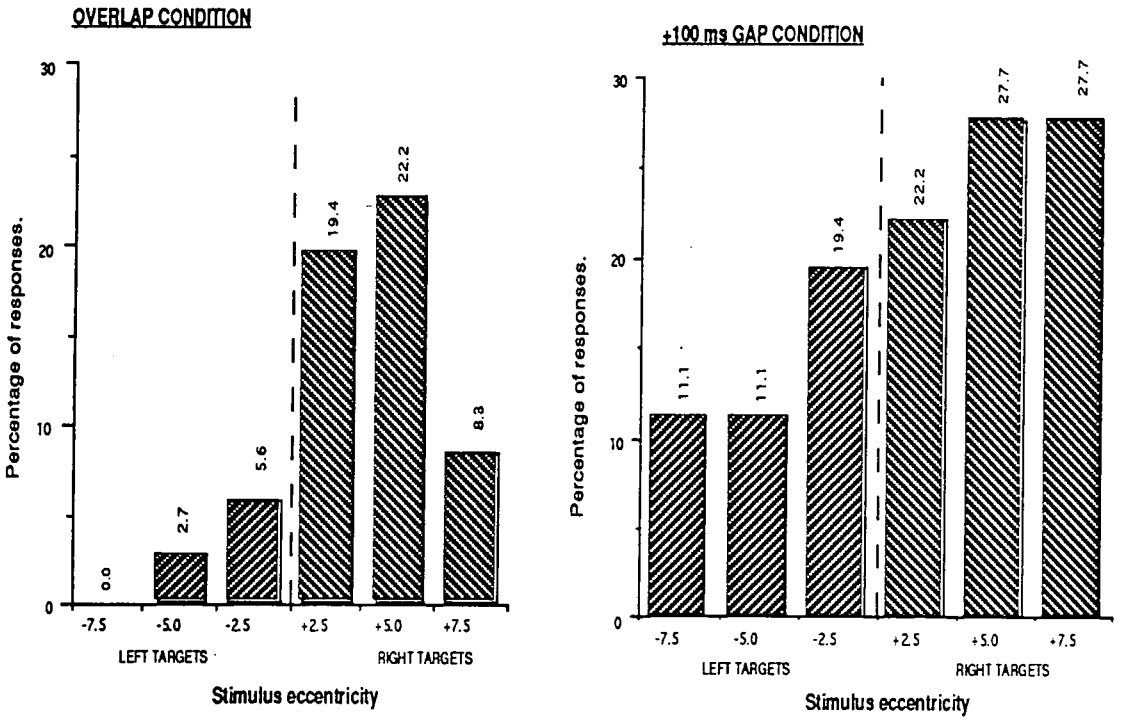


Figure 21.
The eccentricity of left single and right single stimuli reported by B.Q.

A chi-square statistical analysis was performed to compare the results obtained in the +100 gap and overlap conditions (as these results were obtained on the same day). The increase in reporting of LVF targets in the +100 gap condition was highly significant (chi-square = 9.55 $p < 0.01$), as was the increase in reporting RVF targets (chi-square = 6.02 $p < 0.05$) when compared to the numbers reported in the overlap condition. The advantage shown for reporting RVF single targets (77.7%) compared to LVF single targets (41.6%) in the +100 gap condition, was also significant (chi-square = 9.86 $p < 0.01$).

6.2.4 Discussion.

In this experiment B.Q.'s ability to overtly orient towards LVF stimuli has been shown to depend on the current state of the attended fixation point. In the overlap and zero gap conditions, B.Q. reported only 8 - 11% of LVF stimuli, which were all at the near target locations. This performance is consistent with the results of the perimetric field plot which suggested the presence of a left hemianopia with macular sparing at up to 5 degrees. B.Q.'s failure to report more of the targets at the 2.5° location, which should fall within the spared area, is consistent with her showing left neglect in addition to a hemianopia. However, her performance on the +100 gap condition showed a dramatic increase in the amount of left stimuli reported at all eccentricity locations. B.Q. responded to LVF stimuli when the fixation point was extinguished 100 ms before target onset. This suggests that she does not have a left hemianopia but fails to report LVF stimuli under the overlap condition and under perimetric field plotting, because of her profound left neglect (Walker et al., 1991). The 100 ms gap appears to reduce the severity of B.Q.'s neglect and enables her to make saccades to, and overtly report left stimuli. In addition to the increase in left stimuli the use of the +100 ms gap condition also increased the amount of right single stimuli that B.Q. reported by some 20%. This increase in right stimuli reporting suggests that part of the effect obtained in the gap condition could be due to an increase in B.Q.'s overall level of arousal, which is also thought to be a factor involved in neglect (Karnath, 1988; Robertson and Frasca, 1992).

B.Q.'s ability to report LVF stimuli in the 100 ms gap condition, is consistent with Posner et al.'s (1984) deficit of attentional disengagement hypothesis of neglect. Prior fixation offset can disengage the attentional orienting system to enable a normal left saccade to be made to the LVF stimuli. In the overlap condition the presence of the fixation point means that attention remains engaged, which suppresses an orienting response to contralesional stimuli. The results of the present experiment strongly suggest that the disengagement process must take a certain amount of time to complete and that Rizzo and Hurtig (1992) were premature in rejecting the disengagement hypothesis of neglect on the basis of results from a zero gap condition. The idea that attentional disengagement takes a certain amount of time to complete is entirely consistent with gap/overlap experiments, measuring saccade latency. It has been shown that saccade latency decreases consistently as the gap intervals increases (e.g. Saslow, 1967; Reulen, 1984 ab). Fischer's (1987) attentional model of the gap effect suggested that the production of a saccade is inhibited by the engagement of visual attention. There should be a reduction of this inhibition following fixation offset as attention becomes disengaged, thus facilitating saccade latency in normal subjects. In the case of a neglect patient the presence of this inhibition during engaged attention could be great enough to prevent attentional orienting in the contralesional direction. Neglect patients show a deficit of covert and overt orienting of attention which is consistent with the suggestion that a common system could be involved in controlling overt and covert attentional orienting (Rizzolatti et al., 1987; Tassinari et al., 1987).

The use of a 100 ms gap was also shown to have improved the amount of right stimuli

that B.Q. reported. This is consistent with the idea that part of the gap effect reflects a generalised warning signal effect (Ross and Ross, 1980, 1981). Karnath (1988) suggested that there are three factors involved in producing the neglect syndrome and that one of these is a reduction in the general level of arousal which reduces the patients ability to orient attention. Fixation offset increased the patients arousal and improves ipsilesional orienting by a warning signal effect. Neglect is also characterised by a bias of orienting to the ipsilesional side of space, the patient will not respond to LVF stimuli on bilateral trials, thus producing the 'extinction' of left stimuli. The use of the +100 gap has therefore reduced B.Q.'s left neglect to the milder extinction like disorder. This is consistent with the idea of a similar attentional explanation being applied to both extinction and neglect (Karnath, 1988; Young and DeHaan, 1990). Karnath (1988) suggested that the deficit in voluntary contralesional orienting recovers before the automatic orienting of attention to the ipsilesional side. This results in a reduction of neglect, with the tendency to orient ipsilesionally with bilateral stimulus presentation.

An alternative to the attentional explanation of B.Q.'s ability to report left stimuli in the gap condition is that she does have a hemianopia and is responding by a blindsight mechanism (Weiskrantz et al., 1974; Barbur, Forsyth and Findlay, 1988). It is difficult to accept that B.Q. can make a left saccade into a hemianopic field by a blindsight mechanism in the +100 gap condition, but not in the zero-gap or overlap conditions. If fixation offset is acting as a warning signal to trigger a left saccade then there would be expected to be evidence of stimuli reporting increasing in the zero gap condition. Furthermore, blindsight patients such as D.B. (Weiskrantz et al., 1974) are told to make a saccade to a location where he 'guessed' the stimulus had appeared. In contrast B.Q.'s responses were reflexive and made spontaneously in the +100 gap condition. It therefore seems unlikely that a blindsight explanation could account for B.Q.'s performance in the+100 gap condition.

The results of this experiment allow some discussion of the attentional model which provides the best explanation of B.Q.'s deficit. Heilman's akinesia hypothesis states that the damage to the right hemisphere results in the neglect patients selective loss of the orienting response to contralateral space, rendering the right hemisphere 'akinetic'. The nature of B.Q.'s deficit is such that a contralateral orienting of attention was not possible when a fixation point was continuously displayed, but can occur normally when fixation was removed 100 ms prior to the onset of the contralateral stimulus. This suggests that B.Q. cannot be regarded as being akinetic. The selective improvement in contralateral orienting which occurs with fixation offset is not easily accommodated into Heilman's akinesia model of neglect. Kinsbourne's hemispheric activation hypothesis could account for B.Q.'s performance on this task, but only with the addition of some extra assumptions. According to Kinsbourne neglect results from a loss of an inhibitory component acting on the left hemisphere, which results in the left hemisphere being overactive and biases attention to the right side of space. This model could account for the gap effect, if it is further assumed that during active fixation the left hemisphere is over activated (due the loss of the inhibitory component) and this results in the right hemisphere being strongly inhibited. Fixation offset could reduce the activity level of the left hemisphere, with the effect of reducing the inhibition on the right. A reduction in the inhibition acting on the right hemisphere

during the gap interval, could allow a left orienting response to be made.

The present experiment has shown that prior fixation offset improved B.Q.'s ability to orient towards and report left and right stimuli. The effect that fixation offset has on the latency of B.Q.'s saccades is examined in the next experiment.

6.3 Experiment N2: The effect of prior fixation point offset on B.Q.'s saccade latency.

6.3.1 Introduction

In experiment N1, B.Q. failed to report contralesional stimuli in the overlap condition, but did report contralesional stimuli in a +100 ms gap condition. The present experiment aims to record the patterns of eye movements made by B.Q. to left and right targets, using similar gap and overlap conditions. The eye movement record should show if there is any evidence of B.Q. making a left saccade in the overlap condition, and if she makes normal amplitude and latency, saccades in the gap condition. As the gap effect is known to facilitate saccade latency in normal subjects it is of interest to see if a similar speeding is shown by B.Q. The results with normal subjects (see: Chapters two, three and four) showed that bilateral simultaneous target presentation slowed the latency of saccades. The effect of bilateral simultaneous target presentation with B.Q. will also be examined in this experiment, to show if a similar inhibitory effect is noticed on her saccade latency.

This experiment involves presenting saccade targets in 100 ms gap and overlap conditions, while recording B.Q.'s eye movements. The experiment is similar to Experiment N1, except that the targets are presented on the horizontal axis only, for ease of interpreting the saccade records. An overlap and 100 ms gap condition will be used with left and right single targets and bilateral simultaneous targets.

6.3.2 Method.

Apparatus.

In addition to the apparatus used in Experiment N1 the following equipment was used to record and examine the eye movement records. An infrared binocular eye recorder (ACS model EM130) was used to record B.Q.'s eye movements. Analogue records were stored on a DC tape recorder (Tinberg series 100). One channel was used to record the eye movements while another stored a marker signal (from the BBC) which indicated the start and finish of each trial. The analogue data was later sampled at a rate of 10 ms and converted to a digital value for data storage and saccade analysis. The ADC and later saccade analysis was performed using a

Cambridge Electronic design Alpha computer with 502 interface. A chin rest was used to restrict the patients head movements during the eye movement recording sessions.

Procedure.

The stimuli used and the timing sequence of stimuli was identical to that used in experiment N1. The one difference being that targets were presented on the horizontal axis only, at eccentricities of 4^{o} and 8^{o}, left and right of central fixation. A calibration record was used at the start of each session which involved recording the patient's eye movements to a circular target as it tracked from centre to the far left and far right target locations and back to centre. There were two separate testing sessions, one for the overlap and one for the +100 gap condition, carried out on different days. B.Q. was given the same instructions as those in experiment N1, to move her eyes to the targets when they appeared on the screen and report the digit which appeared at that location.

6.3.3 Results.

An examination of each eye movement record indicated that B.Q. maintained her eyes at the central fixation point until the target appeared and made a saccade on some trials. In addition there was some evidence of small head movements being used along with a saccade to detect the targets. The saccades were clearly identifiable in all records (where one was made) except when a blink occurred. Trials with blinks were discarded and not included in later analysis. Saccades of latency less than 100 ms and greater than 500 ms, were not included in the analysis. This resulted in 4% of saccades being excluded, all of which were greater than 500 ms, no saccades of less than 100 ms latency were found. The saccades made on bilateral targets trials were all made to the RVF target, and never to the LVF target.

Figure 22, shows the mean saccade latency made by B.Q. to single and bilateral targets (combined for the two target eccentricities); in the overlap and +100 gap condition. The Figure also shows the numbers of saccades included in each and the standard deviations of those saccade latencies.

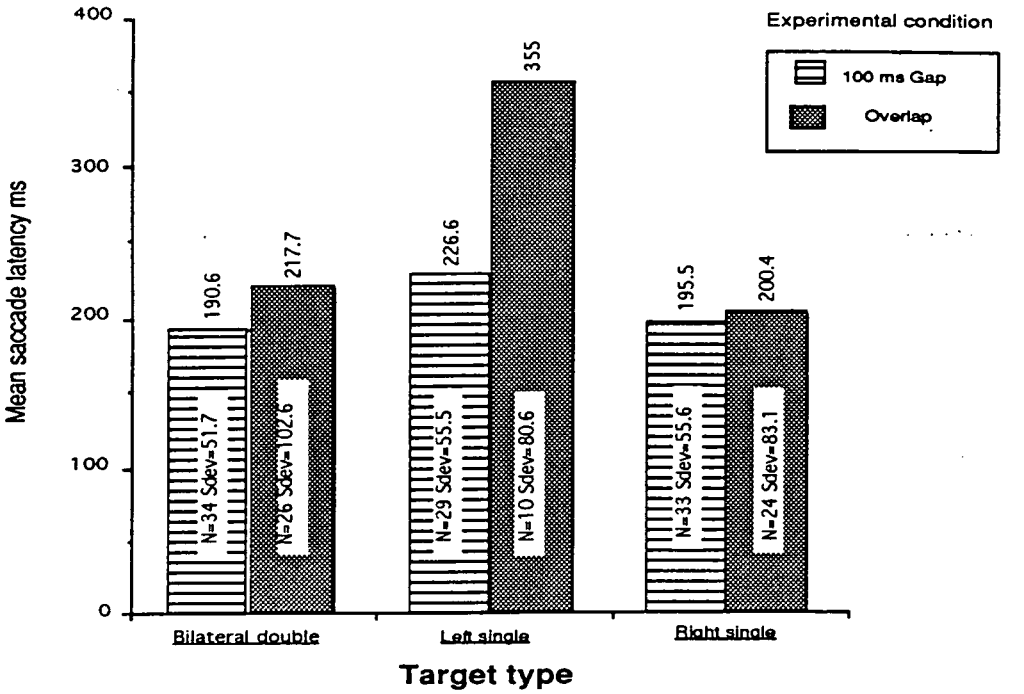


Figure 22.
B.Q.'s mean saccade latency to single and bilateral targets, in the overlap and 100 ms gap conditions from Experiment N2.

The percentages of indicator digits correctly reported by B.Q. during the eye movement recording sessions are shown in Figure 23. The pattern of results is similar to that shown in experiment N1. The use of a +100 ms gap has improved left stimuli reporting from 0% in the overlap condition to 22.5%. There was no increase in the numbers of right single stimuli reported in the gap condition, but an improvement is shown for RVF stimuli on bilateral presentation in the gap condition.

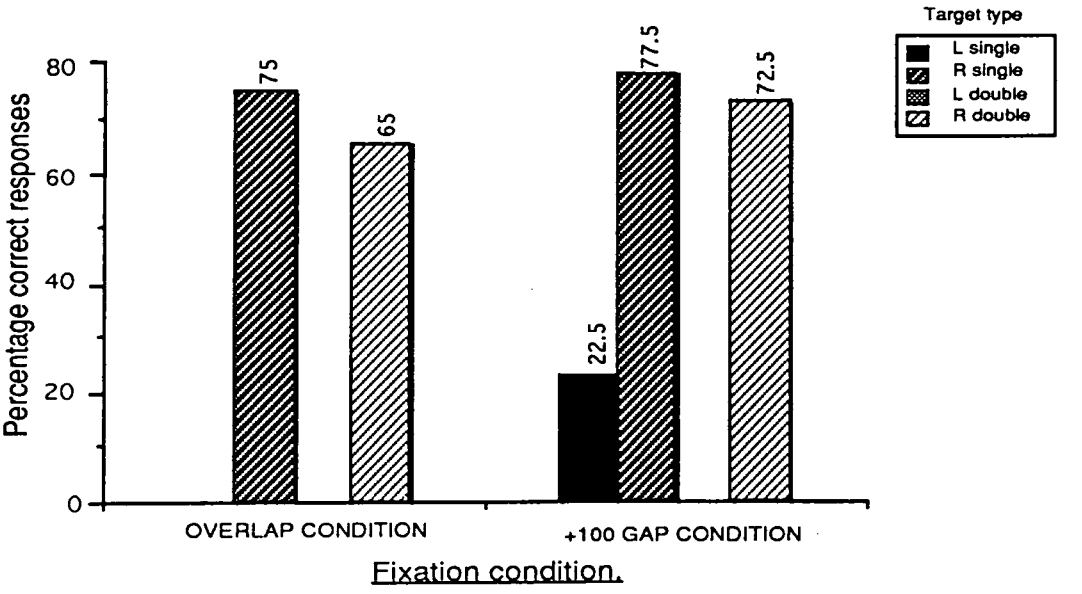


Figure 23.
The percentage of stimuli reported by B.Q. in Experiment N2 during eye movement recording.

A (three way) ANOVA with factors of condition (Gap/overlap), target type (single/bilateral) and target eccentricity (2 degrees and 4 degrees) compared the mean latencies obtained. There was no effect of target eccentricity on saccade latency ($F = 0.54$ $p > 0.46$). There was a significant difference between the mean saccade latency obtained in the +100 gap (204.2 ms) and overlap (255.1 ms) conditions ($F=16.58$ $p < 0.001$). There was no evidence of the +100 gap condition speeding the latency of saccades made to RVF stimuli, under either single, or bilateral target presentation. There was no slowing of saccade latency produced by bilateral target presentation in the gap, or overlap conditions, this is in contrast to normal subjects who do show a slowing with bilateral presentation (Lévy-Schoen, 1969, 1974; Findlay, 1983). A significant two way interaction was shown for target type and experimental condition ($F = 8.45$ $p < 0.001$). The interaction effect results from a greater slowing of saccade latency for left single targets in the overlap condition. The mean latency shown for saccades made to left single targets was slower than to right single, or right bilateral targets, in both the gap and overlap conditions. This slowing of saccade latency made to left targets was examined for the overlap and gap condition results using two (one way) ANOVA's. Each ANOVA had three three levels of target (left single, right single and right bilateral), and both showed a significant main effect overlap ($F = 4.53$ $p < 0.001$); gap ($F = 2.316$ $p < 0.05$). A Tukey (HSD) procedure confirmed that the saccades made to left targets were significantly slower than to right single, or right bilateral targets, in each condition.

In the overlap condition B.Q. failed to report any of the left indicators. Interestingly, an examination of the eye movements revealed that a left saccade had occurred on ten (25%) of left single trials. The latency and amplitude of the saccade should have been sufficient for her to report the indicator at the 4^{o} location. There were a further nine occasion when a left saccade was made in the +100 gap condition and the indicator was not reported. Right single and right bilateral indicators were not reported on 20 - 22% of trials in which a saccade was made.

Table 9 shows the percentages of saccadic responses made in each recording session. This shows the percentage of trials in which:- 1] a saccade was made in the target direction (CORRECT): 2] a saccade was made in the opposite direction to the target (OPPOSITE): 3] no saccade was made (NONE): 4] a search pattern of eye movements was suggested by the presence of two or more small saccades being made away from fixation (SEARCH): 5] the record was discarded due to a blink (BLINK): 6] the record could not be interpreted due to general noise which made it difficult to examine the record (U/S).

Table 9. The percentage of saccadic responses made by B.Q. in Experiment N2.

a) OVERLAP CONDITION.

TARGET	CORRECT	OPPOSITE	NONE	BLINK	SEARCH*	U/S
DOUBLE	62.5	n/a	5	20	0 2.5 R	10
LEFT	32.5	0	20	30	10 L 7.5 R	0
RIGHT	67.5	5	2.5	17.5	0 L 5 R	2.5

*L= left search: R= right search.

b) GAP CONDITION.

TARGET	CORRECT	OPPOSITE	NONE	BLINK	SEARCH*	U/S
DOUBLE	92.5	n/a	0	5	0 0	2.5
LEFT	67.5	7.5	2.5	22.5	0 0	0
RIGHT	90	5	2.5	2.5	0 0	0

*L= left search: R= right search.

In the gap condition B.Q. made more saccades than in the overlap condition. B.Q. failed to initiate a saccade to left stimuli on 20% of trials in the overlap condition, but usually initiated a saccade in the gap condition. In the +100 gap condition B.Q. occasionally made a saccade in the opposite direction to the target, and some of these responses resulted in a left saccade being made following a right target. A small number of saccades were made in the opposite direction to a right target in the overlap condition. This is consistent with the idea of fixation offset acting as a warning signal which can result in a saccade being initiated in the wrong direction, due to an anticipation effect.

The overlap condition records also revealed some evidence that B.Q. was making search patterns of eye movements, both left and right of fixation. There was no evidence of searches being made in the gap condition. These searches were indicated by the presence of one or two small step saccades being made and were most frequent on left target trials. The resulting saccades were made in either direction and did not appear to relate to the target position. Patterns of search saccades also occurred on bilateral and right single trials in the overlap condition and were always made right of fixation. This suggests that B.Q. could have been 'cued' by the timing sequence of the experiment, during a long delay (during which no targets were detected) a random search for a target was prompted. Blinks occurred in both the gap and overlap conditions, although the greatest number occurred following left targets under the overlap condition. The records showed that blinks often occurred at the time of target onset.

The results shown in Table 9 indicate that in the overlap condition B.Q. often fails to initiate a left saccade and her eyes remain at fixation. The gap condition enables B.Q. to produce many more saccades to both left and right stimuli. B.Q. appears to be aware that a target could have been produced on some of the trials where she did not detect its initial onset, which occasionally results in a random search for a target. Trials in which a blink occurred could not be interpreted, but on some of these trials the digit was reported so the blink occurred around the time that a normal saccade was made to the target.

6.3.4 Discussion.

The use of the overlap and +100 ms gap conditions have replicating the finding from experiment N1, in terms of the amount of stimuli that B.Q. reported. Left stimuli were only reported when the fixation point was removed prior to target onset in the 100 ms gap condition.

The gap condition also enabled B.Q. to report more right stimuli. The eye movement recording indicated that B.Q. often fails to initiate a normal latency saccade to left targets in the overlap condition and her eyes remained engaged at the fixation location. The use of a 100 ms gap between fixation offset and target onset enabled B.Q. to produce a left saccade of normal latency and amplitude. In the overlap condition a small number (10) of left saccades were made, but these were all shown to be of long latency and small amplitude. B.Q. did not report the left indicator on any of these trials, even thought the indicator should have been present for a further 200 ms, after the saccade was made. These saccades were shown to be of small amplitude, but some were sufficiently large to detect the indicators at the 4° location. B.Q. made normal amplitude and latency saccades on 67.5% of left stimuli trials in the +100 gap condition, but only reported 22.5% of the indicators. In this case it is possible that the left stimuli could have been 'extinguished' from B.Q.'s conscious awareness and are not reported, even though they may have been fixated. There was some evidence in the eye movement records of a search pattern of saccades being made to locate stimuli. This implies some use by B.Q. of top-down knowledge that a stimulus could have been presented, resulting in her making some attempt to locate a stimulus, by making a saccade to either the left or right of fixation.

In the overlap condition B.Q.'s saccade latency was very much slower when made to left single targets, than when made to right single, or bilateral simultaneous targets. The 100 ms gap condition enabled B.Q. to make a normal latency saccade to left stimuli. The use of the gap condition did not reduced B.Q.'s saccade latency, even when the saccade was made to right single targets. This is in contrast to the speeding noted with normal subjects under gap conditions (e.g. Saslow, 1967; Fischer, 1987). B.Q.'s saccade latency is also slower than observed by normal subjects on a similar version of this task (Chapter two: Exp1 and 2). It should be noted that the normal subjects were younger than B.Q. and it is possible that the gap effect on latency is not as pronounced in older subjects. A second possibility is that the nature of B.Q.'s brain damage has lead to a general increase in saccade latency, which results in the lack of any facilitation in the gap condition. Lynch and McLaren (1989) showed that unilateral lesions to the parietal lobe in monkeys produced an increase on saccade latency. This is consistent with the idea that the parietooccipital cortex is involved in the initiation and control of visually evoked saccadic eye movements. The gap condition has not speeded saccade latency, but does increase the number of saccades made to targets presented left and right of fixation. This is consistent with Fischer and Breitmeyer's (1987) attentional model of saccade generation, in which active fixation inhibits the production of a saccade. In Posner et al.'s (1984) terms attention cannot be disengaged from fixation, when a movement in the contralateral direction is required. This failure of disengagement appears to apply to both overt eye movements as well as to the covert orienting of attention.

There was no evidence of B.Q. making multiple saccades to locate left stimuli in the gap or overlap conditions. This provides further support for the view that B.Q. is not hemianopic and does not rely on a blindsight mechanism to locate left stimuli. Weiskrantz et al.'s (1974) patient D.B. located stimuli in his blind field by making multiple saccades in a search strategy.

Meienberg et al. (1981) revealed that hemianopic patients without neglect, showed staircase patterns of 'step' saccades to locate targets within their blindfield. There was no evidence of a search pattern involving a series of saccades being made by B.Q. who in contrast appears able to initiate a normal single saccade to locate left stimuli in the gap condition. The conclusion being that B.Q. does not respond to left stimuli due to her profound unilateral neglect and a procedure which reduces the severity of the neglect enables her to produce a single, goal directed saccade, to left stimuli.

In the present experiment and also in Experiment N1, there was some evidence that B.Q. reported more right stimuli on bilateral simultaneous target presentation than on single target presentation. This was a weak trend but observed on more than one occasion. As this finding is consistent with the idea of an improvement for ipsilateral stimuli reporting which is suggested by Karnath (1988) and is consistent with Kinsbourne's model of neglect this trend will be examined further in experiment N4. The next experiment examines B.Q.'s ability to make a manual point response to left and right stimuli, under gap and overlap fixation conditions.

6.4 Experiment N3: Manual pointing to left and right targets under gap and overlap conditions.

6.4.1 Introduction.

The use of a gap between fixation offset and target onset has been shown to reduce the severity of B.Q.'s left neglect on the saccadic eye movement experiments (experiments. N1 and N2). The previous experiments described in this chapter have all required a saccade to be made to a target location, so that the indicator digit can be reported. There was evidence that on some occasions B.Q. did not report the indicator, even when a saccade has been made to the target location. This could reflect a reduction in B.Q.'s overall information processing capacity (Karnath's Component C) so that she fails to report a stimulus that she has seen. In the present experiment the gap and overlap conditions were repeated, but in this case B.Q. was required to point to the location on the screen where the stimulus was presented. A pointing task will presumably be less affected by an impairment to B.Q.'s information processing capacity. It should be noted that a manual pointing tasks are more accurate when the target is fixated, but a response can still be made without a saccade. Weiskrantz et al. (1974) showed that the blindsight patient D.B. could point to a stimulus presented into his blind field on request and his pointing responses were much more accurate than were his eye movements. Although a pointing response may not require an accurate saccade to that location, it will presumably require a shift of attention to that location.

The issue of motor responses in neglect patients is a complicated one and has been shown to be related to the distance of the stimuli from the subject and the type of response required to locate that stimulus. Rizzolatti and Camarda (1987) proposed a premotor theory of

spatial attention to account for the specific motor deficits shown after damage to multiple brain areas. For example, monkeys with lesions to brain area 6 (Rizzolatti et al., 1983) could respond to food by grasping with its ipsilesional arm, if it was within reaching distance (distant peripersonal space) and outside its reach (far space), but the animal showed neglect if it was required to grasp for food in the area of its mouth (using a mouth grasping response). These animals showed the ability to shift attention in far and peripersonal space, but not when the stimulus was located within space that required a head/mouth motor response. Rizzolatti et al. (1983) showed that monkeys with lesions in the frontal eye fields showed an inability to produce a saccade to stimuli located in far space, resulting in neglect. In contrast to the monkeys with area 6 lesions they could make an accurate mouth grasping movement to contralesional stimuli in the mouth area.

The finding that neglect depends on the distance of the stimulus from the observer is supported by studies of human neglect patients. Bisiach et al. (1986) studied a large number of neglect patients on tasks in extrapersonal and peripersonal space (reviewed by Rizzolatti and Camarda, 1987). The extrapersonal neglect was assessed by asking the patient to perform a crossing out task, and personal neglect assessed by asking the patient to touch their contralesional hand with their ipsilesional hand. Most patients showed neglect in both extrapersonal and personal space, but some showed only extrapersonal neglect and others showed only personal neglect. Rizzolatti and Carmada (1987) account for these dissociations, in terms of a revised version of Rizzolatti's (1973) premotor theory of spatial attention, where a stimulus triggers neurons which plan actions to be made in space. There are several circuits of these neurons and those controlling the same space sector are activated by the stimulus, while those controlling different space sectors are inhibited. The space sector is responsible for transforming a motor plan into action by producing a suitable motor response. The final suggestion of Rizzolatti and Carmada's model is that monkey studies suggest that there are three main circuits of spatial attention which connect the frontal and parietal lobes. The first conveys propioceptive information, damage to this circuit does not produce neglect. Damage to the second and third circuits produces oculomotor neglect and the peripersonal space-reaching neglect respectively. Oculomotor neglect is thought to result from a lesion to one of several premotor circuits involved in spatial attention.

Tegnér and Levander (1991) used a line bisection task with neglect patients, performed under normal viewing, and mirror viewing, conditions. The use of the mirror resulted in the shift of attention being made in the opposite direction to the motor response made while crossing out. The results showed further evidence for the fractionation of the neglect condition and the distinction between motor, and attentional, neglect. Four patients could direct their attention contralesionally, but could not make an arm movement in that direction. Ten patients failed to cancel lines to the left under normal viewing conditions, but then failed to cancel lines to the right under mirror viewing conditions. This performance suggests an inability to shift attention into the contralesional (left) side of space, but no impairment of making a motor movement into the contralesional side. Our previous experiments with B.Q. have indicated that she is impaired at making an eye movement to contralesional stimuli when she is attending to the central fixation

point. It is of interest to show if this same deficit also affects her ability to make an overt pointing response into contralesional space.

The gap and overlap experiments performed with B.Q. used a screen located some 50 cm away. This is within the area of personal grasping and reaching space. The results showed that B.Q. was impaired at making an overt saccadic eye movement if the fixation point remained on. The present experiment examines the possibility that a similar pattern of results will be produced if a pointing response is required. This would be consistent with the idea that neglect would be expected for all motor responses which would be used to locate stimuli in this space sector and would be controlled by a common spatial attentional circuit. Alternatively, if B.Q.'s deficit primarily affects the oculomotor centre then she may well perform much better in a pointing task than in a saccade task, as was shown to be the case with the blindsight patient D.B. (Weiskrantz et al., 1974).

6.4.2 Method.

Apparatus and procedure.

The apparatus was identical to that used in Experiment N1. The stimulus presentation and timing sequence used a modified version of the gap and overlap conditions of Experiment N1. Single targets were presented to the left, or right, of fixation on either the horizontal , or oblique axis at one of the 18 screen locations (See: experiment N1). There were no bilateral simultaneous targets. The targets were presented for 500 ms (without an indicator, or mask). B.Q. was asked to point using her right hand to the target location. An inter trial delay of 800 ms was used. In the overlap condition the fixation point remained on throughout each trial and targets appeared 1000 ms after initial fixation onset. In the gap condition the fixation point was extinguished after 900 ms, and a delay of 100 ms occurred before target onset. There were 36 left and 36 right target trials in each condition.

B.Q. was instructed to verbally report the fixation cross when it appeared and to point (using her ipsilesional hand) to the location where a stimulus square was presented. B.Q. was encouraged to touch the screen with her finger at this location. The experimenter recorded the number of correct and incorrect responses. The criteria used for a correct response was that the area indicated by her pointing had to be within 1-2 cm of the exact target location. A practice block of twenty gap and overlap trials were given before the main experimental block.

6.4.3 Results.

Table 10 shows the number of left and right stimuli which B.Q. detected by pointing under the gap and overlap conditions. It was noticed that B.Q. often appeared to move her head towards left stimuli, but seemed unable to generate the appropriate hand/arm movement to produce the pointing response.

Table 10. The percentage of left and right targets that B.Q. pointed to under gap and overlap conditions.

	Left single	Right single
OVERLAP	13.8%	58%
GAP	36%	80.5%

The results of the manual pointing experiment show a similar pattern of results to those obtained from the overt digit reporting experiments (experiments N1 and N2). In the overlap condition B.Q. pointed to very few left stimuli (14%), but pointed to 50% of right stimuli. In the gap condition a 20% improvement is shown for the amount of left stimuli pointed to and an similar improved in the amount of right stimuli pointed to. A chi-square analysis was performed on the numbers of correct pointing responses which indicated that B.Q. has detected significantly more stimuli in the gap than overlap condition (Overlap v Gap: $Chi^2 = 6.18$ $p < 0.05$). B.Q. was shown to have pointed to significantly more left stimuli in the gap condition (overlap left v gap left $Chi^2 = 4.58$ $p < 0.05$), and also pointed to significantly more right stimuli in the gap condition (overlap right v gap right: $Chi^2 = 4.04$ $p < 0.05$).

6.4.4 Discussion.

The pattern of results in this experiment are similar to those obtained in the gap and overlap eye movement experiments (experiments N1 and N2). In the overlap condition B.Q. failed to point to 86% of left stimuli, but did point to over 50% of right stimuli. The use of a gap condition improved her ability to point to left stimuli and also increased the amount of right stimuli that she pointed towards The effect of prior fixation offset is to reduce the severity of contralateral neglect in this pointing task, as it did for overt indicator reporting and left saccadic eye movements.

The possibility that B.Q. failed to report the indicator stimuli in previous experiments because of a reduction in her information processing capacity, is weakened by her inability to point to stimuli in the overlap condition of the present experiment. The use of the gap condition again improved the amount of contralesional and ipsilesional stimuli that B.Q. pointed towards, which is consistent with the attentional disengagement explanation of her left neglect. The improvement in pointing towards ipsilesional stimuli in the gap condition, could be due to a warning signal effect that gives a generalised increase in her level of arousal. The increase in arousal would be expected to improve her attentional orienting and therefore her ability to make a manual pointing response to the stimuli. A second possibility that the deficit of attentional disengagement also affects B.Q.'s ability to orient attention ipsilesionally, but to a lesser extent than orienting attention contralesionally.

The results obtained from B.Q. on the pointing task are comparable to those from the overt eye movement experiments, so a similar attentional explanation could be applied to both deficits. The premotor model of Rizzolatti and Camarda (1987) suggested that spatial attention

is controlled by separate circuits depending on the required motor response and is consistent with these findings. Both the digit reporting task and the pointing task were performed in the patients personal grasping/reaching space, so a deficit of one motor response (oculomotor) could be expected to affect another motor response (pointing) that would normally operate at this distance. If the patient is impaired at disengaging attention to make a movement of attention to a stimulus a certain distance away, this could prevents a motor response being made in more than one modality. An impairment in making a saccade to a target located within peripersonal space, would also be expected to impair a manual pointing task.

Although the present experiment did not require an accurate eye movement to be made to the stimuli to enable a pointing response to be made, it is most likely that an eye movement would be made. The results obtained in previous Experiments (N1 and N2), showed that B.Q. is impaired at making a contralesional eye movement, when fixating on a central stimulus. Given that an eye movement would normally be expected to guide the pointing response, on the present experiment, B.Q.'s failure to point to contralesional stimuli may reflect the selective inability to make an oculomotor response.

The conclusion of the present experiment is that the gap effect reduces the severity of B.Q.'s contralesional neglect, due to attentional disengagement. Disengaging attention enables B.Q. to make an eye movement and a manual arm movement to be made to contralesional stimuli. This suggests that a similar model may be involved in orienting attention to a stimulus location which is involved in producing a motor response from more than one motor system.

In Experiments N1 and N2 it was noticed that B.Q. reported more ipsilesional stimuli on bilateral target presentation.. The final experiment in this chapter was performed to investigate this effect.

6.5 Experiments N4: a,b and c: A further investigation into B.Q.'s increased ipsilateral orienting with bilateral target presentation.

6.5.1 Introduction.

This short series of experiments was performed to examine B.Q.'s apparent increase in reporting right (ipsilesional) stimuli with bilateral target presentation.

An increase in ipsilesional orienting on bilateral target trials could be consistent with Karnath's (1988) three component model of neglect. Component A is a directionally specific bias of covert attentional orienting to the ipsilateral visual field. Component B is a directionally specific deficit of of covert orienting in the contralateral direction. Component C is a directionally non-specific deficit of information processing ability. Component A of Karnath's model could

predict that a stimulus presented in B.Q.'s left visual field may produce an orienting response to the right (ipsilesional) side. On bilateral target presentation there could be an automatic ipsilesional response that increases the amount of right stimuli reported, compared to the amount reported with single target presentation. There was some evidence to support this idea from the results of experiment N1, where B.Q. reported slightly more right stimuli with bilateral target presentation. This trend was evident in the overlap and zero-gap condition but was too small to reach significance and was not shown in the +100 gap condition. As this appears to be a weak effect it will require a large number of trials to show if there is indeed a bias of ipsilesional orienting with bilateral target presentation.

The possibility that B.Q. reported more right stimuli with bilateral simultaneous target presentation, was examined in experiment N4a, by repeating the basic overlap experiment (four times) over a period of three months. The aim was to show if B.Q. consistently reported more right stimuli with bilateral simultaneous target presentation than with single target presentation. In experiment N4b a larger LVF stimulus was presented *simultaneously* with the RVF target, to show if increasing the size of the contralateral stimulus increased the tendency to orient in the ipsilesional direction. In experiment N4c the LVF stimulus was presented 100 ms *before* the RVF stimulus. This was to show if the early onset of the left stimuli had a greater facilitation effect than does the simultaneous onset of the left stimulus. The early onset of the left target could give a further improvement in ipsilesional orienting due to prior attentional disengagement, before the onset of a RVF target.

6.5.2 Experiment N4a: Repeated blocks of single and bilateral targets.

6.5.2.1 Introduction.

To examine the possibility that B.Q. reported more bilateral stimuli with bilateral simultaneous target presentation, she was tested on four blocks of the basic overlap experiment over a three month period.

6.5.2.2 Method.

The apparatus and method were identical to those described for Experiment N1. The timing and presentation of the stimulus was as used in the overlap condition.

6.5.2.3 Results and Discussion.

The numbers of left and right stimuli reported with single and bilateral simultaneous target presentation in the three sessions are shown in Table 11.

Table 11. The percentage and frequency of left and right stimuli reported by B.Q. in Experiment N4a.

Session and date	left double	right double	left single	right single
N4a-1. 28/2/90	0 (0%)	36 (50%)	1 (2.7%)	14 (38.9%)
N4a-2. 4/4/90	2 (2.7)	38 (52.8%)	4 (11.1%)	15 (41.7%)
N4a-3. 10/4/90	0 (0%)	44 (61.1%)	0 (0%)	13 (36.1%)
N4a-4. 10/4/90	2 (2.7%)	43 (59.7%)	1 (2.7%)	18 (50%)
Totals (N4a1-4)	4/288	161/288	6/144	60/144
(Exp N1 20/6/90	**0 (0%)**	**38 (53%)**	**3.0 (8.3%)**	**18 (50%))**

Combining the results obtained over the four sessions shows that 41.6% of right stimuli were reported on single target trials and 55.9% were reported on bilateral trials. A chi-square analysis showed this increase to be significant ($chi^2 = 7.735$ $P<0.05$ 1 df).

This experiment has indicated that B.Q. shows a weak trend of reporting more right stimuli in bilateral presentation than under single target conditions. This suggests that a contralesional stimulus can facilitate ipsilesional orienting, as suggested by Karnath's (1988) three component model of extinction and neglect.

6.5.3 Experiment N4b: The effect of increasing the size of the contralesional bilateral target on B.Q.'s ipsilesional orienting.

6.5.3.1 Introduction

This experiment was designed to show if the improvement in reporting right stimuli shown on bilateral target trials, could be increased further by presenting a larger stimulus in the contralateral field.

6.5.3.2 Method.

In this experiment targets were presented on the horizontal axis only, at two eccentricities (4^o and 8^o), left and right of a continuously displayed fixation point. There were 120 trials consisting of; 60 right single targets; 60 bilateral trials with targets the same size appearing left and right of fixation; and 60 trials in which the left target (1^o by 1^o) was four times larger than the right target (0.5^o by 0.5^o). There were no left single targets as they were not required to investigate the affect of a LVF stimulus on ipsilateral orienting and also because B.Q. almost never responded to them on previous sessions. The timing of each trial was otherwise identical to that described for experiment N1 (overlap).

6.5.3.3 Results and Discussion.

The numbers of right stimuli reported by B.Q. with single right targets, and when either: a small (0.5°), or large (1°), left target appeared simultaneously with the onset of the right target, are shown in Table 12.

Table 12. Numbers of right indicators reported on single and bilateral trials.

	Single	Small LVF bilateral	Large LVF bilateral
Number	39/60	47/60	43/60
Percentage	65%	78%	72%

There is a 10% (one tailed = t-test 1 df= 3.95 n.s.) increase in the amount of RVF stimuli reported by B.Q. on bilateral trials compared to single stimulus trials, as was shown in experiment 4a. However, increasing the size of the LVF stimulus has not given any improvement in the amount of right stimuli reported by B.Q., infact, it can be seen that there was a small (non significant) decrease in the amount of right stimuli reported when a large left stimulus was used.

6.5.4 Experiment N4c: The effects of presenting the contralesional target 100 ms before the ipsilesional target, on B.Q.'s ipsilesional orienting.

6.5.4.1 Introduction

This experiment examined the effect of presenting a left (contralesional) target 100 ms before the right (ipsilesional) target, on B.Q.'s level of ipsilesional orienting. In terms of Karnath's (1988) model the prior onset of a left stimulus could trigger the automatic tendency to orient ipsilesionally (Component A) before the right stimulus onset which could improve the amount of right targets reported. The early onset of a target stimulus has been shown to reduce the latency of saccades made in the contralateral direction in normal subjects (Chapter 3: experiment 7.). This facilitation effect could be partly due to a warning signal effect that prepares the attentional/eye orienting system to make a response. The onset of a target in B.Q.'s neglected left visual field may also serve to produce the same warning signal effect and facilitate the production of a saccade to the right target.

6.5.4.2 Method.

Targets were presented on the horizontal and principal oblique axis, to the left and right of a constantly displayed fixation point (overlap), at three eccentricities, as described in experiment N1. On single target trials a target appeared 1000 ms after initial fixation onset, in either the left, or right, visual field. On bilateral target trials the LVF stimulus appears 1000 ms

after fixation onset and remained on for 100 ms, so that the offset of the LVF target was simultaneous with the onset of the RVF target. The fixation point remained on throughout each trial and the timing and procedure were otherwise identical to experiment N1. This experiment was carried out some six months after experiments N4a and N4b.

6.5.4.3 Results and Discussion.

The number (and percentages) of left and right stimuli reported by B.Q. with single target presentation and when the left stimulus appeared 100 ms before the right stimulus are displayed in Table 13.

Table 13. Numbers of indicators reported by B.Q. in experiment N4c.

	Left double	Right double	Left single	Right single
Number	0/72	34/72	4/36	17/36
Percentage	(0%)	(47%)	(11%)	(47%)

The results show that B.Q.has reported a similar amount of right stimuli on single and bilateral target trials, indicating the complete absence of the increase in orienting on bilateral target trials. There was no further improvement in B.Q.'s ipsilesional stimulus reporting when a LVF stimulus was presented 100 ms before the RVF stimulus has not increased the amount of ipsilateral orienting compared to the amount of right stimuli reported in unilateral trials.

There are two possibilities why the expected small increase in orienting with bilateral targets was not shown in this experiment. The first is simply that as this experiment was carried out some six months after those already described the ipsilateral orienting effect on bilateral target presentation was no longer apparent. Unfortunately, no blocks with bilateral simultaneous target presentation were carried out at this time, so it cannot be confirmed that the bias of orienting with bilateral simultaneous target onset was present at this time. The second possibility is that the early onset of the LVF stimulus has a small cueing effect directing attention towards the contralesional side prior to ipsilesional stimulus onset. Directing attention contralesionally could have the effect of a small reduction in ipsilesional orienting, thus eliminating the small advantage for RVF stimuli on bilateral trials.

6.5.5 Discussion of Experiments N4(a b c).

The results of repeating the overlap experiment four times in experiment N4a, confirmed that B.Q. showed a small, but consistent advantage, for reporting RVF stimuli with bilateral and simultaneous target onset. When a target appeared in the left simultaneously with the right target B.Q. was more likely to report the right target than when a single target was presented. This is consistent with Karnath's (1988) suggestion of an automatic bias of orienting in the ipsilesional direction, shown by neglect patients. The presence of a stimulus in the LVF could enable attention to be disengaged from the fixation point, so that a RVF stimulus will initiate an attentional/eye movement. Alternatively the LVF stimulus could be thought of as acting as a warning signal (Braun and Breitmeyer, 1990) which prepares the system to initiate a

saccade in the ipsilesional direction.

Experiment N4b showed that increasing the size of the left stimulus did not enhance the tendency to orient ipsilesionally on bilateral target trials. This could be because the effect is not dependent upon the size of the sensory event presented in the contralesional field, but may be due to appearance of any abrupt onset in the neglected field that triggers the ipsilesional orienting response. A further possibility is that doubling the size of the LVF target (to 1^{o}) was not sufficient to have any significant effect on ipsilesional orienting and a larger stimulus would be required to give any additional improvement in ipsilesional orienting. It would be instructive to examine the effects of various sizes of stimuli presented at various eccentricities away from fixation to examine the ipsilesional orienting effect of a LVF target.

In Experiment N4c B.Q. reported equal numbers of right stimuli with single target presentation and when the left bilateral was presented 100 ms before the right target. This could be because the experimental manipulation actually abolished the small tendency to orient ipsilesionally, or because the effect was no longer apparent. The ipsilesional advantage may not be apparent because the automatic ipsilesional orienting component (Karnath's component B) has shown a certain amount of recovery and was not in evidence on this testing session. As B.Q. was not tested with bilateral simultaneous targets on this session, it is not known if the ipsilesional advantage was present at this time. Karnath's model of neglect suggests that the bias of ipsilesional orienting (Component B) recovers later than does the inability to voluntarily orient attention contralesionally (Component A) which is important in explaining the recovery of neglect to the milder extinction effect on bilateral presentation. Given that B.Q. still showed profound contralateral neglect on single trials and never reported a left double target it would appear unlikely that there has been much recovery of components A or B. As the advantage for reporting RVF targets under bilateral stimulation was a very small effect it was not possible to study it further without performing controlled experiments which would require large numbers of trials and long testing sessions which were not a practical possibility. It is not possible to conclude if presenting a left target 100 ms before the right target reduces the tendency to orient ipsilesionally, or if B.Q.'s tendency to orient ipsilesionally had simply recovered.

6.6 General discussion.

B.Q., a patient with profound left neglect, does not report stimuli presented in the left side of space, when attending to a central fixation point. Removal of the fixation point 100 ms before, the onset of the target enables B.Q. to make a saccade to and report the left stimuli. The use of the 100 ms gap condition also improved the proportion of right stimuli reported by B.Q. in this task. However, when the the fixation point was removed simultaneously with the onset of the target, B.Q. again failed to report the left stimuli. The finding that B.Q. neglects left stimuli in the overlap condition, but is able to report left stimuli in the +100 gap condition provides strong evidence that she is not hemianopic. It has been noted that under standard

perimetric field plotting techniques it is difficult to decide if the failure of neglect patients to report left probes is due to a visual field defect or the higher order deficit of neglect (Heilman et al., 1987). The use of a situation which can reduce the severity of the patients neglect in this experiment has been shown to abolish the signs of a hemianopia.

The results of the gap/overlap experiments are consistent with Posner et al.'s (1984) attentional disengagement hypothesis of neglect. In the overlap condition the attentional system is engaged at fixation, when the target is presented. The inability to disengage attention has the effect of preventing a contralesional attentional/eye movement being made to the left stimuli. Fixation point offset in the zero-gap condition does not allow sufficient time for the attentional system to be fully disengaged and so there is no reduction in contralateral neglect. The failure of the zero-gap condition to reduce contralateral neglect is consistent with the findings of Rizzo and Hurtig (1992) in a comparable experiment. However, it appears that they were premature in rejecting the disengagement hypothesis on this basis, as the use of a +100 ms gap condition has produced a dramatic reduction in B.Q.'s left neglect. Experiments which have measured saccadic reaction times under gap/overlap conditions with normal subjects have shown that the maximum facilitation effect obtained occurs some 150-240 ms after fixation offset (Saslow, 1967; Reulen, 1984 ab). If attentional disengagement accounts for this reduction in SRT's as has been suggested (Fischer and Breitmeyer, 1987) then it appears that the process takes some time to be fully completed. This would explain why neglect is reduced with gap intervals of 100 ms, but not when fixation point offset is simultaneous with target onset.

Karnath's (1988) three component model of neglect is useful in explaining the results obtained from B.Q. Her inability to orient towards left stimuli is consistent with the idea of a deficit of voluntarily orienting attention to the contralesional side of space (component B). An automatic orienting towards the ipsilesional side (component A) can explain the increase of reporting right stimuli on bilateral presentation. Fischer's suggestion that disengagement has to occur before an attentional movement can occur is entirely compatible with Karnath's model. Given the link between the covert and overt orienting system as suggested by Rizzolatti et al. (1987) an impairment of attentional disengagement would be expected to prevent a saccadic eye movement being produced. Karnath's model is also useful in providing an explanation for the increase of right sided stimuli reported in the +100 gap condition. In the overlap condition B.Q., reported some 50% of targets and this could be due to the reduction in the patients information processing capacity (Karnath's component C) that reduces her overall level of performance on the tasks. There is much evidence to suggest that part of the neglect condition is due to such a nonlateralized attentional deficit (Robertson and Frasca, 1992) The increase observed in the +100 gap condition could reflect a simple warning signal effect which increases the patients arousal level, and therefore improves performance on the task. The failure to find an improvement in orienting in the zero-gap condition which should provide some warning signal, confirms that this is only be part of the explanation of neglect. A second possibility as to why the 100 ms gap also improves ipsilesional orienting is that the patient's representation of space has shifted ipsilesionally (Ventre et al., 1984). If this was the case then stimuli located to

the right of fixation could also fall within the part of space (with respect to the body midline) in which the impairment of contralateral orienting is observed (Karnath et al., 1991). Disengagement of attention by fixation point offset in the +100 gap condition would therefore be expected to improve orienting to right and left sided stimuli by a similar mechanism.

The saccade records obtained in Experiment N2, confirmed that in the overlap condition B.Q. typically failed to initiate a contralesional saccade and maintained her gaze on the fixation point. When a saccade was produced it was of slow latency compared to the latency of saccades made to right sided stimuli. These late latency left saccades were also of small amplitude and B.Q. did not report the left stimuli on these trials. The use of the +100 gap enabled B.Q. to produce a normal amplitude and latency left saccade. On some trials in the 100 ms gap condition a normal left saccade was made but the left stimulus was still not reported. This implies that B.Q. occasionally fails to report left stimuli that have been saccaded to, which could reflect an extinction effect. However, there was also some evidence of right stimuli not being reported on trials where a normal amplitude and latency saccade had occurred. This suggests that a large part of the failure to report the digit could be due to a general reduction in information processing capacity. The 100 ms gap condition did not speed the latency of saccades made to right stimuli. The absence of a speeding effect under gap conditions, is in contrast to the performance of normal subjects. However, similar experiments are needed with an age matched control group, before many conclusions can be drawn from this lack of facilitation with B.Q. Parietal lobe lesions in monkeys have been shown to result in a slowing of saccade latency which it has been suggested is due to a mild impairment to part of the system involved in controlling saccadic eye movements (Lynch and McLaren, 1989). Hyvärinen (1982) suggested that the role of the posterior parietal cortex is to interrupt active fixation when an interesting stimulus appears in the periphery. This interruption of fixation is consistent with the notion of attentional disengagement from fixation.

The results of the experiments described with normal subjects (in Chapter two) showed that saccade latency was slowed by some 20 - 30 ms with bilateral simultaneous target presentation (Lévy-Schoen, 1969, 1974; Findlay, 1983). This slowing was found to occur in both the gap and overlap conditions (See: Chapter 2), and could involve a different process to that responsible for the gap effect. It has been shown that bilateral simultaneous target presentation produces a slowing of saccade latency with hemianopic patients (Rafal et al. 1990). In contrast B.Q.'s saccade latency was <u>not</u> affected by bilateral target presentation. It was suggested in Chapter two, that this increase in saccade latency reflects an increase of bilateral inhibition acting on the attentional/eye movement system. This inhibitory component is *crossed* so that a stimulus presented in the left will produce inhibition in the component which moves attention/eyes right. It is possible that there is a loss of the inhibitory connection acting on the right move component. The loss of this inhibitory connection acting on the ipsilesional movement component would explain the lack of the expected increase on saccade latency with bilateral target presentation and could also explain the bias of orienting in the ipsilesional direction (Karnath's component A).

The idea that neglect could result from a loss of inhibition acting on the ipsilesional

movement component is discussed in more detail in Chapter eight, in relation to the proposed attentional model. The finding of the experiments in this chapter that neglect can be reduced by a gap condition is also described in more detail in relation to this model.

The next chapter provides an experimental examination into the left sided word omissions made by B.Q. when reading text. The aim was to examine the possibility that left sided word omissions could reflect B.Q.'s attentional impairment that prevents her making a large left saccade to locate the left side of a page of text. An inability to make a large left saccade during reading would result in left sided word omissions.

Chapter 7

Visual attention and whole word omissions shown by a neglect patient when reading text.

7.1 Introduction.

In this chapter a series of experiments are described, which provide an investigation into the whole word omissions made by B.Q. when reading text. B.Q. is of particular interest in that her reading ability is good and she rarely shows any single word 'neglect dyslexic' errors. However, when reading text B.Q. omits large numbers of words located on the left side of the page. This dissociation between patients who show intact single word reading, but are impaired at reading text, and those that can read text, but make errors when reading single words; has been used as evidence that the two errors may have a different underlying cause (Ellis et al., 1987; Riddoch and Humphreys, 1991; Young et al., 1991). When reading a passage of text, a series of attentive fixations and saccades are required to orient along the line, from left to right. In contrast the recognition of single words (of short and medium length) requires a single fixation of attention on each word, to enable processing of the letter string. The errors shown by neglect patients when reading single words has been quite extensively studied, while left sided word omissions have largely been ignored.

It has been suggested that the omission of words located on the left side of a page, could result from an inability for the patient to make a left saccade to locate the start of subsequent lines (Riddoch and Humphreys, 1991; Young et al., 1991). Riddoch and Humphreys (1991) further suggested that the failure to orient into contralesional space during text reading, could arise due to a failure to detect the left boundary of text, as defined by the margin and first words of each line. The failure of a preattentive system which normally signals the presence of the left text/margin break could results in the return left saccade not being directed far enough to locate the start of the next line. Some support for this view is provided by patients who show left sided word omissions when reading text presented horizontally, but who can read the whole passage when it is presented vertically, with each line being written from the top to the bottom of the page (Ellis et al., 1987). Presentation of text in vertical orientation means that a left saccade is not required to locate the start of each line.

Karnath and Huber (1992) recorded the eye movements of a neglect patient (H.S.) while reading text. H.S. showed left neglect on a range of tasks, but did not have any sign of a visual field defect, tended to omit the words on the left side of a page of text. The patterns of eye movements made by H.S. during text reading were recorded, to investigate the basis of these left sided word omissions. The eye movement records showed that H.S. typically failed to locate the start of the very first line (of text), and then read the remainder of that line with little difficulty. A normal pattern of left-right scanning, separated by fixations on single words, were made along the line, but the large return left saccade typically fell short, landing close to the middle of the next line. The length of these return sweeps was shown to be correlated with the length of that line, indicating that the saccades usually fell towards the midpoint of the line.

H.S.'s problem in making a large left saccade did not result from an impairment of the oculomotor system, as he could make a left saccade to the left side of the screen when asked to do so.

The eye movement record revealed evidence that H.S. used a top-down strategy to search for a linguistic plausible continuation with the last line. This was indicated by the presence of small amplitude left saccades made after the initial return saccade, which landed close to the midpoint of that line. H.S. appeared to be searching for a linguistically plausible continuation to the last line read. This search for a plausible continuation appeared to be confirmed as significantly fewer omissions were made which violated the linguistic continuation of the text, than omissions which did violate the linguistic continuation. It appears that H.S. compensated for the short return left saccades to some degree, by using a top-down strategy of backward reading. There is no indication of a similar strategy being used by B.Q. who appears content to read text without any effort to find a plausible linguistic or semantic continuation.

Karnath (1992) pointed out that the spatial border which separates the normal from abnormal left return saccade, cannot operate in terms of H.S.'s left and right visual fields. This is because the return saccade is made from the last word of a line located in the right visual field. Karnath suggested that the reference frame involved is actually defined in terms of the patients body trunk midline and not the retinal midline; as has been suggested previously (Karnath et al., 1991). The programming of the leftward eye movement is constrained by the midline of the projection screen.

Training neglect patients to direct their eyes to a left sided cue has been shown to be effective at reducing word omissions. Weinberg et al. (1977) used a training package with neglect patients, which involved several elements including training on paragraph reading. The training procedure involved presenting a passage which had a vertical line on the left as an anchoring point and numbers at the beginning and end of each line. Initially the patient was asked to look at the vertical line, then report the number and read out the line. The number at the end of the line is then read and this serves to ensure that lines are not skipped. These cues were withdrawn in sequence starting with the right number cue, until only the left anchoring line remained, and finally no cues were provided. This procedure was shown to reduce the amount of left sided word omissions made by the experimental group, while the control group did not show a significant improvement. The greatest improvement was shown for patients who showed the greatest severity of neglect on other measures. It appears that training neglect patients to scan leftwards can reduce the amount of left sided neglect and left sided word omissions.

The following chapter examines the hypothesis that B.Q.'s whole word neglect dyslexia could result from her inability to produce a large left saccade due to a deficit of visual attention. Our previous experiments (Chapter six) have shown that B.Q. has a selective impairment at making a left saccade, resulting from her inability to disengage attention. This deficit in

disengagement was shown to impair B.Q.'s ability to make saccades to left stimuli, but did not effect her ability to make saccades to right stimuli. This deficit in attentional disengagement could also be involved in B.Q.'s left sided word omissions when reading text. It appears that B.Q. is able to produce a normal pattern of fixations and saccades, moving from left to right, along the line (see: Figure 24, and following eye movement section); but once the last word of a line is reached, she cannot make a large contralesional saccade to locate the start of the next line. Karnath et al.'s (1991) suggestion is that it is the words located to the left side of the patients body midline, rather than suggesting that they are neglecting words located in the contralesional hemifield. Our previous experiments (Chapter six.) showed that B.Q.'s ability to make a left saccade was improved if the current fixation point was removed prior to target onset. These experiments used a situation in which the body/head midline corresponded with the retinal midline, so it is not possible to establish if her deficit involved spatial, or retinal, coordinates.

The first section of this chapter describes an attempt at recording B.Q.'s eye movements made while reading a passage of text. The following sections describe experimental manipulations performed to examine the underlying cause of B.Q.'s left sided word omissions. Experiment N6 examined the possibility that B.Q. cannot produce a large left saccade to locate the start of the next line, due to a failure of disengaging attention from the last words of the line. This hypothesis was tested by measuring the frequency of word omissions made by B.Q., under conditions in which attentional disengagement was manipulated. Attentional disengagement was achieved by presenting passages of text one line at a time onto a VDU screen and then removing each line as B.Q. read out the last word from that line. The next line was presented on the screen after a time delay ('gap' interval) which should enable attention to be disengaged. A baseline measure of B.Q.'s word omissions was obtained in a control condition, in which B.Q. read a comparable passages of text, with all of the lines displayed together on the VDU screen.

The second condition of Experiment N6 used a stimulus flash to automatically cue attention to the left side of the screen, to facilitate the production of the return left saccade. The stimulus flash was presented at the left side of the screen during gap intervals (of either; 800 or 1200 ms), while the screen was **blank**. The stimulus was presented at the left side of the screen, which would normally be indicated by the left text/margin boundary of a whole passage of text. It was hypothesised that the stimulus flash should serve to automatically attract attention to the far left location, which should enable a left saccade to be made further into the contralesional side of space and reduce the amount of left sided word omissions.

The strength of the cueing effect of the stimulus flash was examined in a second experiment. In this experiment the stimulus flash was presented at the left side of the screen, while the line of text was also on the screen. It was hypothesised that the attentional cueing effect of the contralesional flash would not be as great in this manipulation, as the cueing effect of the stimulus flash has to compete with disengaging attention from the stimuli (words) visible on the screen. The amount of word omissions made when reading single lines with a stimulus

flash was compared to the amount made when lines were presented without a stimulus flash.

7.2 B.Q.'s normal reading performance.

The following section aims to describe B.Q.'s reading of single words, and passages of text. The evidence shows little evidence of neglect when reading single words, while large numbers of words are omitted from the left side of a page of text.

7.2.1 B.Q.'s single word reading.

B.Q. made very few neglect dyslexia errors when reading single words (on the criteria of Ellis et al., 1987*) and appeared to have little difficulty in reading and understanding these words. Table 14, shows that B.Q. made two errors neither of which would be classified as neglect dyslexic errors, when reading a list of 18 words 4, 5 and 6 letters long (printed on A4 paper, upper case, 14 Pt. Helvetica). WIFE was read as 'wiff' by spelling out the letters, and TREAD was read as 'thread'. This suggests the B.Q.'s word recognition system is relatively intact enabling the correct recognition of single words. B.Q. was then shown the same list of words printed as if viewed in a mirror; so that they had to be read from right to left. B.Q. found reading these words very difficult (8/18 correct) as indicated by her slow and laborious attempts at spelling out each letter from right to left; while repeating letters. B.Q. often became confused as to which letter had been read and repeated letters more than once (eg. KCOLB 'bloocco'), or on reaching the end of the word started to read it again from right to left (MRAF 'farr'- 'mraf'). The difficulty in reading mirror reversed words, suggests that B.Q. is severely impaired at moving attention from right to left even within a single word. This could produce a problem with making movements of attention within a word, which would be required if the eyes fall to the right of the optimal viewing position of that word, as Riddoch and Humphreys (1991) suggested could be the case with neglect patients. An impairment in making these regressive attentive movements would result in some errors when reading single words, which would be increasingly evident with longer words.

Table 14 Illustrations of B.Q.'s single word reading

Word	B.Q.'s response	Word	B.Q.'s response
WHIP	'whip'	BLIND	'blind'
PLANET	'planet'	BLOCK	'block'
GLOVE	'glove'	HANDLE	'handle'
TREAT	'treat'	BOLT	'bolt'
LETTER	'letter'	PLAYER	'player'
LEAVES	'leaves'	BROW	'brow'
WIFE	'w.i.f.f.'	FANG	'fang'
GRASS	'grass'	HANGER	'hanger'
FARM	'farm'	TREAD	'thread' or 'read'

* The criteria used defined an incorrect response as being a neglect dyslexic error when: the letters to the right of a 'neglect point' are identical to those of the target word, but letters to the left of the neglect point are not the same as those of the target word.

B.Q.'s single word reading has been examined in greater detail by Young et al. (1991). When reading a list of 57 words for which the deletion, or substitution, of the initial letter could leave a real word alternative (CLOVE-LOVE or GLOVE) she made only one error. B.Q.'s performance on reading compound words which were physically contiguous (EGGCUP) or non-contiguous (EGG CUP) was error free and when reading contiguous and non-contiguous scrambled compound words (TIPCAP : TIP CAP) she made only one error. It appears that B.Q. is able to use left sided information in the recognition of single words. However, B.Q. did produce neglect of the initial letters with non-words. Pronounceable letter strings produced 5/30 initial letter errors, and unpronounceable letter strings produced 9/30 initial letter substitution and deletions. Increasing the length of the letter string increased the amount of neglect (4-letter string 0/10 errors, 5-letter string 3/10, 6-letter string 6/10). When reading numbers B.Q. omitted the initial figures and the number of omissions increased with increasing length of the number.

B.Q.'s ability to read single words correctly, whilst being impaired on non-words and strings of letters and numbers is explained by Young et al. (1991), in terms of Behrmann et al.'s (1990) model of neglect dyslexia. B.Q. is thought to have a deficit of early visual processing, which means that left sided features of stimuli are not fully detected, but the presence of the initial letters is encoded. This deficit is unevenly distributed as a gradient from left to right, with left features being most impaired. The impairment of early processing is indicated by the effect of increasing left neglect when the length of the letter and number strings. B.Q.'s representation of words is thought to be intact, which can activate top down processes to overcome any impairment of left sided letter information. This top down knowledge is not available for non words and numbers which accounts for the lexicality effect of words being neglected less than non-words.

7.2.2 B.Q.'s passage reading.

An example of B.Q.'s impaired text reading performance is shown in Figure 24. In contrast to her preserved ability at reading single words, B.Q. has neglected a large proportion of left sided words from this passage. The text was presented on A4 paper printed in upper case (Helvetica 14 pt.) with unlimited reading time.

B.Q. read the passage at a normal reading pace and appeared to be content to read something which (presumably) could not make sense to her. There was no evidence of an attempt being made to look for a plausible continuation with the last line read as was shown by Karnath and Huber's patient, H.S. Almost all of the omissions are for words located on the left side of the page. Interestingly, B.Q. read the first line from start to finish and then read the last three or four words of each subsequent line. The omission of words located on the left side of the page, is consistent with the idea that B.Q. fails to make an eye movement large enough to locate the start of subsequent lines. Occasionally B.Q. omits words on the right side of the page, even though the words on either side of the one omitted have been read. This is consistent with the performance of normal readers who skip predictable words in text (Balota et

al. 1985), possibly coupled with B.Q.'s additional inability to produce a regression saccade back along the line, to locate the skipped word, due to an attentional deficit.

The convention of Ellis et al. (1987) is used where a # indicates the words read correctly and a line indicates the words omitted.

```
   #      #       #     # #     #    #  #     #
SWEDEN TODAY STEPPED UP ITS GUARD ON THE RUSSIAN
________ __________ __   #     #  #       #       #
SUBMARINE STRANDED ON ROCKS NEAR ITS SENSITIVE NAVAL
_____ ___        #           #      #      #   #     #
BASE. THE GOVERNMENT BELIEVES THAT SOVIET WAR SHIPS
_______ _______ ________ _____  #    #  #     #   ____
WAITING OUTSIDE SWEDISH WATERS MAY TRY TO SNATCH THE
_________ ______ ___     #       # #   #
SUBMARINE WHICH RAN AGROUND ON A SPYING
_______ _______ _____ __       #      #      #
MISSION. OFFICERS FROM THE SWEDISH NAVY HAVE
_______ ___ _________ __ ___ __      #      #      #
BOARDED THE SUBMARINE TO TRY TO CONVINCE THE RUSSIAN
_______ __ _____ ___          #       #  #       #
CAPTAIN TO LEAVE FOR INTERROGATION BY THE AUTHORITIES.
```

Figure 24. B.Q.'s performance when reading a passage of text.

7.3 Experiment N5: Recording B.Q.'s eye movements during reading.

7.3.1 Introduction

In this experiment an attempt was made to record B.Q.'s eye movements, while she was reading a passage of text, displayed on a VDU screen. The aim was to examine the patterns of eye movements, to see if she produced normal saccades left to right, along the line. It was also hoped to show if there was any evidence of B.Q. making a large left saccade to try to locate the start of the next line.

7.3.2 Method.

Apparatus and Procedure.

The apparatus was similar to that used previously to record B.Q.'s eye movements (Experiment N2). In this session the eye movements were recorded using a 'Skalar IRIS' binocular infrared system, with corrective lenses attached. A chin rest was used to limit the amount of head movements made. A calibration routine (also described in Chapter six) was performed prior to the text reading. The analogue signal was recorded on a Tinberg (series 100)

tape recorder for later off line sampling, using an Apple Macintosh II computer, interfaced with a Labdriver. One channel of the tape recorded the eye movement signal and one channel recorded B.Q.'s reading of the passage.

The passage of text was displayed on the VDU screen. The words were generated by a BBC microcomputer; using the mode 1, upper case, character set. The passage was taken from 'The Forest People' and was made up of 13 lines, containing five, six or seven words in each line.

7.3.3 Results.

Throughout the recording session B.Q. often moved her head completely off the chin rest and made many large head movements. Repeated attempts to remind her to keep her chin on the rest, did not reduce this problem. The final eye movement recording was therefore confounded with constant changes in the head position. The record could not be used in relation to the calibration record to provide an accurate interpretation of B.Q.'s eye position. The record also contained a large number of blinks which made it difficult to examine the patterns of saccades being made. It was possible to analyse the eye movement record with respect to the voice channel, which provided some indication of which part of the text each part of the eye record corresponded to. This crude analysis provided some evidence of small saccades being made left to right, along the last words of the line. The record did not show if B.Q. made a left saccade to try to locate the start of the next line. Observation of B.Q.'s behaviour implied that the left movement was probably performed by both eye and head movements. Given the problems in interpreting the record no further analysis was performed.

7.3.4 Discussion.

The attempt to record B.Q.'s eye movements while reading text was largely unsuccessful. B.Q. failed to use the chin rest for longer than a few seconds, even when being reminded to do so and it was not thought to be appropriate to use a bite bar, or any further head restraint to reduce this problem. This inability to maintain an instruction over time, could well reflect a reduction in B.Q.'s generalised level of vigilance/arousal, since the previous eye movement recordings described in Chapter six were performed. The eye movement record obtained, did show some evidence that B.Q. made small saccades rightwards along the line she was reading. This finding is consistent with the previous work with B.Q. that showed that she can make right saccades, but is impaired at making left saccades during active fixation.

7.4 An experimental examination into B.Q.'s left sided word omissions.

7.4.1 General Introduction

The following section describes experiments which manipulated the presentation of passages of text displayed on the VDU screen without recording eye movements. The underlying rational of these experiments was that the left sided word omissions result from B.Q.'s failure to make a large left saccade to locate the start of each line. The failure to produce the large left saccade could be due to a deficit of B.Q.'s attentional orienting system. The experimental manipulations in Experiment N6 were designed to: **disengage attention**; and **cue attention** contralesionally while it was disengaged, while reading lines of text. In Experiment N7 it was aimed to **cue attention** contralesioanlly without **disengagement**, while reading lines of text.

In the first manipulation of Experiment N6 (**Gap condition**) a gap interval was used to disengage attention by presenting passage of text one line at a time onto the VDU screen, a gap interval between each line presentation. The thirteen lines from each of the five experimental passages were presented one at a time onto a VDU screen. As B.Q. read out the last word from the line the experimenter initiated its offset by pressing a key to clear the screen. There then followed a temporal interval (gap) during which time the screen remained blank, before the next line appeared on the screen. A control condition was used in which B.Q. read whole passages of text (**Control condition**) to provide a baseline measure of the frequency of word omissions. The hypothesis was that longer gap intervals could reduce the amount of left sided word omissions by enabling attentional disengagement, so that a large left saccade could be made.

In the second manipulation of Experiment N6 (**Gap-flash condition**) a stimulus flash was used to cue attention to the left side of the screen. The stimulus was located to the left of the first word of the next line. It was thought that the stimulus flash should cue attention to the left location, which should facilitate the production of a left return saccade. An increase in the size of the left saccade, should reduce the amount of left sided word omissions made by B.Q.. The amount of word omissions made with the stimulus flash, was compared to the amount made when a comparable gap interval was used without a stimulus flash.

In Experiment N7 (**Line-flash**) a stimulus flash was presented while the line was also present on the screen. This was to examine the strength of the cueing effect of the stimulus flash. In this instance the stimulus flash was used to cue attention to the contralesional location in the presence of competing stimuli (words of the line). It was thought that the stimulus flash would be less effective at cueing attention if attention remains engaged on the words of the line. The use of the stimulus flash was not expected to reduce the amount of word omissions made by B.Q. in this condition, due to her problem of disengaging attention.

7.4.2 Experiment N6a and b: Disengaging and cueing attention during text reading.

7.4.2.1 Introduction

The **Gap condition** examined the hypothesis that B.Q. neglects words located on the left side of the page due to an inability to disengage attention from the ipsilesional end of the line. The inability to disengage attention resulting in a failure to produce a large left saccade to locate the start of the next line. Passages were presented one line at a time onto the VDU screen. A gap intervals of:- 0, 400, 800 or 1200 ms occurred before the onset of the next line, during which time the screen remained blank before the onset of the next line. It was expected that the 0 gap condition would not enable attentional disengagement so that B.Q. would make many left sided word omissions. The use of the longer gap intervals (400, 800 and 1200 ms) should enable attention to be disengaged so that a left saccade could be made to locate the start of the next line and fewer word omissions should be made.

The **Gap-Flash** condition examined the hypothesis that cueing attention to the far left location during the gap interval, should give a further reduction in left sided word omissions. The cue should automatically attract attention and facilitate the production of a large left saccade to the left side of the screen. The production of a larger left saccade will be expected to produce a further decrease in left sided word omissions.

7.4.2.2 Method.

Apparatus.

A BBC microcomputer displayed the passages onto a VDU screen (as described in the previous section). A tape recorder was used to record B.Q. reading each of the passages.

Format of the passages of text.

The passages were taken from 'The Forest People' the original passages were modified when necessary, so that each passage conformed to a set format. There were thirteen lines in each passage, each line containing five, six or seven words, with a total of 78 words in each passage (See: appendix 2). Each line, in each of the passages, contained the same number of words; for example the first line of each passage contained five words, each second line six words, etc. The passages were randomly assigned to either the control, or Gap, or Gap-Flash conditions.

Display sequence: Gap condition.

The timing sequence of single line presentations in the **Gap condition**, is shown in

Figure 25a. As B.Q. read out the last word from a line, the experimenter initiated that line offset by pressing a key to clear the screen. The next line appeared following a fixed delay (0, 400, 800, or 1200 ms) timed from the offset of the line, until the onset of the next line. Each line was presented at a progressively lower screen position (two screen tabs), so that a similar pattern of left, right, and down saccadic eye movements would be required, as when reading a normal passage of text. Only one time interval was used for each individual block of 13 lines. The comparison was between the amount of words omitted for each of the gap intervals used in the line-gap conditions and also the amount omitted in the control (Text) condition.

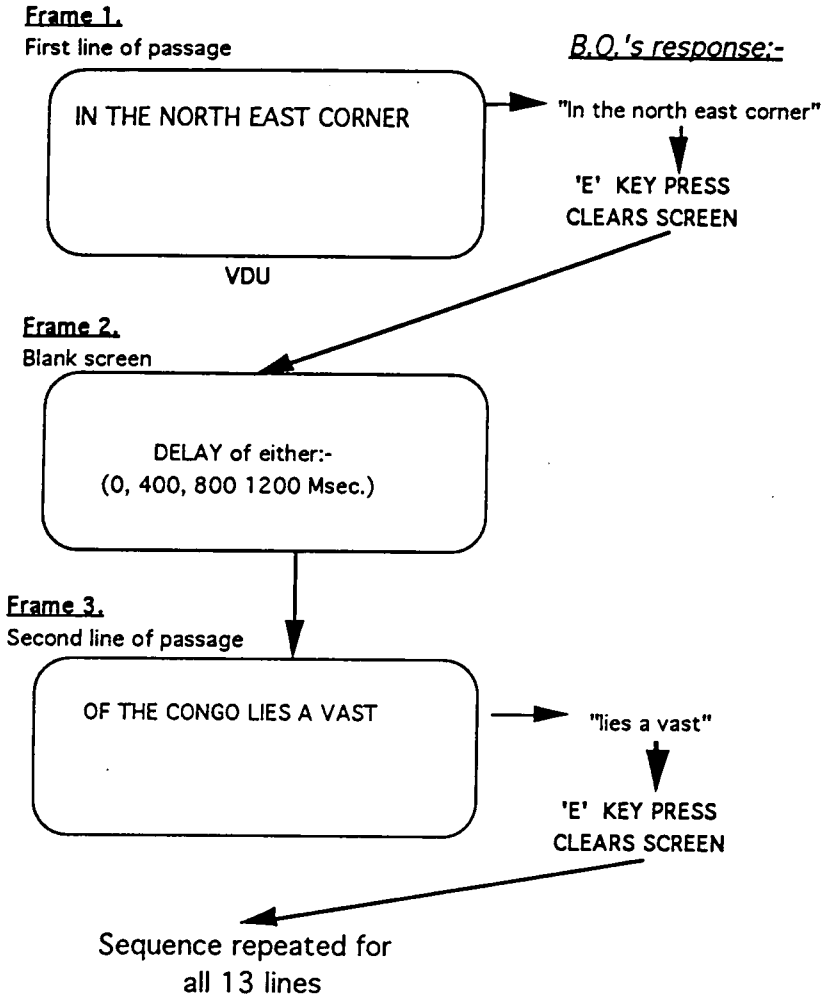


Figure 25a
Sequence of frames in 'Gap' condition
of Experiment N6.

Display sequence: Gap-flash condition.

Figure 25b. shows the presentation sequence of lines in the **Gap-flash condition.** In this condition a stimulus square was flashed (at the location of the first word of the next line) during a gap interval of 800 or 1200 ms. The stimulus appeared immediately after the experimenter pressed a key which cleared the last line of text from the screen. The stimulus

was a square (sides 0.5 degrees), presented ten times for 40 ms., with a delay of 40 ms between each presentation. This gave a total stimulus presentation time of 800 ms, in both the 800 and 1200 gap-flash conditions. In the 1200 gap condition the screen remained blank for an additional 400 ms after the last flash offset. The comparison was between the amount of words read when a stimulus flash was used as to the amount made using the same gap interval between line presentation without a flash (from condition 1).

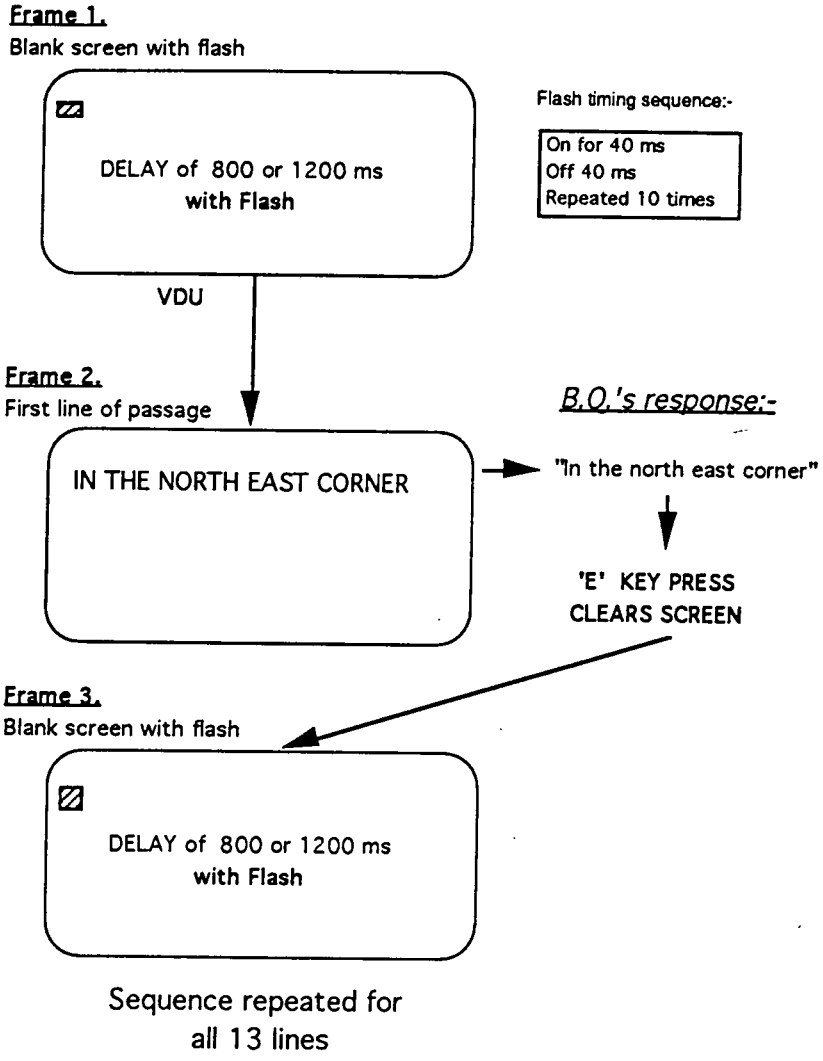


Figure 25b
Sequence of frames in 'Gap-flash' condition of Experiment N6.

Procedure.

The VDU monitor was placed at a distance of 50 cm, with the centre of the screen at eye level. The centre line of the VDU screen was aligned with B.Q.'s body/head centre line. Each testing session was taped to enable later examination of the reading performance. B.Q. was asked to read five passages in the control condition and five passages in each of the gap and gap-flash conditions, giving a total of thirty five passages. Testing was carried out in two

separate sessions (13/11/91 and 18/3/92). The order of presentation of the thirty five passages, was randomised in each testing session. The scoring procedure was a count of the numbers of words that B.Q. read from each passage in each experimental condition. The first analysis compared the amount of words B.Q. read from the passages in each condition, out of the maximum number possible. A second analysis compared the amount of words B.Q. read from the passages in each condition, out of the amount of words displayed in from left and right sides of the screen.

7.4.2.3 Results.

Results: Control Condition.

The following section describes B.Q.'s left sided word omissions made when reading whole passages of text displayed onto the VDU screen. This provides a baseline measure of B.Q.'s word omissions, made from the experimental passages, with VDU presentation of text. B.Q. completed four out of the five control passages, but became emotional during the reading of one passage, which was not included in the following analysis. The amount of words that B.Q. read from each of the four control passages is shown in Table 15.

Table 15. The amount of words read from each control passage in Experiment N6.

	Passage 1	Passage 2.	Passage 3.	Passage 4.	Total.
Number of words read*.	20/60	30/64	23/66	20/73	93/263
(Percentage)	(33.3)	(46.9)	(34.8)	(27.4)	[35.4]

Overall, B.Q. read 35.4 % of words from the four passages of text. This is less than the amount of words read in the passage shown in Figure 24 (58%). The increase in B.Q.'s word omissions during the experimental session could have more than one plausible explanation. Firstly, the experimental sessions were carried out some months after the reading of the passage shown in Figure 24. A degree of variability in the patients level of general arousal/concentration level, which affects the level of neglect, could be expected on different testing sessions. A second factor is that the passages were presented by very different methods (A4 paper vs. VDU screen) and taken from different sources. The passage in Figure 24. was originally a newspaper article ('The Chronicles of 20th. century') and could be easier to read and of more general interest than the experimental passages which were taken from a different source ('The Forest People'). The variability in the complexity of the text, level of neglect/arousal and the method of presentation could all be involved in the variability in word omissions made in the experimental passages and in that shown in Figure 24.

The observation that B.Q. often reads more words from the first line of a passage of text than from the subsequent lines is shown in the control passage results. This trend was examined by showing the amount of words that B.Q. read from each of the thirteen lines, from

the four control passages. Figure 26 shows the percentage of words read by B.Q. from each line of the passages.

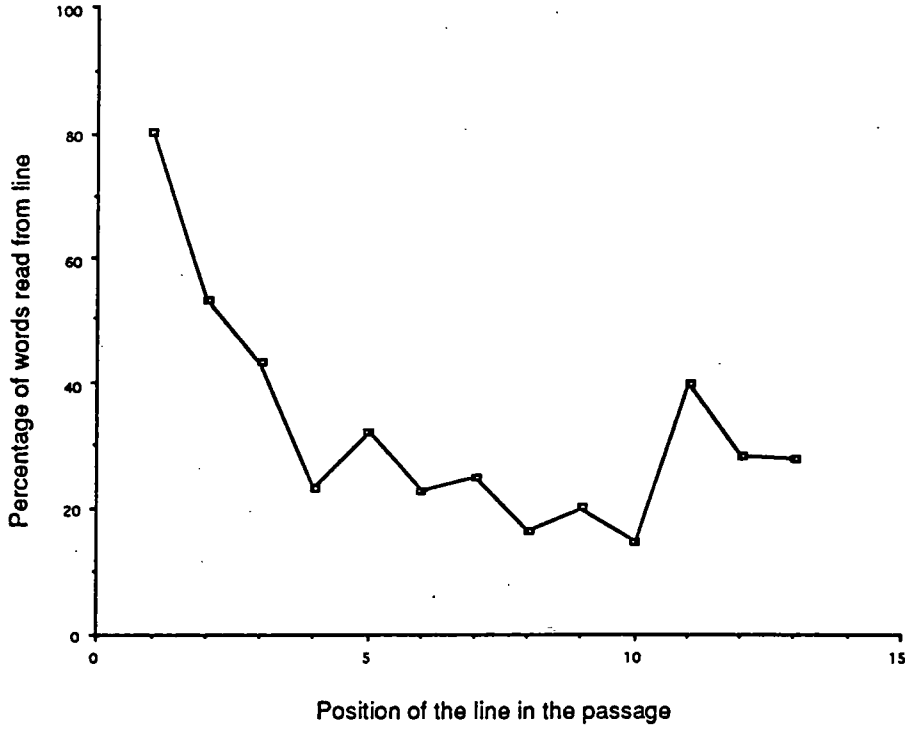


Figure 26.
The percentages of words read from each line from the four control passages in Experiment N6.

B.Q. read 80% of words from the first lines, but this decreased to 53% of words from the second lines. B.Q. then read some 20-40% of words from the remaining 11 lines of these passages. It was not possible to perform any statistical analysis on this data as some lines have missing data. However, the trend shown confirms that B.Q. shows less neglect for the first line from a passage and then neglects over half of the words from the left side of the remaining lines.

Results: Gap procedure.

B.Q. read four out of the five passages in the 800 ms gap condition but became emotional during the reading of one passage which was not completed. During the experiment B.Q.'s concentration occasionally lapsed while reading a line, causing her to stop reading and look away from the VDU screen. When this occurred she was directed to continue reading from the line she stopped at, and that line was not included in the later analysis. This controlled for any improvement in reading when she was directed to continue reading. The number of words read/omitted was then examined in terms of the total number of words read in each condition and the amount of words read from the left and right sides of the screen for each condition. It should be noted that omissions for words in the left and right side of the screen are not the same as omissions for words falling in the left and right visual fields, as the location of fixation moves rightwards along the line while reading.

Table 16. shows the total number of words read (with percentages) by B.Q. when reading blocks of single lines presented with gap intervals of 0, 400, 800 and 1200 ms, between each line presentation. The amount of words read from the four experimental passages is also shown for a comparison.

Table 16. The number of words read by B.Q. from the control passages and with single line presentation in the Gap conditions.

	Control passage	0 gap	400 gap	800 gap	1200 gap
Number of words read*.	93/263	168/372	160/366	136/282	197/374
(Percentage)	(35.4)	(45.0)	(43.7)	(48.2)	(52.7)

* The number of words read is given out of the maximum number possible. The total number of words which could have been read varies between the conditions. This is due to a loss of concentration during testing where whole lines were sometimes skipped and because two passages were not included due to B.Q. becoming emotional during testing (see text).

The presentation of single lines in the 0 gap condition reduced the amount of words B.Q. omitted compared to reading the experimental passages by some 10% (passages =35.4%: 0 gap =45%: Chi^2 1df= 10.28 $p<0.01$)). B.Q. omitted a similar amount of words from the passages in the 400 or 800 ms gap conditions, but a significant reduction in word omissions was shown in the 1200 ms gap condition. Chi-square comparisons confirmed that there was no difference in the number of word omissions made in the 0, 400 and 800 gap conditions. The 1200 ms gap condition produced a significant reduction in the number of words omitted when compared to the 0 gap condition only (chi^2 1df = 4.2 $p<0.05$).

The passages were re-scored to show the amount of words read from the left and right sides of the VDU screen, which is shown in Table 17. This division of left and right sides of space corresponds roughly to the patients head/body midline.

Table 17. The amount of words read from the left and right sides of the VDU screen in the Gap condition.

	Passages		0 gap		400 gap		800 gap		1200 gap	
Side of screen	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right
Percentage of words read*.	17%	65%	24%	81%	18%	85%	24%	89%	34%	87%

Table 17 shows that B.Q. omitted word from both the left, and right, side of the screen. However, many more omissions were for the words located on the left side of the screen. In the 0 gap condition 24% of words to the left of centre were read and 81% of words on the right side. This is consistent with B.Q. having read the last two-three words, from the right end of each line and then making a contralesional saccade, which fell close to the centre of the screen. The use of temporal gap condition can be seen to have increased the amount of words that B.Q. read

from the left side of the screen. A chi-square comparison confirmed that more left sided words were read in the 1200 gap condition than in the other three conditions (1200 vs. 0 gap 1df = 6.01 $p < 0.05$: 1200 vs. 400 gap 1df = 15.69 $p<0.01$: 1200 vs. 800 gap 1df = 4.65 $p < 0.05$). The amount of words read from the right side of the screen was found to be comparable in all cases.

Results: Gap-flash procedure.

Table 18 shows the numbers (and percentages) of words read by B.Q. when a stimulus square was flashed at the left side of the screen during gap intervals of 800 and 1200 ms. The amount of words read when the same gap intervals were used without a stimulus flash (from Table 16) are also shown.

Table 18. The amount of word omissions made in the gap-flash condition.

	800 gap	800 gap+flash	1200 gap	1200 gap+flash
Number words read	136/282	92/149	197/374	88/137
(Percentage)	(48.2)	(61.7)	(52.7)	(64.3)

Presenting a sensory flash at the left side of the screen has produced a reduction in the total amount of word omissions made by B.Q. The two Gap+Flash conditions increased the amount of words read (by 11.5% and 13.5 %) when compared to the amount of words read in the gap only condition. The amount of words read in the 800+Flash condition was significantly greater than in the 800 ms gap condition (chi^2 1df = 7.16 $p <0.05$) and the amount read in the 1200+Flash condition was significantly greater than in the 1200 ms gap condition (chi^2 1 df= 6.12 $p < 0.05$). The percentages of words read from the left and right sides of the screen are displayed in Table 19.

Table 19. The amount of words read from the left and right sides of the VDU screen in the Gap-flash condition.

	800 gap		800 gap-flash		1200 gap		1200 gap-flash	
Side of screen	Left	Right	Left	Right	Left	Right	Left	Right
Percentage of words read*.	24%	89%	41%	95%	34%	87%	52%	86%

The use of the stimulus flash has produced a significant reduction in the amount of words omitted from the left side of the screen, but has not significantly reduced the amount of words omitted from the right side. Left sided word omissions were reduced from 24% in the 800 ms gap condition to 41% in the 800 gap-flash condition (chi^2 1df=7.7 $p<0.05$). Left sided word omissions were reduced from 34% in the 1200 ms to 52% in the 1200 gap-flash condition (chi^2 1df =9.15 $p<0.05$). There was no significant reduction in the amount of right sided word

omissions (800 Vs. 800 gap-flash chi^2 1df=1.65 n.s..) and (1200 Vs. 1200-flash chi^2 1df=0.08 n.s.).

7.4.2.4 Discussion: Experiment N6.

B.Q. read some 34% of the words from the four passages presented in the control condition. Word omissions were primarily for the words located from the left side of each line. When passages were presented one line at a time in the gap conditions, B.Q. made less left sided word omissions than in the control condition. A significant reduction in the amount of word omissions was made in the 0 gap condition, suggesting that some of this improvement may be attributed to the procedure of single line presentation and not attentional disengagement. The abrupt onset of each line on the screen could increase B.Q.'s overall level of arousal and vigilance, which may reduce the severity of her neglect and reduce word omissions. A second possibility is that it may simply be less demanding to read single lines than to read a whole passage of text. The reduction in the amount of stimuli present could reduce the demands on preattentive processes normally required to monitor which line has being read, to enable an orienting response to be made to the next line down.

The amount of words B.Q. omitted when gaps of 400 and 800 ms were used, did not differ from the amount made in the 0 gap condition. The use of the 1200 ms gap did produce a significant decrease in the amount of word omissions that B.Q. made. However, even with a 1200 ms gap B.Q. still neglected a large number of words from the left side of each line. Two explanations are considered as plausible explanations of the failure of long gap intervals to give a greater reduction in B.Q.'s left sided word omissions. The first is that B.Q.'s attention could become engaged onto the blank VDU screen itself, following the offset of the line. A deficit of attentional disengagement would again impair B.Q.'s ability to locate the start of the next line. The second possibility is that a return saccade is not made far enough to the left, as there is no salient left sided feature to act as a saccade reference point during the gap interval. The left side of a complete passage would normally be signalled by the left text/margin break, a feature which is not available during in the gap interval. The left saccade produced during the gap interval will have to be made to a remembered left location. A deficit in B.Q.'s ability to voluntarily produce a saccade using a memory representation of the left side of the screen, would produce a failure to make the large left return saccade.

The use of a stimulus flash presented during the gap intervals produced a further reduction in B.Q.'s left sided word omissions. This can be explained by the cueing effect of the stimulus, which automatically orients attention to the left, which enables a larger contralesional saccade to be made from the ipsilesional side of the screen. The stimulus flash would be expected to automatically orient attention to that location, in the same way that peripheral cues have been shown to attract attention in manual reaction time experiments (eg. Posner, 1980). Given that there could be a link between the attentional orienting and saccadic systems (Rizzolatti et al., 1987; Shepherd, Hockey and Findlay, 1986; Umiltá et al.,1991) then a cueing

procedure which attracts attention, could also be expected to improve the production of an eye movement to that location. The improvement in B.Q.'s ability to saccade contralesionally would also reduce the amount of left sided word omissions made.

B.Q. continued to omit left sided words even when a stimulus flash was presented during the 800 and 1200 ms gap intervals. This suggests that a left saccade is made, but it still falls short of the far left location. This would again result in the first few words from the line being omitted. A second possibility is that B.Q. makes a left saccade sufficiently large to move her eyes to the start of the next line, but still fails to read the left sided words. The suggestion is that B.Q. could fail to consciously report left sided words, that have been scanned, in an extinction type phenomena. The possibility that left sided words are subject to an extinction effect could be resolved by accurate recording of B.Q.'s eye movements.

An alternative explanation of the reduction in word omissions made when a stimulus flash is used is that it does not serve to 'cue' attention, but simply provides a reference point for the saccade to be directed towards during the gap interval. Further control experiments are required which use a static left marker (such as a vertical line down the left edge of the screen: (eg. Weinberg et al., 1977), to see if this produces a benefit on reading single lines presented using the gap procedure.

7.5 Experiment N7: An examination of the strength of the cueing effect.

7.5.1 Introduction

In this experiment the stimulus cue was presented after the next line of the passage had already appeared on the screen. The effect of the cue in this case has to compete with attentional disengagement. B.Q. read one whole passage of text in a **Control condition**; one passage presented one line at a time in a **Line-condition** and a passage presented one line at a time with a stimulus flash presented after the line had appeared on the screen (**Line-Flash**). The apparatus and procedure were identical to that already described in Experiment N6. The passages used in this experiment were taken from a different source ('The Chronicles of the 20th Century') to those used in Experiment N6. The comparison was between the amount of words omitted when single lines were read with and without a stimulus flash. This testing session was performed some months before Experiment N6 (7/8/91).

7.5.2 Method.

Apparatus and procedure.

The apparatus and procedure were identical to those already described in Experiment N6.

Display sequence: Line-flash condition.

Figure 27 shows the timing sequence used in the **Line-flash condition** of experiment N7. The passages of text were presented one line at a time onto the VDU screen. As B.Q. read out the last word from that line, the experimenter pressed a key to clear the screen. After a delay (200 ms) the next line appeared on the screen and the stimulus flash was then presented at the left end of that line. In this condition the stimulus flash was presented simultaneously with the onset of the line on the screen, unlike the Gap-flash condition where the flash appeared during the gap interval while the screen was blank. The stimulus flash was identical to that already described in the gap-flash condition, but in this condition was presented ten times (on for 100 ms off 100 ms) for a total of two seconds. In the **Line-condition** a comparable passage was presented after a delay of 200 ms, but there was no stimulus flash. The **Control-condition** presented a whole passage of text onto the VDU screen.

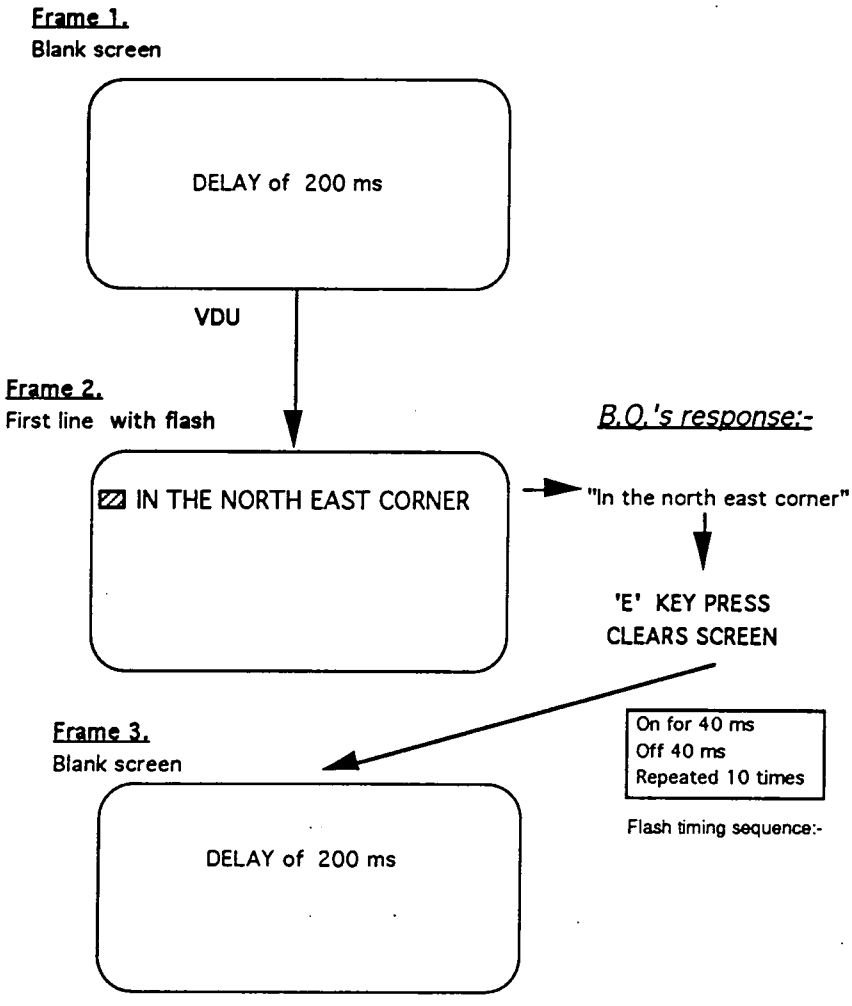


Figure 27
Sequence of frames in 'Line-flash' condition of Experiment N7.

7.5.3 Results

Table 20 shows the numbers of words read by B.Q. with a whole passage of text, single lines and from single lines which used a stimulus flash.

Table 20. Amount of words read by B.Q. in Line-Flash condition of Experiment N7.

	Control-condition	Line-condition	Line-flash condition
Number words read	27/66	38/71	44/78
(Percentage)	(42%)	(53%)	(56%)

B.Q. read more words (+10%) when the passage was presented one line at a time (Line-condition), than when reading a whole passage of text in the Control-condition, as was shown in Experiment N6. The amount of words that B.Q. omitted in the Line and Line-flash conditions is shown to be similar, with some 55% of words being read from each passage. The stimulus flash has not reduced the amount of word omissions made when compared to the amount made presenting single lines only onto the VDU screen.

7.5.4 Discussion: Experiment N7.

A stimulus flash presented at the left side of the line, after the line had appeared on the screen, has had no effect in reducing the amount of left sided word omissions made by B.Q. Experiment N6 however, showed that B.Q. made fewer left sided word omissions when a stimulus flash was presented during the gap interval. This suggests that the stimulus flash can only orient attention when the screen is blank. The presence of the line on the screen, appears to be sufficient to prevent B.Q. orienting to the stimulus flash. It is possible that the onset of the line on the VDU screen, engages her attention and her deficit of attentional disengagement inhibits the orienting response being made to the left sided stimulus flash. The cueing effect of the stimulus flash is not effective at orienting attention when the line is present on the screen.

During this testing session B.Q. read some 50% of the words from the passage in the control condition, which is more than read from the passages in the previous experimental session. This apparent discrepancy could be due to variability in her level of neglect across different testing sessions, or to the fact that the passages used in this experiment were taken from a different source to those used in Experiment N6.

7.6 General discussion and conclusions.

B.Q.'s normal text reading performance (See Figure 24) indicates that she reads most of the words from the first line of a passage, but then omits over half of the words from each subsequent line. B.Q. again showed less neglect for the first lines of each passage when reading passages in the control conditions. This reduction in neglect for the first lines, could have three possible explanations. The first is simply that B.Q.'s level of vigilance/arousal (Karnath's component C), decreases over the time taken to read all of the lines in a passage. A decrease in the overall level of arousal, might be expected to increase the amount of words neglected. This explanation is weakened by the observation that the amount of words omitted remained relatively constant over the remaining thirteen lines. There is little evidence that the amount of word omissions made by B.Q. increased further with increasing time. The second possibility is that B.Q. can locate the first word of the first line of a passage of text, as the demands on the attentional system are initially low before reading has started. Any increase in the general attentional demands once reading has started, could increase neglect and impair attentional orienting on subsequent lines.

A third possibility as to why B.Q. reads more words from the first line from a passage, is that the detection of the starting position of a passage of text might reflect a different process to that required to locate the start of each subsequent line. Nakayama (1989) suggested that the representation of the visual scene is organised in a multilevelled, multi-scaled, 'feature pyramid', where the image is represented at many different spatial scales. It was further suggested that attentional sampling can be performed to either; a large scaled representation of low resolution, or to a small scaled representation of high resolution. Although the idea of the deployment of attention at different spatial scales, is highly speculative, it could be applied to the process of reading a page of text. The attentional process required to locate the starting position of a whole passage, could involve orienting attention within a large coarse level description of the whole text area. Once reading has commenced the process of locating the start of the next single line, could involve orienting within a smaller spatial scaled representation. B.Q.'s ability to locate the start of the first line requires the additional assumption that she is impaired at orienting attention contralesionally when small scaled representation (at the level of individual words) are deployed, but is less impaired at orienting attention contralesionally with the large scaled representations which code the text boundary.

The results of Experiment N6 showed that a deficit of disengaging attention (from the last word of the previous line) does not on its own account for B.Q.'s whole word omissions. This is also indicated during B.Q.'s normal text reading (Figure 24) as the last two-three words were read from each line, suggesting that attentional disengagement is taking place. In the zero-gap condition, the onset of the new line is simultaneous with the offset of the line which had been read, so there should be no attentional disengagement. However, B.Q. made a similar amount of word omissions when gaps of 400 and 800 ms were used which should enable attentional disengagement. Only a 1200 ms gap produced a significant reduction in

word omission, compared to the zero-gap condition, but a large number of left sided omissions were still made. It is possible that a much larger gap interval is required to disengage attention during reading than has been observed in the previous saccade experiments (Chapter six), but B.Q.'s ability to read approximately half of each line suggests that attentional disengagement is taking place. The implication is that B.Q. can disengage attention from the last word of a line, but makes a saccade which falls short of the far contralesional location, close to the midline of the screen. Taken together these results suggest that B.Q.'s left sided word omissions do not result from a deficit of attentional disengagement.

Two further factors could account for B.Q.'s failure to read words from the left side of the screen when the longer gap intervals were used. The first is to suggest that her attention may become 'engaged' onto some part of the ipsilesional side of the VDU screen. This re-engagement of attention may take place while the return left saccade is being made and the engagement of attention at a location causes the saccade to be made to that position. A second possibility is that the saccade falls short of the far left side of the screen, due to the absence of a salient left sided feature. Although attention is disengaged by the gap interval there was no salient left sided feature (as the screen remained blank) for the preattentive processes to use to guide the left saccade. If the absence of a left sided feature was the only reason why the left saccade was not made to the far side of the screen, then B.Q. should have been able to make a saccade when a stimulus flash was used in the Gap-flash condition. However, B.Q. still made left sided word omissions when the left cue was used, suggesting that the absence of a left sided feature is not the only reason why words were omitted during the gap procedure.

B.Q. made significantly less left sided word omissions when a stimulus flash was presented during the 800 ms and 1200 ms gap intervals. This suggests that the stimulus flash can automatically summon attention to the left side of the screen and facilitate the production of the left saccade. The size of the return saccade is sufficient to enable more words located on the left of the screen midline to be read. The reduction in word omissions by the use of the cueing procedure supports the hypothesis that a deficit of attention is involved in producing left sided word omissions. Experiment N7 showed that the cue was not effective at reducing word omissions if the line was present on the screen. This is consistent with the idea that the deficit of attentional disengagement prevents attention being oriented contralesionally.

A further question is why B.Q. still made left sided word omissions when a long gap interval (which should have disengaged attention) and a stimulus flash (to cue attention) were both used with single line presentation. For example, in the 1200 ms Gap-Flash condition, B.Q. still only read 65% of the words from the five passages. Three reasons why B.Q. might still make left sided word omissions in the Gap-flash condition are proposed. The first is that even with a gap and cueing procedure B.Q.'s return left saccade still falls short of the far left side of the screen. The failure to make a saccade to the left side of the screen, being due to the severity of B.Q.'s deficit in orienting attention into the contralesional side of space. An idea proposed by Tegnér et al. (1990) to explain the performance of neglect patients on line bisection tasks, could

be used to explain B.Q.'s failure to orient attention to the far left location. Tegnér used an idea of a compression of the representation of hemispace originally suggested by Werth and Pöppel (1988). Werth and Pöppel had observed that normal subjects showed a tendency to 'compress' the representation of imagined hemispace. The idea is that neglect patients use the right side of a stimulus (or VDU screen in this case) as the reference point to build up an internal representation of space and the 'compression' of the left side of the representation takes place. If B.Q. forms a compressed representation of the left side of the VDU screen then the resulting left saccade could fall short of the far left side of the screen.

The second reason for B.Q.'s word omissions in the Gap-flash condition, involves the possibility that her return left saccade is made to the far left side of the screen, but the abrupt onset of the next line produces a rightward orienting of eyes/attention. This is consistent with Karnath's (1988) model of neglect, in which he suggested that one component involved is the automatic orienting response to the right (Component A). It is possible that B.Q. makes a left saccade to the cued location during the gap interval, but the automatic rightward orienting is produced by the onset of the next line to be read. This results in B.Q. failing to read the first words from that line. The possibility that B.Q. does make a return left saccade to the far left side of the screen, with the additional possibility of a small right saccade being made following line onset, could be shown by accurate eye movement recording. Unfortunately as has already been explained the problems of head movements made by B.Q. makes this difficult to perform.

A further possibility to consider is that B.Q. does make a large return left saccade and scans the first words of the next line, but 'extinguishes' these words from conscious awareness. Young et al (1992) showed that B.Q. possessed some knowledge of the left sided features of Chimaeric photographs, and could describe the features of the left sided face, but overtly reported only the face on the right. B.Q. may also fail to consciously recognise the left sided words of a line due to a similar deficit of conscious awareness for left sided stimuli. Once again an accurate eye movement recording would help resolve this issue. An indication of the scanning of left sided words which are not reported in the eye movement record would support this 'extinction' like phenomena as being another factor involved in left sided word omissions.

The last explanations of B.Q.'s left sided word omissions, ('compression of hemispace' and 'extinction of left sided words'), are both highly speculative. They are provided here to illustrate the possible reasons to account for B.Q. continuing to make left sided word omissions, even when attention was disengaged and cued to the left. The use of the gap procedure produced a small reduction in B.Q.'s left sided word omissions and the cueing procedure, produced a larger decrease in word omissions, but was only effective if presented during the gap interval. The tentative conclusion of the present series of experiments is that an impairment of disengaging attention from the ipsilesional side of the page, and a failure to orient attention far enough contralesionally are both involved in producing B.Q.'s word omissions. This failure to orient attention to the contralesional side of the page (or VDU screen) resulting in the return left saccade falling short of the starting position of the next line, with left sided word omissions occurring. An additional lack of insight (by B.Q.) that she is omitting words could be a factor

which prevents her using a top-down search strategy to search for the start of the next line, even though the return saccade has not been made to the starting position. A further reason for B.Q.'s inability to compensate for her attentional deficit could be a generalised lack of vigilance/arousal (Karnath, 1988; Robertson and Frasca, 1992). This non lateralised deficit would be expected to produce a deterioration in performance on any task which requires a certain amount of attentional load such as reading. Robertson and Frasca (1992) showed that increasing task demands can produce more neglect for some patients, especially those like B.Q. that have shown little evidence of recovery post lesion. Reading is a task that will increase the load on the attentional system, and so could be expected to produce an increase of neglect which is evident for words located in the right side of the screen.

Chapter 8

A functional model of visual attentional orienting.

8.1 Introduction.

The following section describes a functional model of attentional orienting. The model is based on the idea that a similar system is responsible for orienting visual attention as is involved in producing a saccadic eye movement. A link between the eye and attentional, orienting system has been observed (Shepherd, Findlay and Hockey, 1986) and resulted in the suggestion of a 'premotor' model of visual attention (Rizzolatti et al., 1987; Tassinari et al., 1987; Umiltá et al., 1991). However, previous premotor accounts have been based on a somewhat limited awareness of the current models of saccade generation.

Recent models of saccade generation have emphasised a distributed coding of the location of the target stimulus, in what have been termed 'motor maps' (McIlwain, 1986; Findlay, 1987). Models have also disassociated the non-spatial effects of target onset, from the computation of the spatial position of the target by incorporating separate when and where components (Becker and Jürgens, 1979). The aim of the model proposed in this chapter, is to show how a model which orients both the eyes and attention, could account for the findings from the present laboratory experiments with normal subjects, and those obtained from the neglect patient B.Q. Once a precise model has been specified some of its limitations become apparent. The proposed model cannot aim to account for all of the findings from the eye movement literature, nor can it account for all of the many and bewildering manifestations exhibited by patients with visual neglect. Some of the limitations of the model are outlined in the final section of this chapter.

8.2 The proposed model of the attentional and eye orienting system.

The following section contains a description of the component parts of the model, which is shown in Figure 28.

The first aim of the model is to show how the presence of **inhibition** within the orienting system can account for the results of the present laboratory experiments. In these experiments normal subjects directed their attention voluntarily to a cued direction, or location, and the latencies of the saccades made to targets in the attended and non-attended locations were examined. The results indicated a small speeding of saccade latency when the targets were presented at the attended location and a much greater slowing of latency, with targets presented in the non-attended hemifield.

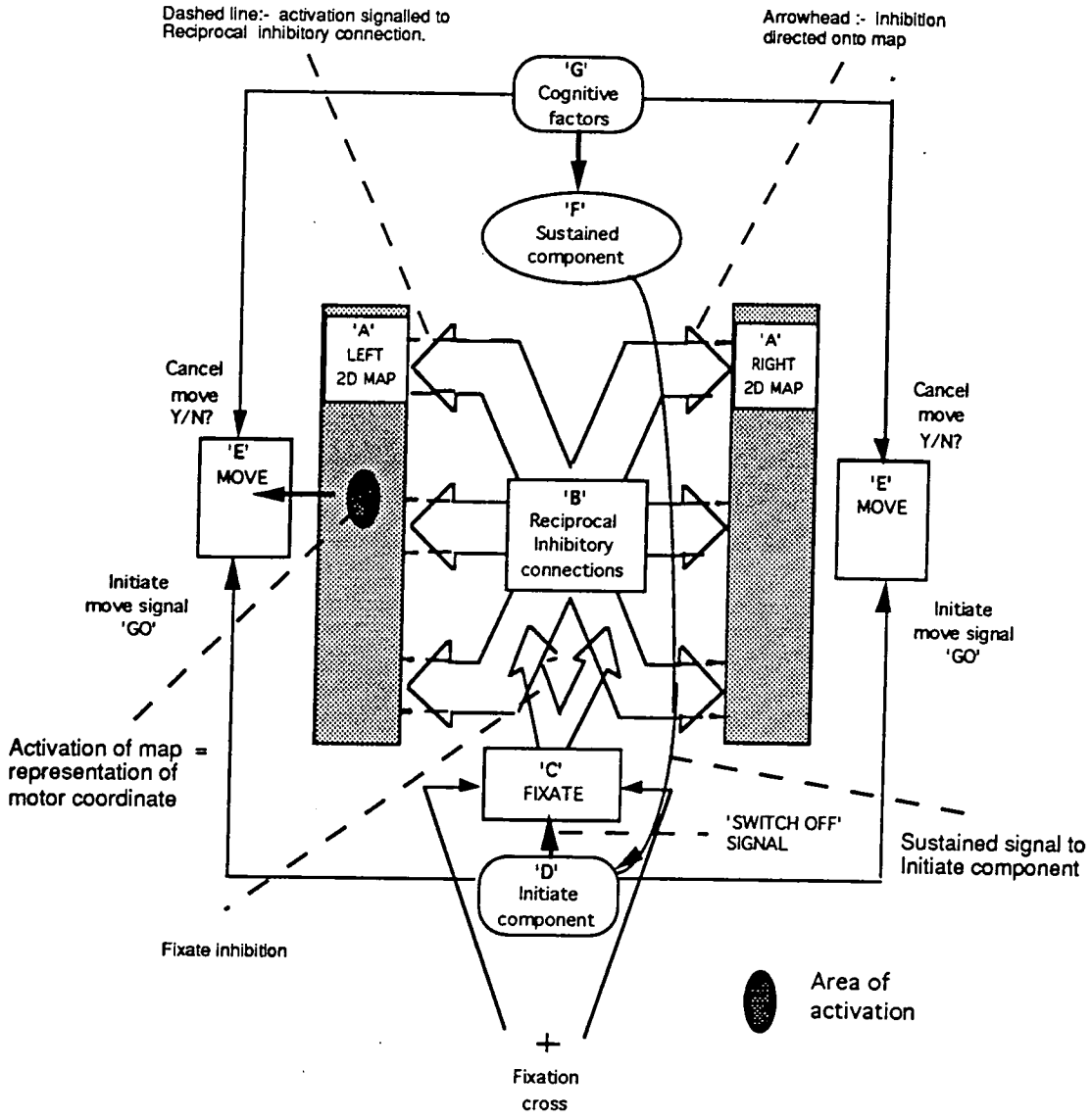


Figure 28
The proposed model of the attentional and eye orienting system.

A. Left and right 2D motor maps: The motor coordinate is represented by an area of activation in the 2D map, which contains the vectors to produce a movement in two dimensions (c.f. McIlwain (1986) and Findlay (1987)). The rise rate of the area of activity depends on the amount of inhibition acting on the map.

B. Reciprocal inhibitory connections: The Reciprocal inhibitory connections are stimulated by the presence of activity in a 2D maps (by the dashed pathways) and directs inhibition to the non-activated regions of both 2D maps (arrowhead pathways).

C. Fixate component: The Fixate component is activated during attentive fixation and generates inhibition on both 2D maps, via the Reciprocal inhibitory connections. The Fixate component is switched off when the Initiate component is activated, or if the attended stimulus is removed.

D. Initiate component: The onset (or offset) of a target stimulus, or a signal from the Sustained component will activate the Initiate component which computes the final 'trigger' signal, sent to the Move component.

E. Move components: The Move component functions as an AND gate which requires a trigger signal from the Initiate component, the motor coordinates represented by activity in the 2D map. The final decision to make a saccade can be vetoed by a signal from the cognitive system.

F. Sustained component: The Sustained component enables eye/attentional movements to be made under voluntary control, by producing an area of activity in a 2D map, and sending a signal to the Initiate component.

G. Cognitive factors: This component represents the higher level cognitive factors which can influence the orienting of the eye and attention. The link with the cognitive system is required to interpret symbolic cues and to orient attention covertly by sending a veto signal to the Move component.

8.2.1 Description of the model components.

A. Left and right 2D motor maps: The model uses two separate 2D maps involved in forming a representation of stimulus position. One map forms a representation of left visual space and one forms a representation of right visual space. The left and right 2D maps transform the stimulus position into a representation of the motor commands required for a saccade to the stimulus location (similar to those suggested by: Deubel et al., 1984; McIlwain, 1986; and Findlay, 1987). The exact coordinates of the motor movement is specified by a spatial average of the area of activation within the relevant 2D map. The area of activity contains the spatial information for the motor signal required to move the eyes (up, down and horizontal, coordinates). In the present model the area of activation is either produced by a stimulus onset; or by the Sustained orienting component which can generate an area of activity in the map without a sensory signal. The strength of the activated area in the 2D map is influenced by the amount of inhibition operating on the map at the time of stimulation. The area of activation specifies the desired motor position in 2D coordinates. This transformation into a motor command is assumed to be completed when threshold level of activity has been reached. The motor coordinates are then signalled to the MOVE component for the production of the final motor output.

B. Reciprocal inhibitory connections: The left and right 2D maps are connected by Reciprocal inhibitory connections. The dotted lines indicates the pathways *from* each of the 2D maps, which switch on the Reciprocal inhibitory connections. The arrowheads indicates the pathways which direct inhibition *onto* the 2D maps. Activation of one 2D map results in inhibition being directed to <u>all</u> areas of the contralateral map. Activation of a particular region of a 2D map also results in inhibition acting ipsilaterally on the other areas of that map. The presence of the inhibition reduces the level of activity in the 2D maps, which reduces the possibility of a response being made to stimuli presented in the inhibited areas.

C. Fixate component: The Fixate component is activated when a stimulus is actively fixated and produces inhibition via the Reciprocal inhibitory connections, which acts on both the left and right 2D maps. The Fixate component activity is suppressed by a signal sent from the Initiate component. The result of active fixation is to produce a bilateral increase in the inhibition operating on both the left and right 2D maps which inhibits a response being made to a stimulus presented at any other location.

D. Initiate component: The onset (or offset) of a target stimulus will activate the Initiate component which starts to programme a saccade initiate ('trigger') signal. The Initiate component can also be activated by a signal from the Sustained component. This enables a voluntary eye movement to be produced without there having to have been a sensory change in the visual field. Activation of the Initiate component switches off the Fixate component. The final output from the Initiate component is the initiate (or 'trigger') signal, sent to both of the Move components. This signals the final decision to initiate the motor coordinates of the desired saccade.

E. Move components: The final decision to produce the motor response can be made by the Move component on receiving a trigger signal from the Initiate component. The Move component functions as an AND gate which requires a trigger (initiate signal) and the desired motor coordinates (from 2D map), before it will orient the eyes to the stimulus location The activity in the relevant 2D map forms a representation of the coordinates of the motor response required to move the eyes to to stimulus position which is sent to the Move component. The final decision to make a saccade can be vetoed by a signal from the cognitive system.

F. Sustained component: The Sustained component is incorporated as a highly speculative account of how the voluntary orienting of the eyes/attention is possible. The Sustained component indicates the process which can voluntarily produce an eye, or attentional movement from higher level cognitive control (without any sensory change). The Sustained component functions by producing an area of activity in the relevant 2D map at the location corresponding to the desired movement. The Sustained component also generates inhibition acting on the non-attended 2D map. This inhibition is directed onto the whole of the non-attended 2D map by the Reciprocal inhibitory connections (indicated in the Figure by the long arrowed lines). For the Sustained component to be able to generate an area of activity at the desired location and direct inhibition to the non-attended locations, it must also contain a representation of the visual scene. In addition to producing activation and directing inhibition, the Sustained component sends a signal to the Initiate component. The link with the Initiate component enables the Sustained component to be able to 'voluntarily' produce a saccadic eye movement. The Sustained component is connected to the cognitive system which enables the cognitive control of the voluntary orienting of eyes/attention.

G. Cognitive factors: This component represents the higher level cognitive factors which can influence the orienting of the eyes and attention. The cognitive system is required to interpret symbolic cues to direct eye/attention movements. The instruction to orient attention covertly without moving the eyes also requires a link between the higher level cognitive systems and the orienting system to suppress the final eye movement. The link with a higher order system is also necessary for an observer to move their attention/eyes to a remembered stimulus location, or to a location opposite to that in which a stimulus has appeared, as is the case with antisaccade (Hallett, 1978; Hallett and Adams, 1980).

8.2.2 Assumptions of the premotor model.

The following is a list of some of the assumptions made about the normal functioning of the proposed model.

1. The activity in the 2D map produced by a stimulus onset, rises at a rate which depends on the level of inhibition acting on that map.
2. The Reciprocal inhibitory connections are activated when a stimulus is projected onto one of the 2D maps and generate inhibition onto the non-attended map.
3. The activity in the map starts to decrease when the stimulus is removed and the inhibition generated by the Reciprocal inhibitory connections will also start to decrease.

4. The inhibition generated by the Reciprocal inhibitory component is fast acting (0-20 ms) but decreases slowly (up to 100 ms).
5. The Fixate component produces inhibition directed onto the 2D maps by the Reciprocal inhibitory connections.
6. The Fixate component is switched off following the activation of the Initiate component.
7. The programming of the initiation ('trigger') signal takes a certain amount of time to complete.
8. The initiation component can be triggered by a stimulus onset, stimulus offset and by a signal from the Sustained component.

8.2.3 The normal operation of the premotor model.

The following section briefly describes the normal functioning of the proposed premotor model. For ease of description and in keeping with the experiments described in this thesis, it is assumed that the viewer is sitting upright with their head, body and retinal midline coincident with a central fixation point.

8.2.3.1 Eye/attentional movement following a peripheral stimulus onset.

The onset of a stimulus in the visual field has two initial effects. The stimulus position is projected onto the relevant (left or right) 2D map, which produces an area of activity. The level of activity in the stimulated region of the 2D map, will rise at a rate dependent on the level of inhibition acting on the map from the Reciprocal inhibitory connections. Activity in the 2D map is then signalled to the Reciprocal inhibitory component (via pathways shown as dashed lines) which serves to produce inhibition on all of the contralateral 2D map and also on non-activated quadrants of the stimulated 2D map (via the pathways shown with arrowheads). A stimulus onset also has the effect of activating the Initiate component, which begins to programme a saccade 'trigger' signal. The process of attending to a central fixation point produces additional inhibition in the Reciprocal inhibitory connections from activation of the Fixate component. The Fixate component is switched off by a signal from the Initiate component which reduces this inhibition. The local area of activity in the 2D map is made available to the Move component and form a representation of the motor command required to make a saccade to that position.

The final decision to execute a saccade is triggered by a signal sent from the Initiate component to the Move component. The Move component serves as an AND gate and can execute a saccade specified in terms of the motor coordinates represented by the output signal from the 2D map. The Move component will not execute this command if a veto signal is received from the cognitive system.

8.2.3.2 Eye movement to a peripheral stimulus onset in gap situations.

The model can account for the speeding effect shown with prior fixation point offset and onset shown in the literature (eg. Ross and Ross, 1980, 1981). The effects of prior fixation **offset** are two fold. Firstly the fixation point offset activates the Initiate component which starts to programme a trigger signal. This allows the initiate trigger signal to be at least partly computed

before the saccade target appears. Activation of the Initiate component also switches off the Fixate component, thus decreasing the level of inhibition in the Reciprocal inhibitory connections. This decrease in inhibition will result in a faster rise rate of activity in the relevant 2D map, when the target stimulus is presented. The representation of the stimulus motor coordinate will be signalled to the Move component, in less time than is the case during active fixation. The preprogramming of the Initiation signal results in the final trigger to execute a saccade being sent to the Move component, in less time than would be the case without an advanced warning signal. The facilitation effect obtained by prior fixation **onset** can also be explained by a warning signal effect, which enables preprogramming of the Initiation component. The difference in this case is that the presence of the fixation point results in the Fixate component maintaining its inhibitory influence. The result in terms of saccade latency is that a facilitation effect is obtained following prior fixation onset, but the size of this facilitation is not a great as that obtained with prior offset of fixation.

8.2.4 The model should be able to account for the following factors:-

8.2.4.1 Normal subjects:

1. Large costs obtained with single targets in non-attended direction.
2. Small benefits obtained when targets were presented in attended direction..
3. How the horizontal and vertical orienting of attention is possible.
4. Facilitation effect obtained with prior fixation offset (gap effect).
5. Increase in latency with bilateral double target simultaneous presentation.
6. The decrease in latency when a non-attended target was presented at long intervals before the saccade target.
7. How the covert orienting of attention is possible.
8. How voluntary movements of the eyes are possible.

8.2.4.2 The neglect patient B.Q.:-

The model also aims to account for the findings obtained from the neglect patient, B.Q. It has been suggested that the deficit in responding to contralesional stimuli, shown in neglect patients, is due to a defective attentional orienting system. The model shows how damage to specific components of the proposed orienting system could produce unilateral neglect. The gap effect improved B.Q.'s ability to orient to contralesional stimuli, so the model needs to be able to explain how this improvement is possible. The results of the reading experiments revealed a slightly more complicated pattern of B.Q.'s failure to orient into contralesional space which also needs to be explained.

1. B.Q. failure to orient contralesionally, when attending to a fixation point.
2. The gap effect improved B.Q.'s ability to orient contralesionally.
3. The gap effect also improved B.Q.'s ipsilesional orienting.
4. The gap effect did not substantially reduce B.Q.'s whole word omissions.
4. A stimulus flash did reduce B.Q.'s whole word omissions.

8.3 An explanation of the experimental results with normal subjects with reference to the proposed model.

The following account aims to explain the results that directing attention had on the saccade latency of normal subjects. Figure 29 shows the model in a state of directed visual attention, and serves to illustrate the following account of the experimental results. Attention has been directed to the left, by the Sustained component (following a symbolic cue). A left saccade target is also shown which produces an area of localised activity in the left map and activates the Initiate component. Activity in an area of the left map may also produce a mirror image area of inhibition in the right map.

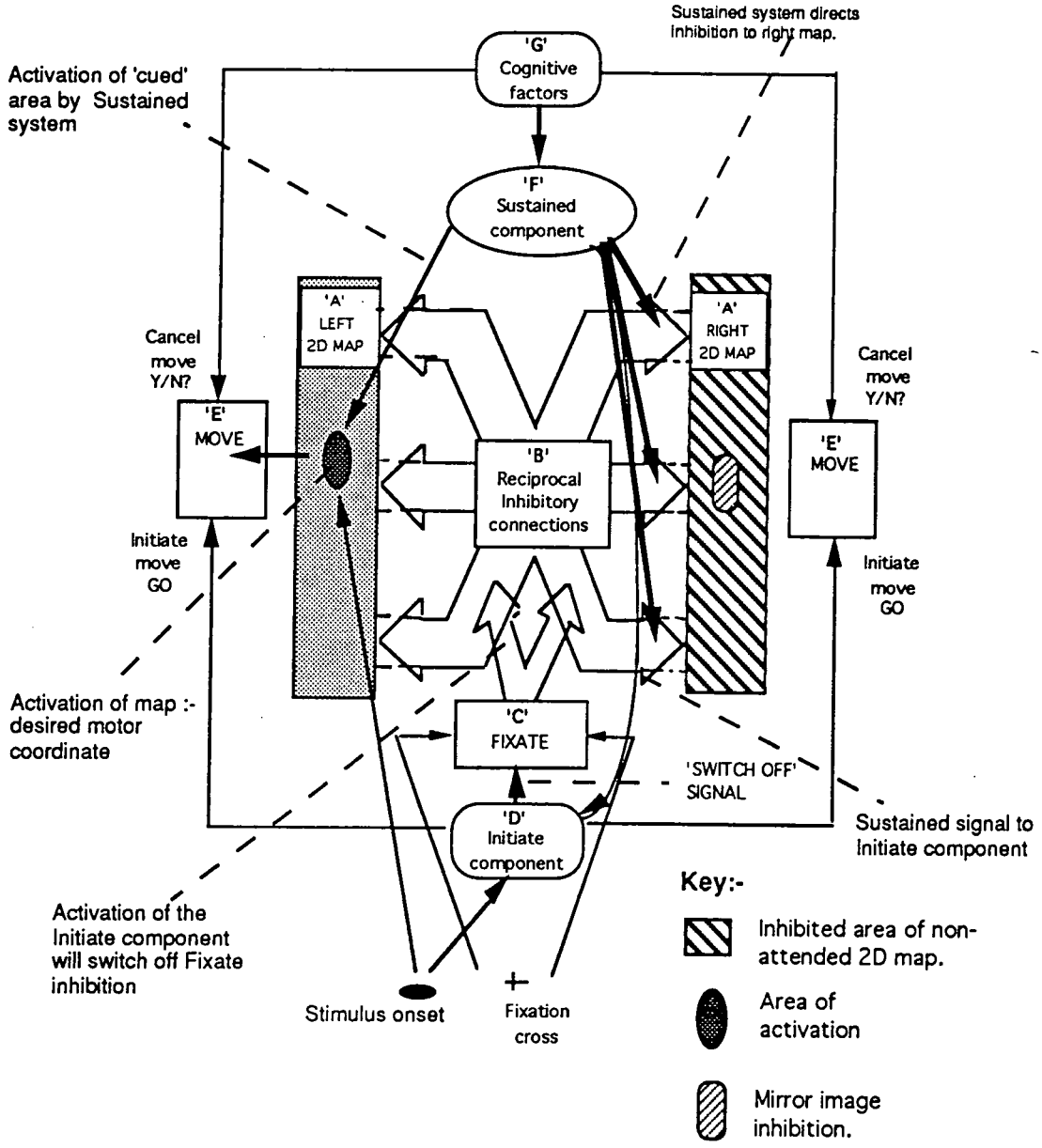


Figure 29
The model shown when attention has been voluntarily directed to the Left.

This results in inhibition being directed to the right map.
A left saccade target is also shown which produces an area of activation in the left map .

8.3.1. The large costs obtained with single targets in non-attended direction.

When attention was voluntarily oriented to one direction/location the latencies of saccades made to targets presented in the non-attended hemifield were very much slower than those obtained to targets in the attended hemifield. This slowing is explained as follows:-

On attentional trials the cognitive system provides an interpretation of the cue (indicating the 'attended' location) which is passed to the Sustained component. The Sustained component serves to produce a weak area of activation in the relevant ('attended') 2D map corresponding to the cued location. The Sustained component also produces inhibition directed onto the whole of the non-attended 2D map by the Reciprocal inhibitory connections. A target stimulus presented in the non-attended hemifield will project into the inhibited (non-attended) motor map. The consequence of the inhibition is that the time taken for the transformation of stimulus position into a motor coordinate, will be greater than under neutral conditions, when there is less inhibition acting on the 2D maps. The extra time is apparent as an increase in saccade latency (cost) when compared to targets presented to the attended hemifield and targets presented in neutral conditions.

8.3.2. The small benefits obtained with single and bilateral targets in the attended direction.

A small facilitation effect was observed with saccades made to targets (single and bilateral simultaneous) which were presented in the attended hemifield (exp. 1-5). This speeding effect reached significance compared to uncued conditions, when the cueing procedure accurately indicated both the direction and location of the saccade (exp. 8). When attention is voluntarily oriented (by a cue) it is assumed that the Sustained component produces a weak area of activation within the relevant motor map. When attention is cued directionally (exp's. 1-5) the area of activation could be widely distributed over the whole map, but when a specific location is cued (exp 8) it may be narrowed onto a smaller area. The prior activation of the motor map produces a small facilitation effect, due to the speeding of the time taken for the map to reach its peak level of activation following a stimulus onset at this location. The deployment of the Sustained component is assumed to produce a low level of activity in the 2D map, so the speeding effect on saccade latency is not great. The greatest activation of the motor map is produced by a stimulus onset.

8.3.3. The small costs for targets at non-attended locations within the attended hemifield.

The mean saccade latency obtained to targets presented at a non-attended location within the attended hemifield was greater than for a target presented at the attended location (exp. 8). However, this cost was greater when the saccade was made to the far eccentricity location following a cue to the near eccentricity location, than when the saccade was made to

the near eccentricity location while attention was directed to the far eccentricity location. This suggests that the area of activation produced by the Sustained component, may actually spread outwards from the area representing the central (foveal) region to the peripheral (cued) location. Directing attention to the near target location produces an area of activation in the 2D map which spreads from the fovea covering the area representing the near eccentricity location. A target presented at the far location following a cue to the near location, will not project into an area of activation. When attention is directed to the far target location the area of activation spreads from the fovea and covers both the near and far stimulus positions. Saccades made to targets presented at both the near and far eccentricity positions will obtain a similar facilitation effect.

8.3.4. The generalised facilitation effect obtained with prior fixation point offset.

Saccade latency was show to be reduced when the fixation point went off 100 m.sec. before the onset of the saccade target. This facilitation was apparent with saccades made to single targets, and with saccades made to targets presented bilaterally and simultaneously, under both attentional and neutral conditions (exp. 2). The 'gap' appears to have a generalised facilitation effect which is independent to the effects of directing visual attention. The Initiate component in the present model can provide part of the explanation of this non-spatial facilitation effect. The initiate component is similar to the 'when' component in Becker and Jürgens (1979) model of saccade generation. The offset of the fixation point is assumed to act as a warning signal, which can activate the Initiation component, thus enabling some preprogramming of the Initiation signal to take place prior to the onset of the saccade target. This would enable the initiate signal being sent to the Move component, in less time than when it is triggered by the saccade stimulus only (exp. 1). The reduction in the time taken for the trigger signal to arrive at the Move component, will enable a motor response to be executed as soon as the stimulus coordinates are signalled by the area of activation in the motor map. The triggering of the Initiation component by fixation point offset occurs independently of the onset of the saccade target, so a facilitation will be obtained in gap conditions with both single and bilateral targets regardless of the attentional condition.

8.3.5. Orienting attention along the vertical axis.

It was shown that directing attention to locations along the vertical axis (exp. 5) produced a similar pattern of costs and benefits on saccade latency as was obtained when attention was directed horizontally (exp. 1-4). This suggests that the model of attentional orienting should be able to account for the effects of voluntarily orienting attention along both the horizontal and vertical axis. This is achieved in the proposed model by assuming that the Reciprocal inhibitory components can operate within a 2D map, as well as between 2D maps. As already stated an instruction to: "direct attention to the left of fixation", involves the Sustained component weakly activating the left 2D map, and directing inhibition onto the whole of the right 2D map. When attention is oriented along the vertical axis (for example to the upper field), by an instruction to: "direct attention above fixation" the Sustained component will weakly activate the

upper areas of both the 2D maps and direct inhibition to the lower areas. The small speeding on saccade latency for targets presented in the attended (upper regions) will be due to the prior activation of these regions. The large costs shown for targets in the non-attended (lower fields) will be due to the presence of inhibition which slows the rate of activity generated when the stimulus is projected to this area. This ability to direct inhibition into quadrants is similar to the proposal in Hughes and Zimba's (1987) hemifield inhibition model of attention.

8.3.6. The slowing of saccade latency observed with bilateral simultaneous target presentation.

When two targets were presented bilaterally and simultaneously, the mean saccade latency was greater than when a single target was presented (exp. 1-7). This increase was observed even when a saccade was always made in the attended direction (exps. 6/7). The onset of bilateral targets produces an area of activity in both the left and right 2D maps. This activity is signalled to the respective Reciprocal inhibitory connections, which generates inhibition on both 2D maps. This bilateral increase in inhibition will slow the rise rate of activity produced by the targets, within both of the 2D maps. The consequence is the observed slowing of saccade latency with bilateral targets, regardless of the attentional instruction. This account has suggested that activation of one 2D map inhibits all of the contralateral 2D map. However, it is possible that it is the area of the map diametrically opposed to the stimulated area that is inhibited in the contralateral 2D map. As the experiments performed always used bilateral targets at equal and opposite eccentricity locations it is not possible to choose between the two interpretations.

8.3.7. The effects of presenting non-attended targets at intervals before and after the saccade target on saccade latency.

The onset of a target in the non-attended field was shown to have facilitatory and inhibitory consequences on the latency of saccades made to a target in the attended field (experiment 7). The pattern of facilitation and inhibition was shown to depend on the time interval between the presentation of these two targets. An attempt is made to explain these results in light of the proposed model.

a) The greatest inhibitory effect occurred with bilateral simultaneous target presentation.

The slowest mean latency was obtained when the non-attended target appeared simultaneously with the presentation of the saccade target. The slowing with simultaneous target onset is explained by the fast acting inhibition directed to both 2D maps and the absence of any 'preprogramming' of the Initiate component. The presence of the inhibition is assumed to act on both 2D maps almost instantly, following the onset of the two targets. This inhibition starts to decrease when the stimuli are removed, but the decay rate of this inhibition is gradual and takes up to 100 m.sec. before falling back to its resting level. With simultaneous presentation the rise rate of activity in the 2D motor maps is slowed by the presence of this

inhibition. The simultaneous onset of the non-attended target does not allow the Initiate component to be activated before the onset of the saccade target. Bilateral simultaneous target onset produces inhibition without any facilitation effect from preprogramming of the initiate signal.

b) The inhibitory and facilitatory effects obtained when a target is presented in the non-attended field 20-80 ms before the saccade target.

When the non-attended target was presented at short intervals (20-40 ms.) before the onset of the saccade target, the resulting mean latency was faster than with simultaneous target presentation, but slower than with single target presentation. When the non-attended target onset occurred 80 ms. before the onset of the saccade target, the mean latency was comparable to that obtained in the single target baseline condition. This suggests that there is both a facilitation effect and inhibitory effect produced by the early onset of the non-attended target. The facilitation is due to the early activation of the Initiation component, enabling the partial programming of the initiation signal before the saccade target onset. The inhibitory effect is due to the onset of the target in the non-attended 2D map activating the Reciprocal inhibitory connections thus directing inhibition onto the 2D map which the saccade target is presented. The level of this inhibition starts to decay following the offset of the non-attended target and has decreased to its resting level by approximately 100 ms. The presence of inhibition acting on the relevant 2D motor map by the Reciprocal inhibitory component is indicated by saccade latency being slower than in the single target baseline condition. However, the early onset of the non-attended target also activates the Initiate component which switches off the Fixate component thus reducing the inhibition acting on both of the 2D maps. The reduction in inhibition from the Fixate component results in the area of activity having a faster rise rate than occurs with simultaneous target onset. The inhibition produced from the onset of the non-attended target results in saccade latency being slower than with single target presentation. The early activation of the Initiate component also serves to enable some preprogramming of the trigger signal prior to saccade target onset. This preprogramming reduces saccade latency compared to that obtained with bilateral simultaneous targets which do not enable any preprogramming.

c) The facilitatory effects observed when a target was presented in the non-attended field 160/240 ms before the appearance of the saccade target.

The onset of a stimulus in the non-attended hemifield at a long interval (240/160 m.sec.) before the onset of the saccade target produced a facilitation effect, with the mean saccade latency being faster, than was obtained with single targets. This facilitation could be due to a warning signal effect (of the non-attended target) which activates the Initiate component and enables preprogramming of the saccade trigger signal, before saccade target onset. When the saccade target is presented the Initiation programme will be (at least) partly completed, enabling a saccade to be made soon after the onset of the target produces an area of activity in the relevant 2D map. The onset of the non-attended target will also generate inhibition on the contralateral (attended) 2D motor map from activation of the Reciprocal

inhibitory connections. However, it is assumed that this inhibition is relatively short lasting and has dissipated after approximately 100 ms; and will not affect saccade latency to a target appearing at long intervals after the non-attended target. The activation of the Initiate component by the non-attended target serves to switch off the Fixate component inhibition. There is therefore very little inhibition acting on the 2D map, when the saccade target appears, so the rise rate of activity within that 2D map is very fast. The overall effect will be a reduction in saccade latency due to the preprogramming of the Initiate signal and the low level of inhibition in the saccade target 2D map.

d) The inhibitory effect observed when a target was presented in the non-attended field at short intervals after the appearance of the saccade target.

When a target appeared in the non-attended field shortly (20-40 ms) after the saccade target had been presented, the mean saccade latency was slower than in the single target baseline condition. The slowing of latency in this instance is accounted for by the activation of the inhibitory component by the onset of the non-attended target. However, the consequence of this inhibition does not appear to be as great as was observed in the simultaneous onset condition. It is possible that the effect of the inhibition produced by non-attended target onset is not as great once the 2D map has been activated by the saccade target. The onset of the non-attended target after the saccade target, does not give an early activation of the Initiate component. There is no facilitation effect obtained from either the preprogramming of the Initiation signal, or the switching off of the Fixate component inhibition.

8.4 The covert orienting of visual attention.

The proposed model can also account for the covert orienting of visual attention, as illustrated in experiments which have shown similar costs and benefits to those obtained in the present study in the absence of an eye movement (eg. Hughes and Zimba, 1987). The final decision to execute a saccade can be vetoed by a cognitive decision not to make a saccade. A link is shown between the final Move component and the higher order cognitive factors to account for the ability a person has at suppressing the final motor output. This is the same as the suggestion of Rizzolatti et al. (1987) and Umiltá et al. (1991) that covert orienting of attention involves the cancellation of the final command to produce the final motor response. The programming of the saccade motor coordinates and trigger signal, is assumed to produce an advantage with manual RT's in covert orienting experiments. The interaction between the different motor systems (eg. eye vs. hand) is not clear, but the systems involved in orienting attention for different motor responses could be closely linked.

8.5 An explanation of the deficits observed in the neglect patient B.Q. in terms of the proposed premotor model.

The proposed model could also account for some of the deficits that B.Q. was observed to have when orienting her eyes/attention. The following account explains how some of the experimental observations from the study of B.Q., could result from damage to two separate components of the model shown in Figure 30. Neglect could involve the loss of the 'Switch off' signal to the Fixate component and the loss of the Reciprocal inhibitory connections operating on the ipsilesional 2D map.

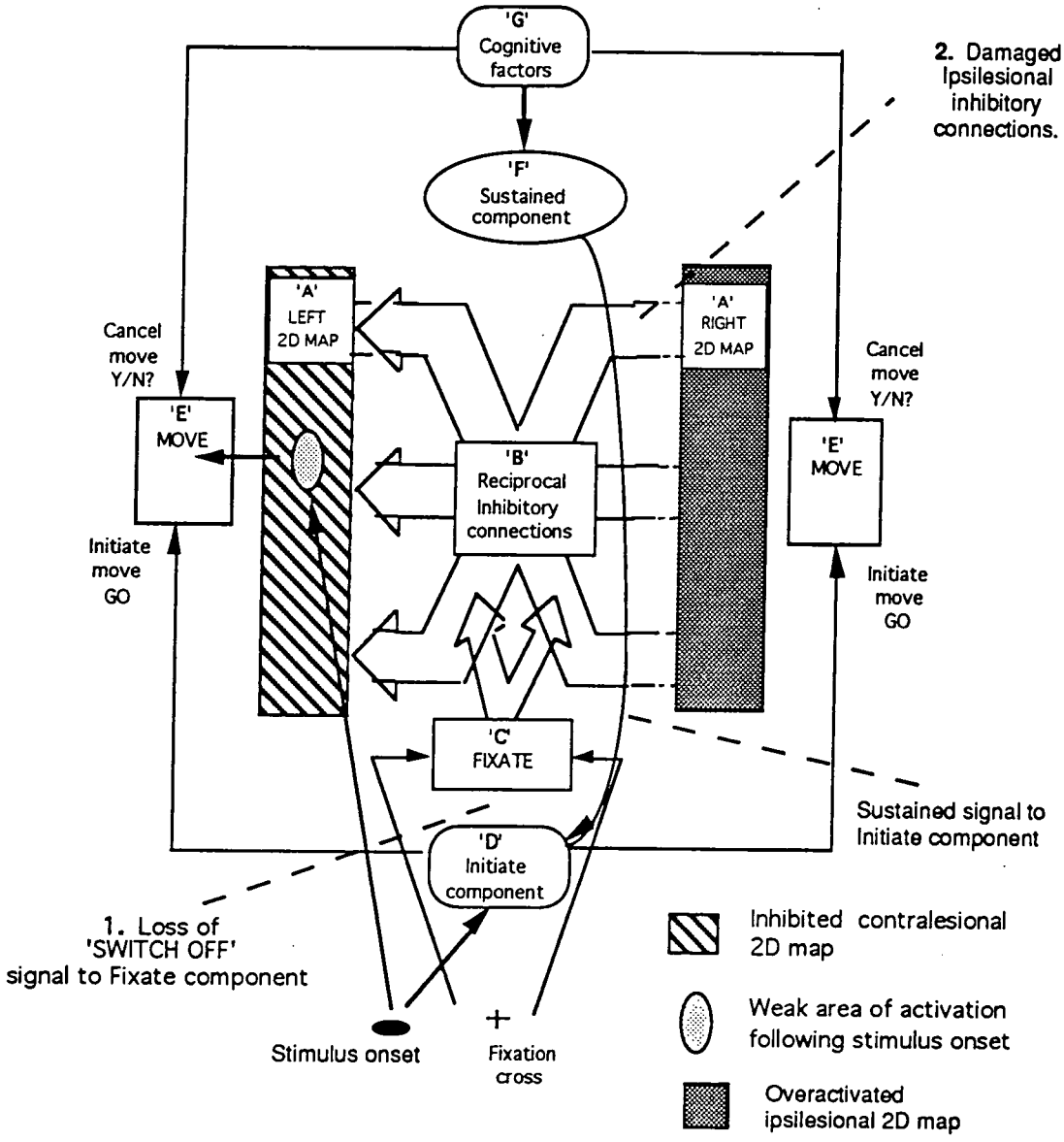


Figure 30.
The model shown with damage to specific components which may account for B.Q.'s inability to orient towards left stimuli.
The 'switch off' signal is lost (1) and the ipsilesional Reciprocal inhibitory connections damaged (2).

8.5.1. B.Q.'s failure to orient to contralesional stimuli during active fixation.

When a stimulus was presented to the left (contralesional) side of a continuously presented central fixation point B.Q. typically failed to make a saccade to, or an overt report of, that stimulus. B.Q. could overtly report and make a saccade to stimuli presented to the right (ipsilesional) side of fixation. This can be explained by suggesting that B.Q. has damage to two separate components of the premotor model. These are: the loss of the link connecting the Initiate component to the Fixate component; and the degradation of the Reciprocal inhibitory connections which project onto the ipsilesional 2D motor map.

To account for unilateral neglect the damage to the Reciprocal inhibitory connections is suggested to be selective, so that the connections which project from the ipsilesional map are intact, while the connections which direct the inhibition onto the ipsilesional map are lost/degraded. This means that the ipsilesional map is not subject to any inhibition, but a stimulus projected into the ipsilesional map will produce an area of activation (motor coordinate) and activate the Reciprocal inhibitory connections projecting to the contralesional 2D map. The loss of the Reciprocal inhibitory connections acting on the ipsilesional 2D map has two inter-related consequences. Firstly, the ipsilesional map is not subject to inhibition and will be in an 'over activated' state. The result of the higher activity level in the ipsilesional map is signalled to the Reciprocal inhibitory component which produces inhibition acting on the contralesional 2D map. A contralesional stimulus will therefore produce very little activity in the contralesional 2D map, which may not reach the threshold level required to signal the motor coordinates to the Move component. This suggestion is similar to Kinsbourne's hemispheric activity hypothesis of neglect, but instead of relying on overactivity of the whole of the contralateral hemisphere centres on overactivity within the ipsilesional component of attentional orienting system.

The damage to the link between the Initiate and Fixate components means that a stimulus onset activates the Initiation component (enabling a trigger signal to be programmed), but will not switch off the Fixate component inhibition. Attending to a stimulus (ie. a fixation point) will result in inhibition being produced from the Fixate component which cannot be turned off. As the inhibition is signalled by the Reciprocal inhibitory connections (of which the ipsilesional connections are thought to be lost) the process of fixating will produce inhibition acting on the contralesional 2D map only. The onsets of either a contralesional, or ipsilesional stimulus, can both activate the Initiate component, resulting in a normal trigger signal being sent to both Move components. A contralesional stimulus will not produce a strong area of activation in the contralesional map, so the representation of the motor coordinates are not made available to the Move component. The Move component functions as an AND gate which requires both a trigger signal and a motor coordinate so a contralesional movement will not be produced. An ipsilesional movement is possible as the ipsilesional map is not subject to the inhibition generated continuously (during fixation) from the Fixate component. The inhibition is not directed to the ipsilesional map (due to the loss of the ipsilesional Reciprocal inhibitory connections), so an area of activation representing the target motor coordinate occurs following

target onset. The onset of the ipsilesional target will activate the Initiate component and enable a trigger signal to be programmed. The presence of the initiate trigger signal and the motor command enables ipsilesional orienting.

8.5.2. A 100 ms gap improves contralesional and ipsilesional orienting.

The removal of the fixation point 100 ms before the onset of the target stimuli, improved B.Q.'s ability to overtly report and make saccades to contralesional targets. This is assumed to occur as the offset of the attended fixation point results in the Fixate component inhibition being switched off, which decreases the level of inhibition acting on the contralesional motor map (produced by the Reciprocal inhibitory connections). The level of inhibition should have fallen during the 100 ms gap, to a level that allows the contralesional stimulus to produce an area of activation in the 2D map, which signals the target motor coordinates. The presence of this activity is signalled to the Move component which can then produce a movement to that location on receiving the trigger signal.

The amount of ipsilesional stimuli reported by B.Q. also increased in the 100 ms gap condition. This increase in ipsilesional orienting could have more than one possible explanation. The first is that the offset of fixation could have a generalised alerting effect which will improve B.Q.'s ability to make any response due to an improvement in her overall level of arousal/vigilance. This increase in arousal produces a generalised increase in B.Q.'s level of performance and does not result from activation of any one specific component of the proposed model. The second possibility is that the Reciprocal inhibitory connections projecting to the ipsilesional 2D map are not completely destroyed. This would mean that the presence of the fixation point will still produce some inhibition in the ipsilesional map. The offset of the fixation point 100 ms before, the onset of the ipsilesional stimulus, could improve orienting by increasing vigilance/arousal and by reducing inhibition in the ipsilesional 2D map.

The latency of saccades made by B.Q.'s, to ipsilesional stimuli cautions against the possibility that the Reciprocal inhibitory connections projecting to the ipsilesional 2D map are partly functioning, for two reasons: Firstly, in the 100 ms gap condition B.Q.'s mean saccade latency to ipsilesional stimuli, was comparable to that obtained in the overlap condition. In contrast to the performance of normal subjects there was no facilitation effect on saccade latency with a 100 ms gap. This suggests that the rate of activity in B.Q.'s ipsilesional 2D map, rises at a comparable rate, in both the gap and overlap, conditions. Secondly, B.Q.'s mean saccade latency was not slower with bilateral simultaneous target presentation. The presence of the contralesional bilateral target should produce inhibition on the ipsilesional 2D map which would be expected to slow the saccade latency compared to the latency of saccades made to a single ipsilesional target. As B.Q.'s saccade latency was comparable with single and bilateral simultaneous target presentation, it would appear that the ipsilesional inhibitory connections are not functioning.

A further consequence of the complete loss of the ipsilesional Reciprocal inhibitory

connections is that neglect patients could produce faster responses to ipsilesional stimuli, than do control subjects. Such an effect has been reported by Ladavas, Petronio and Umiltá (1990). This is due to the ipsilesional 2D map being in an overactive state so that ipsilesional stimuli produce a fast acting representation of the motor coordinates of that stimulus.

8.5.3. A zero ms gap does not improve contralesional orienting.

B.Q. failed to make a contralesional movement when the central fixation cross offset occurred simultaneously with target onset. This failure to make a response is consistent with the idea that the inhibition generated by the Reciprocal inhibitory connection dissipates slowly following fixation offset, and takes up to 100 ms to fall back to its resting level. The removal of the fixation point will switch off the Fixate component inhibition acting on the contralesional map. However, the level of inhibition will fall off at a gradual rate from the time of fixation offset. As the saccade target appeared simultaneously with fixation offset, the contralesional target will project into an inhibited 2D map and will not produce an area of activation to signal the motor coordinates. The zero gap condition does not enable any preprogramming of the Initiate component so there will be no facilitation effect on saccade latency with ipsilesional targets.

8.5.4. Bilateral simultaneous targets improved ipsilesional orienting.

Bilateral simultaneous presentation produced a small increase in the amount of ipsilesional stimuli that B.Q. reported, compared to unilateral ipsilesional stimuli presentation. A simple explanation of this small improvement is that the two stimuli produce a small arousal effect which improves B.Q.'s level of vigilance, as suggested could also occur with fixation offset, in the 100 ms gap condition. The loss of the Reciprocal inhibitory connections projecting to the ipsilesional map, means that the presence of the target in the contralesional field does not affect the level of inhibition acting on the ipsilesional 2D map. The effect of a small increase in B.Q.'s level of arousal, without any increase in the level of inhibition acting on the ipsilesional map (as would occur in normal subjects) is to produce a small improvement in ipsilesional orienting. A possible objection to the idea that the increase in ipsilesional orienting is primarily due to an alerting effect which increases the patient level of arousal is that there was little evidence of any improvement in ipsilesional orienting with simultaneous offset of the central fixation point in the 0 gap condition. However, this could be explained by a stimulus onset (the contralesional bilateral target), having a greater alerting effect, than does a stimulus offset (offset of fixation).

8.6. B.Q.'s text reading performance.

When reading a passage of text B.Q. omitted words located on the left side of the page. In terms of the model this is consistent with the Fixate component maintaining inhibition due to the continued presence of the words on the page. In the case of B.Q. the loss of the connection between the Initiate and Fixate components will result in a failure to switch off the Fixate component. During normal reading it could be assumed that a signal is sent from the

Sustained component to the Initiate component, which indicates the end of a fixation on an individual word and produces a rightward movement onto the next word. In B.Q.'s case the signal from the Sustained component does not switch off the Fixate component, and due to the loss of the Reciprocal connections the inhibition is only directed onto the contralesional 2D map. In this instance B.Q. should be able to make an ipsilesional (rightward) movements, but will not be able to move leftwards to read the words which are located in the contralesional 2D map. B.Q.'s reading pattern indicated that she was making a small return left saccade, as she read the last 2-3 words from the next line. This is consistent with the return left movement of eyes/attention being computed within the ipsilesional map. A saccade cannot be made further into the contralesional side of space, due to the inhibition acting on the contralesional 2D map.

8.6.1. The gap effect does not improve B.Q.'s text reading performance.

The reading experiments showed that presenting single lines onto the VDU screen with relatively long gap intervals (eg. 1200 ms), did not give a great reduction in the amount of B.Q.'s left sided word omissions. In terms of the model removal of each line should switch off the Fixate component. This should reduce the inhibition acting on the contralesional 2D map and enable B.Q. to make a left saccade to locate the start of the next line. Her actual reading performance was consistent with a failure to initiate a large contralesional saccade which fell past the mid-line of the VDU screen, even with long gap intervals. One possible reason for this failure is that during the gap interval B.Q. starts to Fixate onto the ipsilesional side of the VDU screen. The process of fixating on the VDU, will again produce inhibition on the contralesional 2D map, which will inhibit a large contralesional saccade being made. A second possibility is that the lack of any indication of the location of the far left side of the screen during the gap interval (indicated by the presence of the left text/margin break in normal reading) results in a failure of the Sustained component to produce a large left saccade. Normal subjects would presumably be able to read single lines in the line-gap procedure, by using a memory of the left sided location, to produce a return left saccade during the gap interval. A deficit of the Sustained component, which prevents the use of cognitive factors is entirely consistent with the inability of B.Q. to use of top-down knowledge (such as the passage not making sense) to produce contralesional saccades. The damage to the Sustained component in this case results in B.Q. having a deficit of the memory representation of the location of the left side of the screen.

8.6.2. A stimulus flash improves contralesional orienting if presented in the gap interval.

The use of a left sided stimulus flash presented during the gap interval produced a decrease in the amount of B.Q.'s left sided word omissions. This is consistent with the idea that the stimulus flash produces an area of activation in the contralesional 2D map and generates an initiation trigger. The stimulus flash was only effective if presented during the gap interval, when it can be assumed that inhibition in the contralesional 2D map has decreased. As B.Q. still omitted some of the first words from each line, it appears that the left saccade still falls short of

the far left location. This could be because of the continued presence of inhibition within the contralesional 2D map preventing a saccade being made to peripheral left sided location. A second possibility is that B.Q. did make a saccade to the far left location, but the onset of the next line produced a saccade rightwards, so the first few words from the line are again omitted. This could occur as the onset of the line produces an Initiate signal and an area of activity in the ipsilesional 2D map. An ipsilesional saccade would occur if, the ipsilesional map is constantly overactive so that the words on the right side of the screen would quickly produce an area of activity signalling a right saccade motor coordinate. The third possibility could be that the left words of the line are scanned but not reported due to a failure of conscious awareness. This interpretation is outside the scope of the proposed orienting model. The exact position of the eye while reading would be required to examine these three possibilities.

The failure of a left stimulus flash to produce an orienting response while the line of text is present on the screen can be explained in terms of the impaired model. The presence of the line in the visual field will result in the Fixate component generating inhibition. This inhibition will be directed towards the contralesional 2D map. The stimulus flash will fall into a strongly inhibited area and will not produce an area of activity at that location. In this case the Move component will receive a trigger signal, but will lack the motor coordinates to produce the final movement. The stimulus flash will only be effective in generating a saccade when it is presented onto an otherwise blank screen.

8.7 Further implications for neglect.

Karnath (1988) suggested that visual extinction is due to the recovery of the neglect patients ability to orient contralesionally, while there remains an automatic tendency to orient ipsilesionally. In terms of the proposed model this suggests that there is some recovery of the connection from the Initiate component which switches off the Fixate component. A contralesional stimulus will activate the Initiate component and thus reduce the level of inhibition directed onto the contralesional 2D map from the Fixate component. A normal contralesional eye/attentional movement can be made when the Initiate component completes the programming of the final trigger signal. There is however, little or no recovery of the Reciprocal inhibitory connections acting on the ipsilesional 2D map. The ipsilesional 2D map remains in an over-activated state. The consequence is that bilateral simultaneous targets generate an area of activity in the ipsilesional 2D map faster than in the contralesional 2D map. This results in an ipsilesional movements of the eyes/attention when the final trigger signal is produced by the Initiate component.

8.8 Limitations of the proposed premotor model of attention.

The proposed model of attention makes the assumption that a common underlying neural mechanism is involved in orienting visual attention as is involved in producing a saccadic eye movement. If this model is to be used to account for both movements of the eyes and the

orienting of visual attention, then it should also be instrumental in describing how damage to parts of the model could result in unilateral neglect. One important consideration involves the frame of reference involved in orienting the eyes and visual attention. Models of saccade generation have suggested that the target coordinate is specified in a spatial coordinate system with respect to the head position (Sparks and Mays, 1983; Robinson, 1975) and it has also been suggested that neglect involves a deficit in deploying attention into the contralesional side of space in head/body centred (Karnath et al. 1991; Ladavas, 1987) or environmental coordinates (Ladavas, 1987). However, recent models of saccade generation involve a retinotopic representation (Findlay, 1987; McIlwain, 1986). How could a premotor model of attention explain both visual neglect and also provide a feasible model of saccade generation? One important point to realise is that the issue of the reference frames involved in the models of saccade generation and neglect are still unresolved. The experiments described in this thesis have not aimed to dissociate the retinotopic coordinates from a head/body spatial coordinate system. For this reason some of the relevant points regarding the coordinates involved in saccade generation and implicated in neglect will be mentioned in the next section.

8.8.1 The coordinates involved in models of saccade generation.

The foveation hypothesis of saccade generation (Schiller and Koerner, 1971; Robinson, 1972) stated that the superior colliculus contains retinotopic motor maps which bring the fovea onto a target, the retinal error signal being represented in the retinotopically organised layers of the superior colliculus. However, there is evidence that visual targets are represented in non-retinotopic coordinates. Hallett and Lightstone (1975 ab) showed that saccades can be made to targets located in spatial rather than retinal coordinates. The models of saccade generation proposed by Robinson (1975) suggest that saccades are goal directed and made towards a stimulus location specified in head centred coordinates. The coding of stimulus location in head centred coordinates will require a signal of the position of the eyeball in the head to perform the transformation from retinotopic, to spatial coordinates. Sparks (1986) suggested that the superior colliculus is in a position to perform such a transformation. Sparks current view of the role of the superior colliculus is that it contains a representation of the motor coordinates required to move the eyes to the stimulus position. These motor maps are thought to be dynamic and move with changes in the position of the eye, head, or body (Sparks, 1986; Sparks and Hartwich-Young, 1989).

The superior colliculus is not the only region of the brain involved in producing saccadic eye movements. Areas of the cortex such as the frontal eye fields (Goldberg and Bushnell, 1981) and parietal lobe (Andersen, 1989; Andersen and Gnadt, 1989; Mountcastle et al., 1975) when stimulated can also result in a saccade being produced; although their ability to do so depends on an intact superior colliculus (Schiller, 1977). The parietal lobe and frontal eye fields are thought to contain non-retinotopic representations of visual space. Andersen et al. (1985) showed that the receptive fields of parietal cells moved with the eyes (retinotopic), but the responsitivity varied as the eyes moved. The conclusion is that the parietal cortex contains a

non-retinotopic map of space which may be involved in generating motor commands and saccadic eye movements. The role of the frontal eye fields is thought to be involved in producing saccades to remembered locations and the production of saccades without a stimulus change in the visual field from voluntary control

Although, there is a large body of evidence to suggest that the superior colliculus and the cortical areas involved in saccade generation, contain spatial representations of the visual scene, some recent models have relied on a retinotopic coordinate system. Findlay (1987) asserted that the translation of retinocentric coordinates, to spatial coordinates (within a two dimensional representation) is a formidable computational problem. The models proposed by McIlwain (1986) and Findlay (1987) suggest that the retinotopic map of the target position is transformed into a retinotopic motor map coordinate in the deep layers of the superior colliculus.

The models of saccade generation described above have incorporated either retinotopic, or spatial, coordinate frames of the target stimulus. The issue of the coordinate system is still thought to be open to question, although current models of the superior colliculus favour a retinotopic motor map of stimulus coordinates, saccade generation is also known to involve the parietal and frontal cortex thought to contain non-retinotopic maps of visual space. This suggests that saccade generation could well involve both retinotopic and spatial coordinate systems.

8.8.2 The coordinates involved in unilateral neglect.

The issue of the frame of reference in neglect was discussed in Chapter five. The conclusion was that the evidence shows that neglect reflects a deficit of attentional orienting that could involve one or more than one level of representation. A viewer centred representation could represent space in terms of the two visual fields, or in terms of the left and right sides of head space or in terms of the left and right sides of body trunk space. Although neglect is not thought to operate in terms of visual fields, it is possible that the neglect condition produces an impairment of attentional orienting at the retinotopic level of representation which impairs the allocation of attention into the contralesional hemifield, as well as a deficit of orienting attention into the contralesional side of head/body space (Ladavas, 1987; Karnath et al., 1991). There is evidence to show that neglect impairs the orienting of attention into the contralesional side of space coded in terms of an environmental (allocentric), representation (Farah et al., 1990) and also impairs orienting at a higher level object centred representation (Driver and Halligan, 1991). Given that neglect appears to produce an impairment in orienting attention in more than one representation of space, there could be separate impairments affecting orienting in these separate representations. The proposed premotor model of attention aimed to describe the results obtained from the investigation of B.Q.'s attentional impairments. It is possible that one part of her overall condition is a deficit in the deployment of attention within a retinocentric system which is involved in producing a saccadic eye movement. There is evidence (Young et al. 1992) that B.Q. is also impaired at orienting attention within other

representations, which could involve a similar models of attention orienting to that proposed which use a different coordinate system in the motor maps.

The proposed model is therefore limited in that it cannot account for all of the illustrations of neglect indicated even in a single patient like B.Q. One important factor to be considered in neglect is that the patients also have a reduced level of non-specific vigilance and arousal (Karnath, 1988; Robertson and Frasca, 1992). This is a further, but separate, factor to be considered along with the damage to the proposed eye/attentional orienting system. The model is also poor at providing an explanation of B.Q.'s failure to recognise the left sided feature of an object when it is presented entirely within her right visual field (Young, et al. 1992). Given that neglect could affect more than one level of representation of the visual world it could be hypothesised that deficits of this kind reflect damage to a separate system which is involved in deploying attention within an object centred reference frame. The present model does not claim to account for all of these possible impairments.

There are many difficulties in trying to ascribe a physiological basis onto a functional model such as that put forward in this chapter. However, the proposed model is supported by some of the known physiology of the saccadic system. The 2D motor maps suggested in the model are based on the ideas of McIlwain (1976; 1986) and Findlay (1987) which are thought of as being realised in the superior colliculus. The possibility that the superior colliculus could be involved in an inhibitory system as suggested in the present model has been implicated in recent work recording the discharge rates in the colliculus of awake monkeys (Munoz and Wurtz, 1992). Munoz and Wurtz (1992) showed that a subset of neurons discharged while the monkeys were fixating and inhibiting the discharge of these neurons (GABA injection) resulted in the monkeys producing faster saccades. This suggests that cells within the superior colliculus are involved in maintaining active fixation by inhibiting the production of a saccade. The presence of inhibition acting within the superior colliculus has also been indicated by Rizzolatti et al. (1974). They showed that presenting a stimulus in one part of the cats superior colliculus reduced the responsitivity to a stimulus presented at other areas within the same visual field. This evidence is consistent with the proposed model that uses the idea of inhibition acting within the motor maps to suppress an eye movement.

A further implication of the recent finding of Monoz and Wurtz (1992) for the proposed model is that there may not be a separate Fixate component. If active fixation causes activation of a specific subset of neurons in the superior colliculus that inhibit the production of a saccade, then the idea of Fixate inhibition can be accommodated into the 2D map itself. There is no need to suggest that a separate component is involved in producing the Fixate inhibition. However, in terms of the functioning of the model described in this chapter it does not matter if the Fixate inhibition is shown pictorially as a separate component.

Neglect is typically associated with parietal lobe damage. As the parietal lobe is connected to the superior colliculus it could could be expected to exert some influence on the saccadic system. A large body of evidence indeed shows that the parietal lobe has a role in

saccade generation and visual fixation (See: Hyvärinen, 1982; for review). Andersen (1989) suggested that the functional role of the parietal cortex is to programme a saccadic eye movement based on a sensory signal. The transformation of the retinotopic information is transformed into spatial and motor coordinates (See: Andersen and Gnadt, 1989). The parietal lobe is also extensively connected to other cortical regions (Hyvärinen, 1982) and as such could enable an interaction between the higher level cognitive system and the saccadic system. The role of the parietal lobe has in producing neglect and extinction would suggest that it has a functional involvement in the sustained orienting of the eyes/attention.

Chapter 9

Conclusions.

The aim of this thesis was to examine the orienting of visual attention by an examination of overt saccadic eye movements. This was performed with normal subjects and a patient with a condition thought to reflect a deficit of visual attention termed 'unilateral spatial neglect'. The results obtained have been discussed in terms of a tentative model of the attentional/eye movement system. The following section provides an overview of the main conclusions from this thesis, with relation to the model.

The examination with normal subjects centred on an examination of the time taken to make a saccade under conditions in which attention was directed (cued) to spatial locations. These experiments showed that directed visual attention produces a small facilitatory benefit for saccades made to targets presented in the attended regions of space. The latency of saccades made to targets presented in the non-attended regions were subject to much greater costs. A comparable pattern of results was obtained when attention was directed along either the horizontal, or the vertical axis, emphasising that model of the attentional/eye orienting system needs to be able to account for the deployment of attention in two dimensional space. Whereas most current models of visual attention emphasise the facilitatory effect of attention, these results suggest that the inhibitory consequences should also be considered.

When two saccade targets were presented bilaterally and simultaneously in opposite hemifields the effect was a slowing of saccade latency. This was interpreted as reflecting an automatic inhibitory effect where a stimulus onset produces inhibition for the region contralateral to that in which the stimulus was presented. The spatial extent of this inhibition may be broadly distributed over the whole of the contralateral spatial location and could also involve an area of mirror symmetric inhibition. The idea of reciprocal inhibition was incorporated into the proposed model. Presenting the bilateral target at intervals over 160 ms *before* the saccade target was found to have a facilitatory effect on the latency of saccades made to a target presented in the opposite direction. A possible explanation of this facilitation was that it reflects preprogramming of the non-spatial component of the model that triggers 'when' the saccade should be made. Such an explanation requires the additional assumption that the inhibition produced by the

early onset of the target has decayed by some 100 ms, after the offset of the initial bilateral target. The overall result being a facilitation effect on the production of the orienting response.

The gap paradigm was used and was found to give a speeding of saccade latency, both for saccades made to targets in the attended and non-attended directions. The implication is that the gap effect has a generalised speeding effect on the production of a saccade, which is independent to the facilitatory and inhibitory consequences of directing attention by the sustained attentional system. The findings from the gap experiment support models which suggest that part of the facilitation is spatially non-specific. The facilitation could reflect a warning signal effect that enables preprogramming of the decision process 'when' to execute a saccade, in addition to prior attentional and oculomotor disengagement. The act of attending to a fixation point can be incorporated into the proposed model, by suggesting that attending to a fixation point results in inhibition for all of the non-attended locations. Fixation offset reduces this inhibition and speeds the time taken to initiate a saccade. In the proposed model the idea of '*attentional disengagement*' is the equivalent to the switching off of this inhibition, a process that is assumed to take a certain length of time to complete.

The experiments with the neglect patient B.Q., showed that when she was attending to a central fixation point she could not orient her eyes towards, or overtly report, contralesional stimuli. Prior removal of the fixation point enabled her to make a contralesional saccade and improved her ability to report the presence of contralesional targets. The first implication of this result is that B.Q. does not have a left hemianopia as indicated by perimetric field plotting techniques. Secondly it appears that B.Q.'s contralesional neglect can be explained in terms of a deficit of attentional disengagement. The basis of B.Q.'s deficit was outlined in detail with relation to specific components of proposed orienting model. It was suggested that B.Q.'s neglect is due to: firstly, an inability to suppress the inhibition produced by attending to a fixation point; and secondly, a loss of the inhibitory connections acting on the ipsilesional motor map. The damage to these components enables a normal ipsilesional movement to be made and accounts for the inability to orient contralesionally during fixation. The model also accounts for the lack of a facilitation effect on ipsilesional saccades in the gap situation, and the lack of the inhibitory consequences of presenting bilateral saccade targets. The loss of inhibition acting on the ipsilesional map is consistent with the underlying basis of an automatic tendency for patients to orient in the ipsilesional direction, which is consistent with the presence of *visual extinction,*

often shown in patients that have recovered from profound neglect. The account of neglect is consistent with many recent attentional models of neglect, and develops the idea further by showing what a deficit of disengagement is in a premotor model of attentional orienting.

The proposed model is useful in providing an account of the underlying basis of visual attention in relation to the saccadic system. The model incorporates the idea of attentional inhibition and moves away from older views (such as the attentional 'spotlight') which have suggested that attention operates by facilitating a spatial region. The model can account for a large body of results obtained in saccadic eye movement experiments, as well as providing a plausible account of one of the deficits shown by a neglect patient.

Appendix

A.1 Saccade detection and calculation of saccade latency.

The presence of a saccade was detected using the following algorithm. The amplitude difference between each data point of the digit record was calculated. If the amplitude difference between two points, N and N1 was within a preset threshold then the algorithm moved on to sample the next points. However, if the difference between N and N1 exceeded a the threshold then the algorithm calculated the difference between the points N and N2. If this difference exceeded the threshold then the algorithm again checked the difference between points N and N1, if the difference was found to exceed 25% of the threshold then the algorithm designated point N as the start of the saccade. Saccade latency was calculated as being the number of data points from 0 to N (where 0 represents the time of saccade target onset) multiplied by the sampling rate of the ADC.

Each digital record was then displayed onto a VDU monitor (Figure 31) with a cursor indicating the position that the algorithm had designated as being the start of the saccade, an example of a saccade record is illustrated below. At this stage it was possible to alter the position of the cursor manually using the keyboard and to discard some records as being 'bad' data. A record containing a blink, or evidence of more than one saccade being used to detect the target would be discarded and not included in the analysis. If the saccade detection algorithm had wrongly positioned the cursor due to noise in the record then the experimenter could decide to manually reposition the cursor if it was thought that the noise was not excessive. The decision to accept such a record was largely a subjective one. The cursor was placed at the start of the saccade on the basis of the visual analysis of the displayed record.

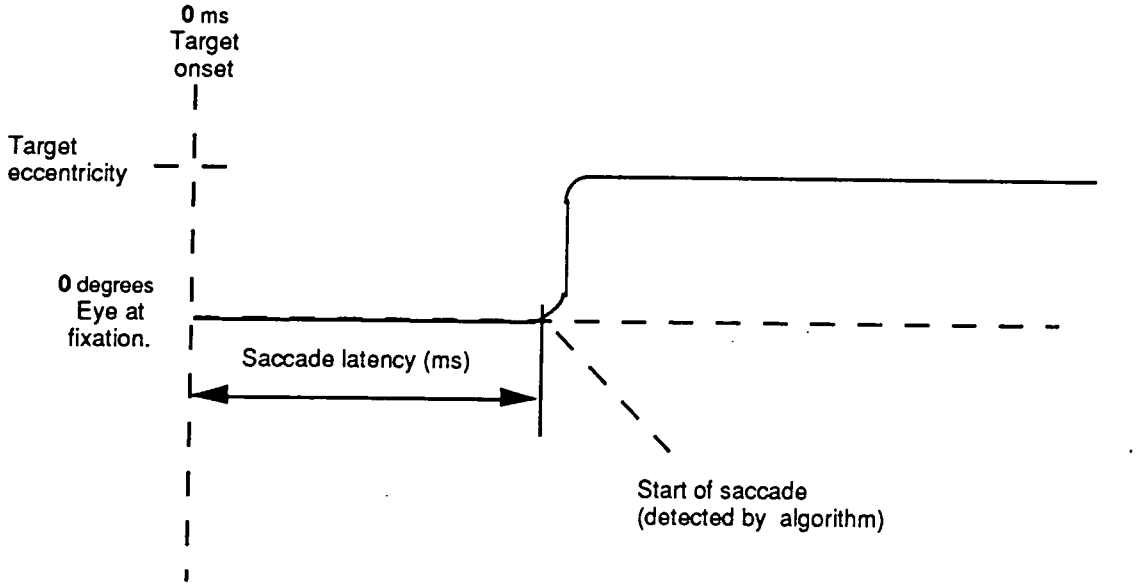


Figure 31.
Illustration of a record of a saccade made to a target and the latency of that saccade.

A.2. The format of the passages of text used in Experiments N5 and N6.

The passages of text used in the examination of B.Q.'s whole word omissions were modified to conform to a particular format. Each passage consisted of thirteen lines and each passage was balanced so that the same amount of words were contained in each of the lines. The number of words that each line should contain is shown in brackets. These numbers were not displayed during the experiment. A sample passage is shown in Figure 32.

IN THE NORTH EAST CORNER (5)
OF THE CONGO LIES A VAST (6)
EXPANSE OF DENSE, DAMP, DARK FOREST. (6)
PUT YOUR FINGER IN THE MIDDLE (6)
OF A MAP OF AFRICA AND IT (7)
WOULD NOT BE FAR AWAY. HERE IS (7)
THE HEART OF THE COUNTRY THAT (6)
STANLEY, LOVED AND HATED. THE SCENE (6)
OF HIS ILL-FATED EXPEDITION, WHICH (6)
COST HUNDREDS OF LIVES AND (5)
IMPOSED ALMOST UNBEARABLE HARDSHIP ON (5)
THE MEN WHO TREKKED ACROSS THE GREAT (7)
FOREST NOT ONCE, BUT THREE TIMES. (6)

Figure 32.
Example of a passage used in the examination of B.Q.'s whole word omissions.

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