

Revisiting unilateral neglect.

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Abstract

Unilateral neglect, a neurological disorder in which patients fail to detect or respond to contralesional stimuli, has long been considered a failure of attentional orienting mechanisms. This review provides a selective overview of the prominent biases in spatial orienting and exploratory motor behaviour observed in these patients before considering the impact of other factors on the presentation of the disorder and how those factors might inform current neurological models of neglect. In the latter part of the review we intend to suggest that neglect is likely to be a combination of distinct but interacting impairments including biases in attentional orienting, exploratory motor behaviours and a deficit of spatial working memory. That is, we suggest that the cardinal symptom of neglect – a loss of awareness for contralesional stimuli and events – arises as a result of a combination of these impairments rather than being associated solely with the more dramatic and immediately evident biases in spatial attention.

Key Words: Unilateral Neglect, Spatial Working Memory, Attention

1. Introduction

Unilateral neglect is traditionally defined as a failure to report, respond to or orient towards stimuli in contralesional space (Heilman, Watson & Valenstein, 1993; Driver & Mattingley, 1998; Halligan, Fink, Marshall & Vallar, 2003). Perhaps a more appropriate description, especially for severe neglect patients, would be to suggest that the patient behaves as if one half – the contralesional half – of their world has simply ceased to exist (Mesulam, 1981). The contrast between the two descriptions is not trivial. The first emphasizes the immediately obvious spatial biases in attention and exploratory motor behaviours of the patient. Failing to eat from one half of a plate of food, bumping into objects and people in one half of space and in general, shifting their posture, gaze and gait towards ipsilesional space (see Halligan et al. 2003 for review). The second description, at first blush, seems to be rather vague in that it emphasises only the loss of awareness for all things contralesional. What is compelling about this description, however, is that this loss of awareness is often evident even in the face of orienting behaviours directed towards the same region of space. That is, despite moving their eyes or hand towards contralesional space or the contralesional side of a centrally presented object, the patient may nevertheless be unaware of what they have just explored (Ferber, Danckert, Joanisse, Goltz & Goodale, 2003; Walker & Findlay, 1997; Young, Hallowell & Welch, 1992). We would not suggest that one description of the disorder is superior to the other. Instead this review intends to explore the ways in which the subtle differences briefly outlined above may inform neurocognitive models of the neglect syndrome.

1.1 The classic case of neglect.

Neglect is most commonly seen after right hemisphere lesions, leading the patient to behave as if the left half of their world has ceased to exist. We will briefly return to the issue of the critical lesion location for neglect later in this review, but for now will talk about neglect of left space following right hemisphere damage.

The tasks used to examine neglect typically require perception of and responses towards both ipsilesional and contralesional stimuli. For example, cancellation tasks – perhaps the most widely used bedside test of neglect – require the patient to place a mark through target objects (e.g., stars), presented on a sheet of paper aligned with the patient's own body midline (Ferber & Karnath, 2001). Similarly, in the line bisection task the patient is presented with a horizontal line, and is asked to place a mark on the line at a point they think represents the centre of the line (Binder, Marshall, Lazar, Benjamin, & Mohr, 1992). Finally, figure copying or free drawing tasks require the patient to draw, either from a model (copying), or from memory (free drawing), objects that are generally symmetrical around the midline (e.g., a typical free drawing task would be to draw a butterfly; Halligan, Marshall & Wade, 1989). So while figure copying and free drawing tasks may not explicitly invoke representations or responses tied directly to the patient's body midline, they do require that perceptual processing and responses be directed towards both the left and right half of the image (i.e., either the direct model or an internally generated image). Typically, the patient fails to cancel targets on the left of the page, places their midline mark to the ipsilesional side of the true centre of lines in the bisection task and omits or distorts aspects of drawings on the contralesional side of space (Figure 1).

--- insert Figure 1 about here ---

Any clinician accustomed to assessing neglect patients will be all too familiar with the fact that few patients fit this text book description perfectly. Some will demonstrate predominantly personal neglect, failing to dress the left side of their body or shave the left side of their face, while others will show their most severe deficits on tasks of extrapersonal neglect, failing to respond to events or objects beyond personal space (Beschlin & Robertson, 1997; Bisiach, Perani, Vallar, & Berti, 1986; Cowey, Small & Ellis, 1994; Halligan & Marshall, 1991; see also Berti, Smania & Allport, 2001 and Pegna, Petit, Caldara-Schnetzer, Khateb, Annoni, Sztajzel, & Landis, 2001 for a discussion of how tool use can modify personal and extrapersonal neglect). Neglect can also be observed for stimuli defined strictly by their location in space or alternatively, patients may neglect the left half of objects irrespective of their location in space (although the two types of impairment can co-occur within individual patients; Driver, Baylis, Goodrich & Rafal, 1994; Tipper & Behrmann, 1996). Furthermore, ‘sub-syndromes’ of neglect symptoms, such as neglect dyslexia in which the patient fails to read the left half of words, are present in some but not all, neglect patients (Ladavas, Shallice, & Zanella, 1997; Miceli & Capasso, 2001; Vallar, Guariglia, Nico, Tabossi, 1996). Finally, the related disorder of extinction to double simultaneous stimulation in which patients can detect single targets presented in left or right space but ‘extinguish’ left targets when presented simultaneously with right targets, is often but not always evident in neglect patients (see Rafal, 1996 for review).

Perhaps this plethora of symptoms and the vagaries of their combinations within individual patients has been the cause of the difficulty in finding or developing conclusive neurocognitive models of the disorder. Important aspects of the disorder that tend to be evident in most patients diagnosed with neglect are a loss of awareness for contralesional events (demonstrable on at least some subset of the clinical tests of the disorder and in at least one frame of reference – personal, peri-personal or extrapersonal) and a shift in exploratory behaviours (e.g., attentional shifting, eye movements) towards ipsilesional space. In this review we will examine some of the more prominent demonstrations of the latter constellation of symptoms (i.e., a shift in exploratory behaviours) before then exploring the role played by spatial working memory in the disorder and how a combination of impairments of spatial working memory, exploratory motor behaviours and attentional orienting may inform models of neglect.

2. Exploratory and goal-directed motor behaviour in neglect.

One immediately obvious aspect of the presentation of some patients with severe neglect concerns their posture. Patients who are wheelchair bound tend to slump towards the ipsilesional side of their chair and direct their gaze towards ipsilesional space (Karnath, 1997). The latter observation can be so severe that the patient's head and eyes are deviated towards ipsilesional space requiring some effort to coax them into looking straight-ahead, much less orienting towards contralesional space. For patients capable of weight-bearing, an imbalance in the amount of pressure placed on each foot has been observed, with more weight being placed over the right foot (Tilikete, Rode, Rossetti, Pichon, Li, & Boisson, 2001). When directly tested, their judgement of a subjective point

in space straight ahead of their body midline is also biased towards ipsilesional space (Ferber & Karnath, 1999; although see Chokron & Bartolomeo, 1997 for a detailed discussion of factors, such as starting position of a responding hand, that influence this behaviour). That is, if the neglect patient is asked to determine where they think straight-ahead of their body is in the absence of any external reference frame, they typically demonstrate a deviation towards the right of an objective midline position (i.e., relative to the patient's own body midline; Ferber & Karnath, 1999; Rossetti, Rode, Pisella, Farné, Li, Boisson, Perenin, 1998). Interestingly, patients seem to anchor their motor behaviour around this shifted straight ahead position as if this were the new default centre for exploratory eye and hand movements (see below). Also, reaction times to visual stimuli were found to be fastest at a mid-periphery location on the ipsilesional side (Smania, Martini, Gambina, Tomelleri, Palamara, Natale, Marzi, 1998).

More subtle impairments of motor control can also be observed in neglect. For example, when patients are required to make pointing movements towards targets using their unimpaired, ipsilesional limb, they nevertheless demonstrate slower RTs for leftward movements made in either left or right visual space (Husain, Mattingley, Rorden, Kennard, & Driver, 2000; Mattingley, Husain, Rorden, Kennard, & Driver, 1998; Mattingley, Bradshaw & Phillips, 1992; Mattingley, Phillips, & Bradshaw, 1994). The bias is further exaggerated for leftward movements made *towards* left visual space (Husain et al. 2000; Mattingley et al. 1998). Taken together, these results suggest that the initiation and execution of leftward movements can be impaired in left neglect patients even in the absence of any overt spatial distortions of the movement trajectory¹. While

¹ There have also been demonstrations of curved reaching trajectories in patients with right hemisphere damage (Goodale et al. 1990) that have proven somewhat difficult to replicate.

the control of visuomotor actions is deficient for leftward movements in neglect patients, they are nevertheless *capable* of making such goal-directed movements. That is, when required to make a goal-directed pointing movement towards a single target in left hemispace the patient is generally able to acquire that target (Mattingley et al., 1994; Husain et al., 2000).

A very different picture is observed when patients with neglect are asked to explore space, either with their eyes or via hand movements (Karnath & Niemeier, 2002; Himmelbach & Karnath, 2003). In these instances, although the patient will make as many leftward as rightward eye or hand movements, the region of space to which they direct their eye (or hand) is dramatically shifted and constricted relative to controls. Typically, the patient explores a region of space largely to the right of an objective midline as defined by trunk position (Karnath & Niemeier, 2002; Himmelbach & Karnath, 2003). So while a patient with neglect may be *capable* of directing purposeful acts towards single objects or locations in any region of space, their default setting for *exploring* extrapersonal space is dramatically shifted toward the ipsilesional side.

3. Disengaging attention in neglect.

It is beyond the scope of this review to give a comprehensive account of all the attentional biases observed in neglect. However, we feel it is important to outline one of the more prominent disturbances in orienting behaviour seen after right hemisphere brain lesions that has been invoked as an explanation for the disorder of neglect. While we typically explore our surroundings with overt movements of the eyes (or hands), we can also redirect our attention covertly (i.e., while the eyes remain centrally fixated) toward a

location in space. In a typical task examining covert shifts of attention a cue is presented directing the patient's attention towards one location or another at which an upcoming target will soon appear (Posner, Nissen, & Ogden, 1978). On some trials, the cue accurately indicates the target location (valid trials) while on other trials the target appears on the opposite side of space (invalid trials)². Posner and colleagues (Posner, Walker, Friedrich & Rafal, 1984, 1987) demonstrated that patients with right parietal lesions show similar reaction times (RTs) for validly cued contralesional and ipsilesional targets, indicating that they were able to orient attention to either side of space with equal success. On invalid trials, however, when the cue was presented to the ipsilesional side and the target appeared on the contralesional side, the parietal patients showed abnormally long RTs. Posner and colleagues (1984) concluded that damage to the parietal lobe leads to a deficit in disengaging attention from the ipsilesional side. This so-called 'disengage deficit' (sometimes referred to as an 'extinction-like' pattern of RTs) was present in patients with either left or right parietal lesions, although the deficit was larger for the right parietal patients.

Although the patients described above did not present with neglect at the time of testing, the lateralized attentional bias associated with parietal lesions led some researchers to suggest that a 'disengage deficit' was the underlying mechanism of neglect (Morrow & Ratcliff, 1988; Robertson & Eglin, 1993). Subsequent research has demonstrated a disengage deficit in neglect patients that is especially evident when exogenous or reflexive attentional mechanisms are engaged (see Bartolomeo & Chokron,

² Cuing can be achieved via an exogenous stimulus (e.g., abrupt luminance increase of a peripheral landmark; see Collie et al., 2000 for review) or via a centrally presented symbolic stimulus (e.g., an arrow indicating a particular direction; see Egeth & Yantis, 1997 for review). These different methods of cuing are thought to engage distinct attentional mechanisms.

2002 and Losier & Klein, 2001 for reviews). A similar disengage deficit has also been observed in visual search tasks which may reflect more closely the attentional demands of the real world. For example, Egly and colleagues (1989) tested neglect patients on a conjunction search task (Treisman & Gelade, 1980; Treisman & Gormican, 1988) and found that search times for contralesional targets increased with increasing number of *ipsilesional* distractors, indicative of a difficulty in disengaging attention from ipsilesional stimuli in order to reorient attention to contralesional space (see also Mark, Kooistra, & Heilman, 1988).

The effect of ipsilesional cues (or distractors) on detection times (or search times) for contralesional targets suggests that information in the non-neglected hemifield influences the ability to detect or respond to contralesional stimuli. While this kind of deficit may help explain the fact that patients with neglect fail to explore spontaneously the left half of space, it does not explain the failure to detect targets in ipsilesional space (Fig. 1). That is, although less dramatic than their impairment for contralesional space, neglect patients commonly fail to detect targets in ipsilesional space. One potential factor at play here could be the observed deficits in temporal aspects of attention in neglect patients. That is, although the most obvious and dramatic impairments in patients with neglect are seen in spatial behaviours, recent research has also demonstrated substantial impairments on tasks assessing temporal aspects of attention (Husain, Shapiro, Martin & Kennard, 1997; Rorden, Mattingley, Karnath & Driver, 1997). For example, the so-called ‘temporal order judgement’ (TOJ) task requires patients to determine which of two targets presented to the left and right of a central fixation point appeared first. For healthy controls the subjective point of simultaneity – the point at which participants respond

‘left’ or ‘right’ around 50% of the time – coincides nicely with the objective point of simultaneity. That is, when the left and right objects are presented simultaneously controls will report the left target as having appeared first on around 50% of all trials. However, neglect patients require the leftward target to precede the right by around 250 ms before they judge that both targets have appeared simultaneously (Berberovic, Pisella, Morris, & Mattingley, 2004; Robertson, Mattingley, Rorden & Driver, 1998; Rorden, et al. 1997)³. While these results are indicative of impaired allocation of attention to temporal events, the procedure still involves an inherent spatial component. In a non-spatial test of temporal attention, patients are required to attend to a rapid stream of stimuli presented centrally and must detect one or two targets appearing within that stream (Husain, et al. 1997). For healthy controls, the ability to detect the second target is quite poor when it is presented in close temporal proximity (~100 – 500 ms) to the first target (Chun & Potter, 1995; Raymond, Shapiro & Arnell, 1992). This refractory period after the identification of target one in which target two is poorly identified, is referred to as an ‘attentional blink’ (Raymond et al. 1992). In patients with unilateral neglect the attentional blink is substantially larger than in controls (Husain et al. 1997; Shapiro, Hillstrom & Husain, 2002). That is, for these patients target two is poorly identified when it appears up to one second or more after target one – double the attentional blink of healthy controls (see also Shapiro et al. 2002).

The performance of neglect patients on both the TOJ and attentional blink tasks suggests that there are severe limits on their ability either to allocate attention over time or to disengage attention in a timely manner regardless of location in space. One potential

³ Note that the Rorden et al. 1997 study involved patients who demonstrated extinction to double simultaneous stimulation.

explanation for this impairment may involve decreased levels of arousal commonly observed in neglect patients. In a recent review of the non-spatial impairments inherent in neglect, Husain and Rorden (2003) suggested that temporal deficits of the kind discussed above serve to exacerbate the spatial problems characteristic of the neglect. Indeed, if patients demonstrating the characteristic impairment on the TOJ task are presented with spatially non-predictive auditory cues prior to making their judgements, performance improves dramatically (Robertson, et al., 1998). In other words, merely by providing an alerting cue for the patient that presumably increases their level of arousal (the cue was a loud noise), performance can be improved. The question remains, however, how deficits in allocating attention over time can explain the commonly observed failure to cancel targets in right visual space – the putatively non-neglected region of space. One could argue that once attention has been allocated to a target in a rapid stream of stimuli (i.e., as is the case in the Attentional Blink task) that those resources are not able to be fully marshaled for detecting a second target for a prolonged period of time, regardless of where that target is in space. In contrast, cancellation tasks are static displays in which the patient's performance is unimpeded making such an explanation less plausible for poor performance in detecting right visual targets. Below we will discuss an alternate hypothesis to explain the failure to detect ipsilesional targets.

4. Lost in space – spatial working memory impairments and the neglect syndrome.

When completing cancellation tasks many patients with neglect cancel the same target multiple times (Figure 1B). It is as if the patient is unaware of the mark they themselves had previously made and revisit the target as if it were new again. This very

common clinical observation in neglect patients suggests that they have a deficit in spatial working memory (see Na Adair, Kang, Chung, Lee, & Heilman, 1999 and Rusconi, Maravita, Bottini, & Vallar, 2002 for an alternative account). Two recent single-case studies have demonstrated just such an impairment to working memory functions in neglect (Husain, Mannan, Hodgson, Wojciulik, Driver & Kennard, 2001; Wojciulik, Husain, Clarke & Driver, 2001). Both groups used visual search paradigms somewhat akin to the cancellation tasks used clinically and found that neglect patients repeatedly revisited previously explored locations as if they were treating these 'old' locations as 'new'. Interestingly, this behaviour was also evident in the pattern of eye movements made by the patients, with many targets being repeatedly fixated despite instructions to look at targets only once (i.e., to avoid fixating previously viewed targets; Husain et al., 2001). More recently, Pisella and colleagues (2004) used a change detection paradigm in which patients were asked to report changes in previously determined stimulus attributes including location, shape and colour. They found that neglect patients were more impaired in detecting changes in target location relative to their ability to detect changes in target colour or shape. Importantly, this difference was most evident when a one second delay was introduced between stimulus presentation and response. Finally, recent work has adapted cancellation tasks for presentation on a touch screen allowing for manipulation of target characteristics post cancellation. For example, once a target had been touched (i.e., cancelled) it could then be dimmed or even eliminated from the display entirely (Parton, Malhotra, Nachev, & Husain, 2004; see also Wojciulik, Rorden, Clarke, Husain & Driver, 2004). Patients revisited previously marked target locations far less often if the target itself had been eliminated from the display after the initial mark.

This is consistent with earlier work showing that extinguishing targets once they had been cancelled also led to fewer omissions of left-sided targets compared with conditions in which the targets remained present in the display throughout (Mark, et al., 1988).

The findings discussed above could suggest that patients with neglect suffer from a spatial working memory deficit – a failure to mentally maintain visited locations. There are, however, several alternate interpretations that warrant further consideration. First, when patients revisit ‘old’ or previously marked locations it is unclear whether or not this is due to a working memory problem per se or a problem related to the programming of successive eye movements (Duhamel et al., 1992a, Colby & Goldberg, 1999). In all the studies discussed above, the target displays (and any accompanying distractor stimuli) extend along the horizontal axis to the left and right of the patient’s body midline, much the same as the clinical tests of cancellation. Therefore, these visual search tasks require the planning and execution of many horizontal eye movements. It is possible then, that the patients suffer from an impairment in the ability to ‘remap’ space as a consequence of previously executed saccades (Pisella & Mattingley, 2004). This hypothesis is derived from research using the double-step saccade paradigm (Duhamel et al., 1992a, 1992b) in which two targets for successive saccades are presented and extinguished within 180 ms – too short a period of time to direct eye movements to both targets prior to them being removed from the display. In this task, if subjects based their saccades on retinal signals alone they would exhibit substantial errors when attempting to acquire the second target. Instead, subjects anticipate the outcome of the first saccade and program their second saccade based on the anticipated end point of the first. This process has been termed saccadic remapping and has been shown to be disrupted in at least one neglect patient

with a large right fronto-parietal lesion (Duhamel et al., 1992a). Furthermore, this patient's impairment was direction specific. That is, the patient was unable to acquire accurately the second target only when the target for the first saccade was in left visual space and the second saccade was to be made towards a target in right visual space. In a subsequent study, patients with either right or left posterior parietal lesions were shown to be impaired on the double-step saccade task in conditions in which the second saccade crossed the midline (Heide, Blankenburg, Zimmermann, & Kömpf, 1995). That is, patients demonstrated larger errors in their final eye position if the second saccade to be made was from a right visual field target to a left visual field target or vice versa (Heide et al., 1995). In addition, patients with right parietal lesions also exhibited larger errors for double-step eye movements made to targets presented entirely within the left hemifield (Heide et al., 1995).

A saccadic remapping problem of the kind described above may help explain why neglect patients tend not to explore the left half of the displays in visual search and cancellation tasks. Indeed, Pisella and Mattingley (2004) recently proposed an account of the neglect syndrome suggesting that a spatial (not simply saccadic) remapping impairment is at the heart of the disorder. They suggest that neglect is a combination of a pathological gradient of attention, such that patients direct their attention more towards the ipsilesional side of space, coupled with a deficit in spatial remapping. There are several key components to the remapping deficit they propose. First, the spatial remapping deficit inherent to neglect can occur for both overt and covert shifts of attention. Second, for neglect patients, directing attention to the contralesional field leads to a problem in remapping the entire visual space, while directing attention ipsilesionally

leads only to a problem in remapping contralesional space. This explains the inability to acquire accurately the second target in a double-step saccade paradigm if it appears in the ipsilesional hemifield following a contralesional target (Duhamel et al. 1992; Heide et al. 1995). Furthermore, they contend that this account explains the ‘revisiting’ behaviour of neglect patients on cancellation tasks (Fig 1B). In support of this model they point out that providing a columnar organisation to target stimuli in cancellation tasks can lead to improved performance for some patients presumably by minimising the number of horizontal saccades needed. With this in mind we examined the performance of two neglect patients on a spatial working memory task that should place low demands on processes of spatial remapping. In addition to addressing the possibility that spatial working memory deficits are evident in neglect patients independent of any problems of spatial remapping, we also intended to address several issues arising from previous work examining cancellation and visual search performance in neglect patients (Husain et al., 2001; Wojciulik et al., 2004). First, previous studies had made use of visual search stimuli that covered portions of both the ipsi- and contralesional visual fields. Such displays are likely to lead to competition between target (and distractor) locations which may then interfere with the ability to accurately keep those locations in mind. In addition, given the biases in spatial orienting, exploratory motor behaviour and spatial remapping discussed above, displays in which stimuli on the left side must compete with stimuli on the right are more likely to favour processing of ipsilesional stimuli independent of any impairment of spatial working memory. Therefore, our task examined spatial working memory without placing targets within a horizontally arranged display and without involving distracting stimuli (Figure 2). In our task the patient was presented with three

vertically aligned squares to the right of central fixation and was instructed to attend to the squares and to attempt to maintain their locations in memory. After 2 seconds of inspection time the squares are replaced by a blank screen for a three second delay period. Following the delay a circle was presented in the same vertical column as the squares to the right of fixation. The circle could appear in one of the locations previously occupied by a square or in a number of locations in which no stimulus had been present. The patient's task was to indicate whether or not the circle was presented in one of the locations previously occupied by a square. Our patients performed poorly on this task despite demonstrating intact verbal working memory capacity for the same delay length (Figure 2).

--- insert Figure 2 about here ---

This result suggests that patients with neglect do indeed have an impairment of spatial working memory. Importantly, what this work shows is that this deficit of spatial working memory can be demonstrated to be independent of any problems the patient may have in either saccadic remapping or in resolving competition between targets and distractors. Each patient was asked to report the number of squares that were present on each trial. This ensured that the patient had indeed seen the squares and given the fact that the stimuli were arranged in a vertical column it is highly unlikely that any direction specific deficit in either saccadic remapping or disengaging attention typically observed for horizontal saccades and shifts of attention, was responsible for their poor performance. The above discussion is not intended to suggest that neglect can be

explained by reference to a spatial working memory deficit alone. It may well be the case that impaired spatial working memory and deficits in spatial remapping both contribute to the neglect syndrome, a point also made by Pisella and Mattingley (2004). Further research will be needed to determine the extent to which these two distinct types of impairment co-occur or can be dissociated in individual neglect patients.

Given that spatial working memory can be shown to be impaired for vertically arranged stimuli presented to the putatively non-neglected side of space (Fig. 2), how might such a deficit help in explaining the most prominent symptom of neglect – the loss of awareness for contralesional stimuli? On its own it may not be sufficient to fully explain a *contralesional* loss of awareness. That is, there is no a priori reason to suspect that the ability to maintain the spatial layout of the environment is lateralised such that the right hemisphere subserves processes of spatial working memory for only the left visual field and vice versa. Alternatively, if one assumes that processes of spatial working memory operate independently of visual field, then one might expect to see impairments for target stimuli that also extended across the full extent of the visual field. Indeed, some aspects of the performance of neglect patients on cancellation tasks (i.e., the failure to detect ipsilesional targets) are indicative of this kind of impairment (Figure 1B). Nevertheless, the dominant impairment in neglect patients is a failure to consciously represent *contralesional* stimuli. To explain this crucial aspect of the syndrome more fully we would suggest requires a combination of the spatial working memory deficit we have demonstrated above (Figure 2) and the strong biases in spatial attention and exploratory motor control discussed earlier. Below we discuss findings from a novel

rehabilitation technique for neglect in an attempt to see how this technique may influence these two aspects of the neglect syndrome.

5. Prism adaptation and neglect.

Attempts at rehabilitating neglect have had limited success both in the range of behaviours that have been successfully modified and in the duration of the observed benefits (although see Robertson, 1999). Techniques such as caloric stimulation or neck muscle vibration do lead to dramatic changes in overt behaviours but unfortunately they last for only brief periods of time (Adair, Na, Schwarz, & Heilman, 2003; Karnath, Fetter, & Dichgans, 1996; Karnath, Christ, & Hartje, 1993; Rubens, 1985). More recently, Rossetti and colleagues (1998) produced remarkable changes in the overt behaviours of neglect patients following a period of adaptation to prismatic lenses (see also Rossi, Kheyfets, & Reding, 1990). The patients wore wedge prisms that shifted their visual perception further towards the right. They were then required to make pointing movements to targets placed to the left and right of their body's midline for around 5 minutes. The visuomotor transformation required to acquire targets accurately while wearing the prisms led to after-effects that had a dramatic influence on the patient's performance of clinical tests of the disorder. That is, subjective judgements of straight-ahead that were initially deviated to the right before prisms, were now shifted towards the left (i.e., closer to the true objective midline; see Figure 3A). Performances on figure copying and line bisection tasks were dramatically improved with some patients even showing leftward biases after prisms where they had shown rightward biases prior to adaptation (Rossetti et al., 1998).

This early work has spawned a relatively large number of studies attempting to understand the effects of prism adaptation in neglect. There are several reasons why this technique has piqued more interest than any other. First, it is non-invasive, not aversive (unlike the administration of ice water used in caloric stimulation) and very simple to administer, allowing for multiple administrations if needed. Second, the effects of prism adaptation have been shown to last much longer than any previous rehabilitation attempts, with improvements in performance being observed anywhere from two hours post adaptation to a week post (Farné, Rossetti, Toniolo, & Ladavas, 2002; Frassinetti, Angelini, Meneghello, Avanzi, & Ladavas, 2002; Pisella, Rode, Farné, Boisson, & Rossetti, 2002; Rossetti, et al. 1998). Finally, the effects of prisms appear not to be restricted to the hand used during the adaptation procedure. That is, changes in postural balance, visual imagery and exploratory eye movements have been observed post prisms (Ferber, et al. 2003; Tilikete, et al., 2001, Rode et al. 2001; Rode, Rossetti, & Boisson, 1998). Thus, the procedure seems to alter higher level internal spatial representations and is not limited to influencing only those visuomotor networks involved in executing the pointing movements made during the adaptation procedure itself.

So what can the effects of prism adaptation tell us about the disorder of neglect? One might assume that the prism adaptation procedure may have several potential sites of action in the brain. The primary candidate may be the cerebellum as it is well known that patients with cerebellar lesions fail to adapt and consequently show no after-effects from prismatic lenses (Thach, Goodkin, & Keating, 1992). One might expect, however, that the role of the cerebellum may be more restricted to the fine tuning of the motor movements involved in the adaptation process and in post-adaptation tasks that also

required skilled visuomotor control. This would leave changes such as improved visual imagery unexplained (Rode et al. 1998, 2001). Other candidate brain regions such as portions of the frontal lobes or spared regions of superior parietal cortex suffer from much the same problem. Put another way, the difficulty in explaining the effects of prism adaptation in neglect lies in the gulf between what we know about how the technique itself operates in the healthy brain (e.g., Redding & Wallace, 1996) and the myriad of changes it seems to induce in neglect patients. In other words, explaining the unusual prism effects in neglect (i.e., that they are long-lasting, not restricted to the effector used, influence multiple levels of spatial representation) by recourse to models of how prisms influence the behaviour of healthy individuals in which those unusual effects are not observed may not prove to be entirely fruitful.

The difficulty in explaining the effects of prism adaptation in neglect may seem less insurmountable if one assumes a more restricted explanation of the effects observed to date. That is, it is possible that prisms influence exploratory motor behaviours and spatial biases in attention while leaving more perceptually based performances unaltered. For example, in a recent single case study, we showed that prisms dramatically shifted exploratory eye movements made towards chimaeric faces while failing to alter the perceptual bias exhibited by the patient for those same stimuli (Ferber et al. 2003). Chimaeric faces are constructed by combining two halves of a face, one half depicted as smiling and the other half with a neutral expression (Figure 3B). Two faces are then vertically arranged with the smiling half appearing randomly on the right or left side of either face. The task is to judge which face appears happier and while healthy individuals select the left-smiling face as appearing happier more often (Heller & Levy, 1981; Levy,

Heller, Banich & Burton, 1983), neglect patients consistently choose the right-smiling face as appearing happier (Mattingley, Bradshaw, Nettleton & Bradshaw, 1994). Consistent with this research, our patient reported that the chimaeric face depicted as smiling on the right half appeared ‘happier’ on almost all trials. When the patient’s eye movements were recorded we found that he failed to explore the left half of the chimaeric face stimuli, perhaps not surprising given his strong bias for perceiving the right-smiling faces as being happier. After prism adaptation the patient’s exploratory eye movements now encompassed the full extent of the face stimuli, even demonstrating a slight bias towards fixating the left side of faces more often (Figure 3B). Thus, prisms had led to a dramatic change in the patient’s exploratory motor behaviour, also evidenced by a dramatic shift, from right towards left space, in his subjective judgement of straight-ahead.

--- insert Figure 3 about here ---

More importantly, despite this dramatic alteration in the patient’s exploratory eye movements, he continued to report that the chimaeric face shown to be smiling on the right side appeared to be happier. In other words, despite the fact that his eye movements now demonstrated that he had explored the full extent of the stimuli, his perceptual bias for the right side remained unaltered (see also Dijkerman, McIntosh, Milner, Rossetti, Tilikete & Roberts, 2003 for a similar dissociation in eye movements and a perceptual size distortion bias post prisms). When questioned more closely about his perception of the chimaeric faces, the patient reported that there was nothing unusual about them,

indicating that he never perceived the unusual split down the middle in each of the faces. This is, of course, in stark contrast to healthy controls who immediately report that the chimaeric faces appear unusual. A similar phenomenon, not reported in the literature but commonly observed by clinicians and researchers using prisms (Rossetti, personal communication), is the complete lack of awareness of the shift caused by the prisms. That is, even when asked directly, many patients will report that there is nothing unusual about the prisms they wore during adaptation despite the fact that they caused a 10 to 15 degree shift in visual perception! What the single case study described above suggests is that while prisms may have profound and long-lasting effects on the spatial orienting and exploratory motor biases evident in neglect, it does not necessarily alter the subjective perceptual biases. This dissociation highlights the point made earlier, that biases in spatial attention and exploratory motor behaviour are not in and of themselves sufficient to explain the loss of awareness for contralesional stimuli or events inherent to the neglect syndrome. Such biases, which obviously contribute substantially to the presentation of neglect patients, must be coupled with some other impairment, perhaps of spatial working memory (see Fig 2), before the full neglect syndrome becomes apparent.

Most studies examining the influence of prism adaptation on neglect have explored performance on clinical tests of the disorder such as line bisection or figure copying (e.g., Pisella et al. 2002; Rossetti et al. 1998). Changes in the performance of these tasks can also be explained in terms of changes to exploratory motor movements or alterations in the allocation of attention. Given the fact that patients now *detect* targets they previously omitted it would be difficult to argue that *only* attentional orienting or exploratory motor behaviour have been altered with *awareness* of the stimuli being

detected remaining unchanged. That is, the patient must be aware of a target they report seeing. Similarly, in a recent single case study, Maravita and colleagues (2003) demonstrated substantial improvement in tactile perception (i.e., a reduction in tactile extinction) following prism adaptation. They argued that this represents an alteration in the patient's conscious awareness of tactile stimuli. Similarly, Berberovic and colleagues (2004) showed a reduced bias in temporal order judgements post prisms. It would be difficult to argue that awareness has not been improved in these two instances. What we are suggesting here is that the change brought about by prisms may not represent a change in conscious awareness of stimuli per se but may be the result of alterations in other behaviours that are in turn necessary antecedents for awareness. Further research that directly explores biases in spatially directed behaviours (e.g., eye and hand movements) and perceptual judgements of the kind explored in the chimaeric faces task are needed to determine the extent to which the impairments characteristic of the neglect syndrome are altered by prism adaptation.

6. Carving neglect at its joints: Where is the critical lesion?

The earliest descriptions of unilateral neglect that were able to localise the underlying lesion with any degree of certainty come from cases initially described by Paterson and Zangwill (1944; see Mattingley, 1996 for a detailed review of this classic case). The penetrating head wound of one of their patients primarily affected the angular gyrus and underlying white matter. Since then, neuroimaging techniques have allowed us to determine more precisely the locus of the critical lesion for producing neglect, although this has yet to provide a definitive answer free from controversy (Karnath,

Ferber & Himmelbach, 2001; Karnath, Fruhmann-Berger, Kuker, & Rorden, 2004; Mort, Malhotra, Mannon, Rorden, Pambakian, Kennard & Husain, 2003; see also Rorden & Karnath, 2004 for a discussion of the lesion method in general). Identifying the neuroanatomical correlate of spatial neglect in humans is challenging because human brain lesions vary tremendously in size and the neglect syndrome itself is multifaceted. While Paterson and Zangwill's case (1944) described a patient with a discrete lesion resulting from a penetrating head wound, the more common cause of neglect is a middle cerebral artery stroke causing widespread damage to the lateral cortical surface and underlying white matter that this artery subserves (Duvernoy, 1999).

It is beyond the scope of this review to give a detailed account of the current controversy surrounding the critical lesion site for neglect. Suffice to say that recent studies making use of MRI scans in neglect patients have suggested that the critical region of overlap in a series of neglect patients' lesions is either in the superior temporal gyrus (STG; Karnath et al., 2001; Karnath, et al., 2004) or the temporo-parietal junction (TPJ; Mort et al., 2003; see Figure 4 for a schematic of these regions)⁴.

--- insert Figure 4 about here ---

Perhaps what the differences between these studies reflect is the fact that disorders such as neglect that are behaviourally heterogeneous are almost certain to have some degree of heterogeneity in their underlying pathology. The challenge now is not to determine whether or not the STG or the TPJ is the *critical* lesion site for neglect, but instead to

⁴ Early studies using computerised tomography (CT) scans indicated substantial overlap in the inferior parietal lobule (IPL) proximal to the temporo-parietal junction (TPJ; Vallar & Perani, 1986).

explore the functions of each region – perhaps through human fMRI or monkey neurophysiology, which can both provide greater spatial precision than lesion studies – to determine how lesions to these distinct regions contribute to the various presentations observed in neglect patients. What we intend to do for the remainder of this review, is examine how lesions to different brain regions inform functional aspects of the neglect syndrome. The regions commonly affected in patients with neglect include (but are not restricted to) the angular gyrus (AG), the TPJ (Vallar & Perani, 1986; see also Mort et al., 2003 for a more recent anatomical demarcation of the TPJ) and the superior temporal gyrus (STG; Karnath et al, 2001, 2004). In the following sections we will discuss the different functions that could be ascribed to these regions and how they may inform our understanding of the neglect syndrome.

6.1 The angular gyrus.

From the very earliest studies of neglect the angular gyrus became the prime suspect as the critical lesion site needed to produce the disorder (Paterson & Zangwill, 1944; Mattingley, 1996). While more recent studies have cast some doubt over how critical this region is to neglect, evidence from psychiatric disorders such as schizophrenia, and from studies examining out-of-body experiences (OBEs), may shed a little more light on what the specific role of the angular gyrus may be (Blanke, Ortigue, Landis, & Seeck, 2002; Danckert, Saoud & Maruff, 2004; Spence, Brooks, Hirsh, Liddle, Meehan, & Grasby, 1997; Tong, 2003).

For some time now researchers interested in schizophrenia have suggested the disorder affects association cortex in the parietal lobes (Frith, 1992; Perlson, 2000; Ross

& Perlson, 1996). Initially, emphasis was placed on the left parietal cortex and the possible role this region may play in one of the more common positive symptoms of schizophrenia – auditory hallucinations (Perlson, 2000). The source-monitoring hypothesis (Harvey, 1985; Seal, Aleman & McGuire, 2004) suggested that patients with schizophrenia were unable to identify accurately internally generated thoughts as being their own and thus misattributed the source to something external. More recently, this hypothesis has been applied to passivity phenomenon in schizophrenia, in which the patient believes that their thoughts or actions are controlled by an external agent (Blakemore & Frith, 2003; Blakemore, Smith, Steel, Johnstone, & Frith, 2000; Blakemore, Frith & Wolpert, 1999; Danckert et al., 2004; Spence et al., 1997). In this instance, the patient may be generating a faulty forward model of their intended actions or alternatively, may make errors when comparing the anticipated sensory outcomes of such a forward model with the actual sensory feedback – a process thought to depend on parietal cortex (Danckert, Ferber, Doherty, Steinmetz, Nicolle, & Goodale, 2002; Danckert et al., 2004; Sirigu, Duhamel, Cohen, Pillon, Dubois, & Agid, 1996). Recent neuroimaging studies suggest that abnormalities in functioning of the right parietal cortex, including the angular gyrus, may be at the heart of the passivity phenomena in these patients (Franck, O’Leary, Flaum, Hichwa, & Andreasen, 2002; Spence et al., 1997). This suggests that the angular gyrus may function as a comparator of anticipated (based on forward models) and actual sensory outcomes of goal-directed actions. Such a function would play a critical role in directing exploratory eye and hand movements to different regions in space and may also be important for maintaining an internal representation of one’s own body in space – both functions that have been demonstrated

to be impaired in patients with neglect (Karnath & Niemeier, 2002; Niemeier & Karnath, 2003).

Finally, a recent study taking advantage of the in-dwelling electrodes of an epilepsy patient, found that stimulating the angular gyrus could lead to an out-of-body experience in the patient (Blanke et al., 2002; see Tong, 2003 for review). Again, this implicates the angular gyrus in processes involved in maintaining an accurate representation of one's own body (corporeal awareness) and the consequences of self-generated actions (see also Berlucchi & Aglioti, 1997 for review). The result then of a lesion to the angular gyrus would be to disrupt awareness not only of one's own body but also of the actions generated by the individual. Coupled with a bias in orienting towards one side of space this could help explain many of the symptoms typically associated with deficient exploration of left visual space in neglect patients.

6.2 The temporo-parietal junction (TPJ).

Recent neuroimaging evidence suggests that the TPJ is crucial in directing spatial attention to behaviourally relevant stimuli in the environment (Corbetta & Shulman, 2002). More specifically, increased neural activation is observed in the right TPJ when subjects reorient attention to a target appearing at an uncued location or shift their attention covertly from one visual field to the other (Corbetta, Kincade, & Shulman, 2002; Corbetta, Kincade, Ollinger, McAvoy, & Shulman, 2000; Yantis, Schwarzbach, Serences, Carlson, Steinmetz, Pekar, & Courtney, 2002). This was the case regardless of the direction in which attention had to be shifted. That is, increased activation of the right TPJ was seen when attention shifted from a cue in the left visual field to a right visual

field target and vice versa. In addition, one imaging study that explored the effects of distractor stimuli on current task processing, found that the right TPJ was more activated only when distractor stimuli were task-relevant (Downer, Crawley, Mikulis, & Davis, 2001). Taken together, these imaging results would suggest that the right TPJ is activated when attention must be directed towards behaviourally relevant stimuli regardless of their physical location in space (Corbett & Shulman, 2002).

A very different pattern of activation is observed in the superior regions of the parietal cortex along the intraparietal sulcus (Corbetta, Shulman, Miezin, & Petersen, 1995; Shulman, Ollinger, Linenweber, Petersen & Corbetta, 2001). In this region of the parietal lobe, activation is more specifically related to searching for and detecting a salient target. In contrast to the activity seen only in the right TPJ when behaviourally relevant information is present, activation in this system has been observed in both hemispheres depending on the direction to which attention must be oriented (Corbetta et al. 1995; 2000; Shulman et al. 2001). The dissociation between the patterns of activation in these two regions led Corbetta and Shulman (2002) to propose a model of attentional orienting comprising two major components. The first, involving regions of the superior parietal cortex along the intraparietal sulcus, is responsible for voluntary shifts of attention to behaviourally relevant stimuli and is represented bilaterally. They go further to suggest that this system may play an important role in forming links between incoming sensory information and the relevant behavioural (i.e., motor) responses (Corbetta & Shulman, 2002). This fits well with what is known of the disorder of optic ataxia (Perenin & Vighetto, 1988; Vighetto & Perenin, 1981). That is, in this disorder, typically associated with superior parietal lesions, patients exhibit a difficulty in directing their

hand towards peripheral targets. Importantly, for patients with lesions of right superior parietal cortex the greatest deficit is observed for reaching with the left hand in left hemispace, whereas left superior parietal lesions produce the opposite pattern – a severe deficit in reaching with the right hand in right hemispace (Perenin & Vighetto, 1988; Vighetto & Perenin, 1981). This kind of deficit may be due to impaired attentional orienting towards peripheral stimuli and the fact that it is direction and hand specific fits well with the first component of Corbetta and Shulman’s model discussed above.

The second component of attentional orienting proposed by Corbetta and colleagues is subserved by the right TPJ and serves an interrupting function – or in their words, acts as a ‘circuit breaker’. Obviously, there is likely to be a constant interaction between the two systems. A circuit breaker that does not know where the current focus of attention is would be largely useless. Disruption of this kind of function for the TPJ would produce many of the spatial biases in attention and exploratory motor control that are characteristic of neglect patients. That is, behaviourally relevant information, presumably from the entire visual field, will no longer be processed efficiently by the damaged right TPJ. Although the superior parietal module described above is thought to exist bilaterally, there is some evidence for asymmetrical coding of space such that the left intraparietal region codes for right visual space while the right intraparietal cortex codes for both left *and* right visual space (Corbetta et al. 2002). A disruption to the interaction between the right TPJ and the right superior parietal cortex would lead to impairments in voluntary orienting throughout the visual field and may leave the left superior parietal system unchecked, creating a bias towards processing and exploring right, ipsilesional space (Corbetta & Shulman, 2002).

6.3 The superior temporal gyrus (STG).

Even though spatial neglect in humans has typically been associated with lesions to the parietal cortex, a lesion to the same area in the monkey brain does not lead to neglect. Instead, Watson and colleagues (1994) found that spatial neglect was observed in monkeys after lesions of the superior temporal cortex that included both banks of the superior temporal sulcus and extended into the superior temporal gyrus, while ablation of the inferior parietal lobe did not lead to neglect. Furthermore, Luh and colleagues (1986) observed that lesions of the superior sulcal polysensory cortex of the monkey impair the ability to orient the head or the eye to contralesional visual stimuli, particularly when an ipsilateral stimulus is presented simultaneously. Finally, Scallidhe and colleagues (1995) found that macaque monkeys with lesions to the superior temporal polysensory (STP) area show significant increases in saccadic latencies to contralesional targets. Taken together, this evidence suggests that the STP is involved in attending to stimuli located on the contralateral side of space. According to Bruce and colleagues (1981) virtually all STP neurons are visually responsive and about one-half of these neurons also respond to auditory stimuli. Further, neurons in the STP have large receptive fields that extend well into both visual fields. Also, the STP receives input from both the dorsal and ventral stream (see for example Morel & Bullier, 1990; Seltzer & Pandya, 1978). All of these properties make the neurons in the STP ideally poised to integrate visual information about the identity (ventral stream) and location (dorsal stream) of objects across the entire visual field. It is possible that an evolutionary shift of the polysensory area of the

macaque in the anterior portion of the superior temporal sulcus might have taken place to a more dorsal location in the human brain – the superior temporal gyrus.

What are the known functions of the superior temporal gyrus in humans that are relevant for our discussion of neglect here? Friedrich and colleagues (1998) demonstrated difficulties in reorienting attention (i.e., longer RTs to contralesional targets following ipsilesional cues) in patients with lesions to the temporo-parietal junction including the posterior portion of the superior temporal gyrus. In addition, the superior temporal cortex is involved in encoding the locations and identities of objects (Köhler, Moscovitch, Winocur, Houle, & McIntosh, 1998). We would not suggest that the superior temporal cortex necessarily *subserves* all of the faculties involved in neglect discussed above (motor control, attention, working memory), but rather that it may provide the major relay (maybe via the superior longitudinal fasciculus which connects parietal and dorso-lateral pre-frontal areas) to integrate these faculties over time and space to generate a coherent percept of an ever-changing environment. In other words, while the angular gyrus and the TPJ may support more specific functions such as corporal awareness or attentional deployment, the superior temporal gyrus may be the site in the brain where all of these different faculties are integrated into the coherent whole that we perceive and act upon.

As we have mentioned earlier, it is beyond the scope of this review to resolve the debate regarding the critical lesion site in neglect. What the above discussion highlights is that neglect is the result of a constellation of separable deficits that include spatial working memory, attention and motor control. None of these deficits alone may be sufficient to result in neglect, but in conjunction they will lead to the profound lack of

awareness for stimuli located on the left side that characterises the syndrome.

7. Conclusions

Unilateral neglect is a behaviourally complex disorder in which patients present with a heterogeneous cluster of symptoms and deficits. What is common for all neglect patients is a loss of awareness for events or stimuli in contralesional space. This may present itself as either perceptual or motoric biases in exploratory behaviour such that only the right half of space is explored. Importantly, some perceptual biases indicative of a loss of awareness for contralesional space can remain even when exploratory motor behaviours have been modified (i.e., via prisms) such that the left half of space is now explored. What this dissociation suggests is that the neglect syndrome requires more than just a bias in spatially directed overt behaviours to produce the characteristic loss of awareness for one side of space. Additional impairments, perhaps to spatial remapping processes or to the ability to represent spatial information in working memory may represent the critical impairments needed to produce this loss of awareness. None of these deficits alone would be sufficient for the full-blown neglect syndrome but in conjunction they will lead to the characteristic lateralized loss of awareness. Obviously, this hypothesis requires further experimentation in patients and perhaps via neuroimaging in healthy individuals before it can be fully accepted.

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Figure Captions

Figure 1. Panel A. Examples of neglect behaviour on clinical tests of the disorder. Top left is a copying task in which the patient has distorted or neglected the left side of the figures to be copied (model is to the left in black and the patient's copy is to the right in red). Top right is a clock drawing task in which the patient is asked to insert the numbers on the clock face provided and has placed the numbers to the right of the clock face only (there is also an organizational error in this patient's performance in which the order of numbers is incorrect). In the middle of the figure is a typical line bisection performance in which the patient's mark (in red) is to the right of the true centre (as indicated by the dashed line). Panel B. This figure shows two examples of a typical cancellation performance in which the patient fails to cancel targets on the left. The left example (star cancellation) shows the patient's marks in red and demonstrates the failure to cancel most targets on the left of the page. In addition, the patient also failed to cancel some targets on the right putatively 'non-neglected' side of the page (as indicated by the transparent grey circles). The right example (Albert's line cancellation) demonstrates 'revisiting' behaviour. That is, the red marks indicate the patient's initial cancellations of the lines, while the blue marks indicate a second cancellation made by the patient suggesting that he was treating the 'old' target as if it were 'new'.

Figure 2. Panel A. Schematic representation of the spatial working memory task. Patients saw three squares to the right of a fixation point that were vertically aligned. After 2 seconds the squares were replaced with a blank screen for a delay period of 3 seconds. A target circle then appeared with the patient asked to determine whether or not

the circle was in a location previously occupied by one of the squares (in the example given here the answer would be ‘yes’). Data from two neglect patients (with representative CT images of each patient above the graph) are presented in Panel B. Accuracy was calculated as the number of hits (i.e., correctly identifying that the target circle did appear in one of the locations previously occupied by a square) minus false alarms (i.e., indicating that the target circle appeared in one of the locations previously occupied by a square when in fact it did not) represented as a percentage of total trials. The verbal working memory task included here as a comparison, presented three numerals in a vertical column to the right of fixation in a manner analogous to the spatial task. Timing of presentation was also identical with the three probe numbers appearing for 2 seconds before a 3 second delay. After the delay a single numeral was presented to the right of fixation (always at the same location from trial to trial). The patient had to indicate whether or not this number was among the three numbers presented earlier. Both patients performed at ceiling on this task.

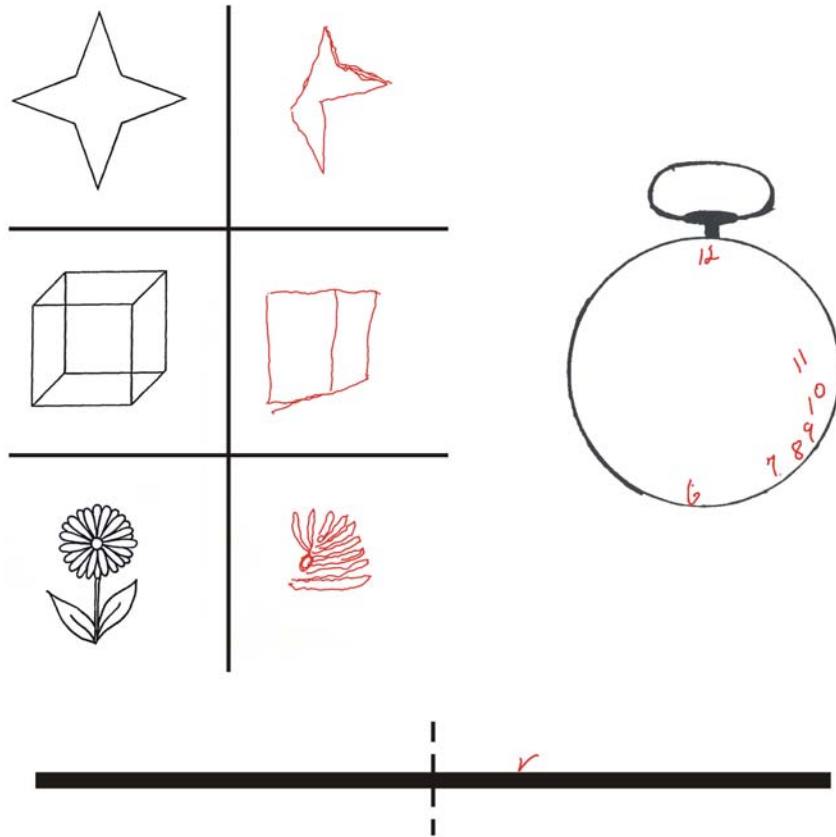
Figure 3. Panel A. Straight-ahead pointing data in patients with neglect (left) and healthy controls (right) prior to (grey arm) and after (black arm) a period of adaptation to rightward shifting prisms (adapted from Rossetti et al. 1998). Prior to prism adaptation patients with neglect indicated that their subjective notion of straight-ahead was shifted to the right of an objective midpoint defined by their body’s midline. After prisms, their notion of straight-ahead had shifted to the left and now coincided more closely to the objective midpoint. Panel B. Performance of one patient with neglect on a chimaeric faces task prior to and after prism adaptation. To the left is a schematic of the typical

chimaeric faces task in which the patient must indicate which of the two faces (author JD) appears to be happier. Eye movement data (upper section) prior to prism adaptation indicated that the patient failed to fixate the left most section of the chimaeric faces. After prisms he now fixated the whole stimulus and even exhibited a slight leftward bias for fixations (adapted from Ferber et al. 2003). Despite the change in eye movements the patient continued to demonstrate a rightward perceptual bias for choosing which of the two chimaeric faces appeared to be happier (lower section of figure).

Figure 4. Schematic outlining the regions of parietal and temporal association cortex commonly involved in the neglect syndrome. The region of the angular gyrus is outlined in red, while the superior temporal gyrus is outlined in purple (the posterior portion of which is commonly lesioned in neglect patients). The region of the temporo-parietal junction is indicated by the red circles joined by a dotted line (see Mort et al. 2003 for a more detailed description of demarcating this region).

Figure 1. Danckert and Ferber

A



B

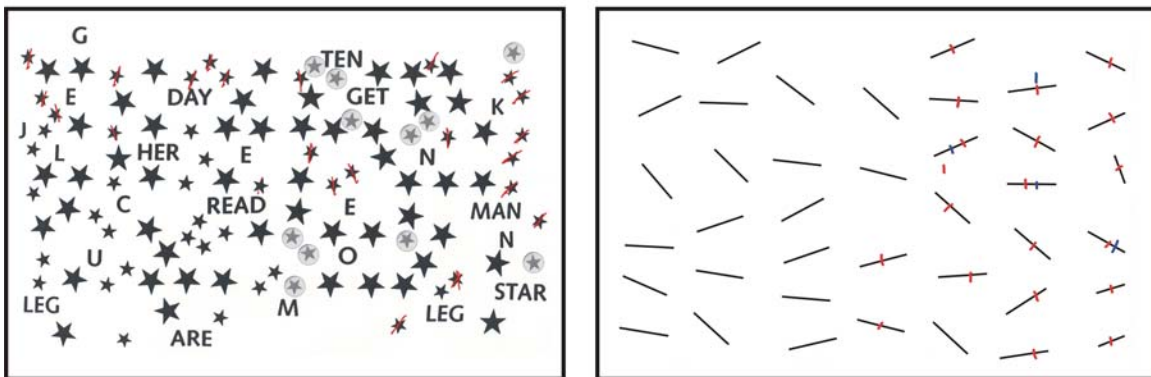
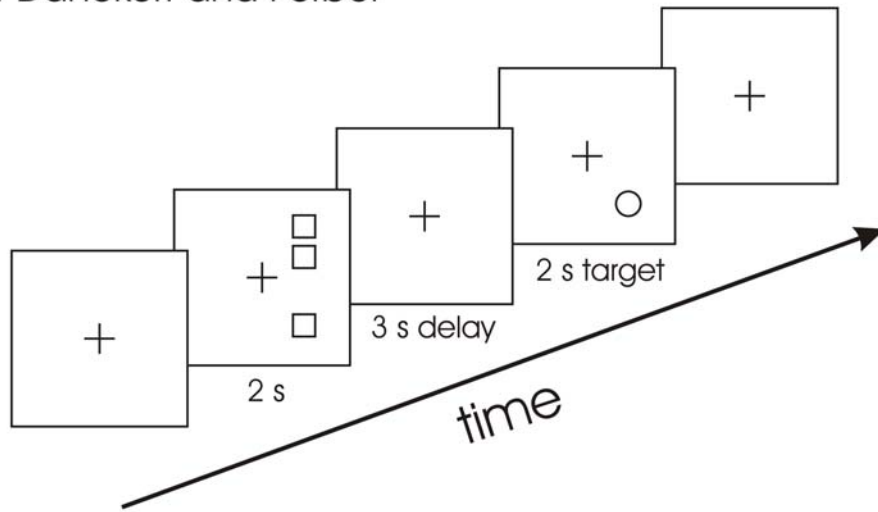


Figure 2. Danckert and Ferber

A



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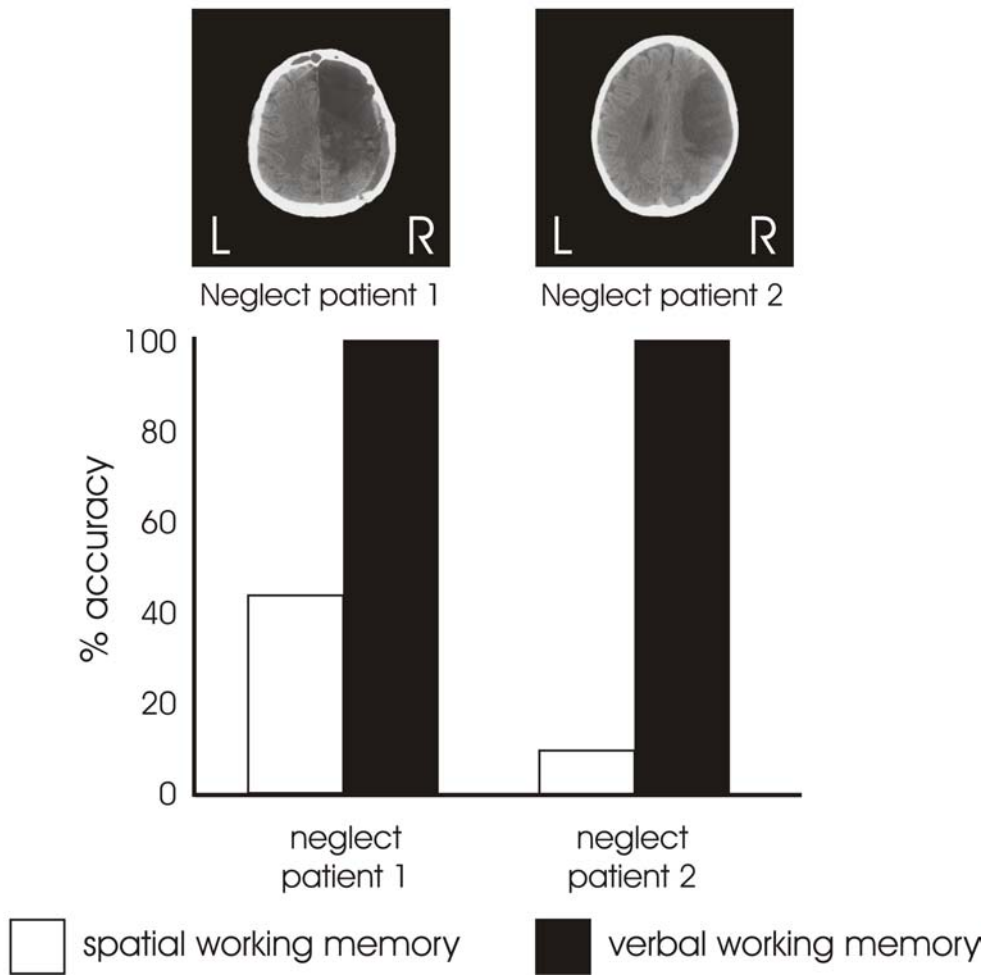
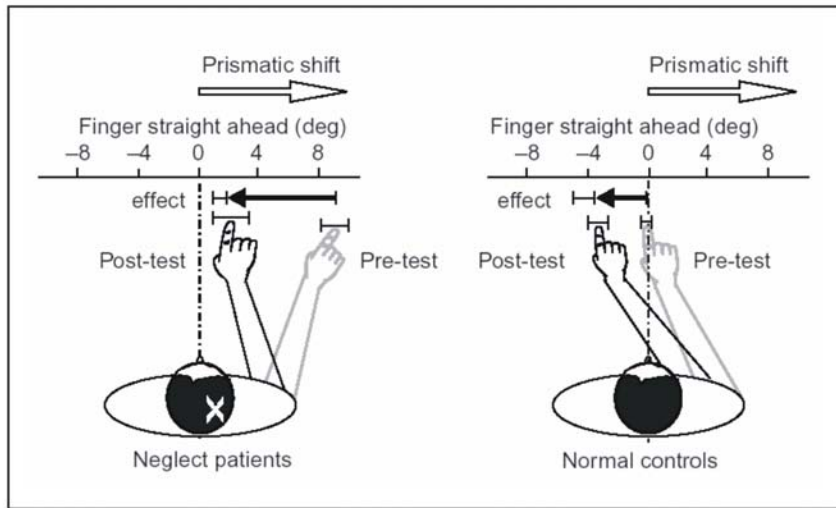


Figure 3. Danckert and Ferber

A



B

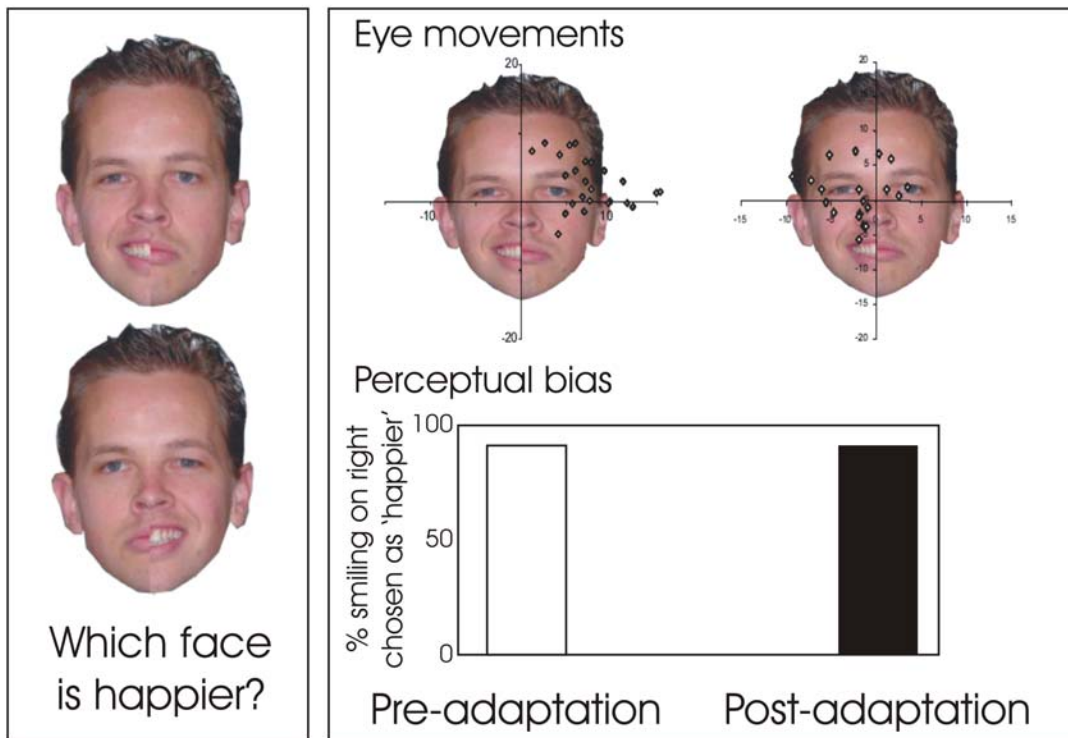


Figure 4. Danckert and Ferber

