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Perspective

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.
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1. Introduction

Unilateral neglect is traditionally defined as a failure to report, respond to or orient towards stimuli in contralesional space (Driver & Mattingley, 1998; Halligan, Fink, Marshall, & Vallar, 2003; Heilman, Watson, & Valenstein, 1993). 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 and Kerkhoff, 2001 for review). The second description, at first blush, seems to be rather vague in that it emphasizes 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 (Bisiach & Rusconi, 1990; Ferber, Danckert, Joanisse, Goltz, & Goodale, 2003; Walker & Findlay, 1997; Young, Hellawell, & 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 return to the issue of the critical lesion location for neglect later in this review, but for now will talk about the behavioural symptoms associated with 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

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detect a target amidst distractors and to place a mark through the 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 midpoint (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 (Fig. 1).

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 (Beschin & Robertson, 1997; Bisiach, Perani, Vallar, & Berti, 1986; Cowey, Small, & Ellis, 1994; Halligan & Marshall, 1991; see also Berti, Smania, & Allport, 2001 and Pegna et al., 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

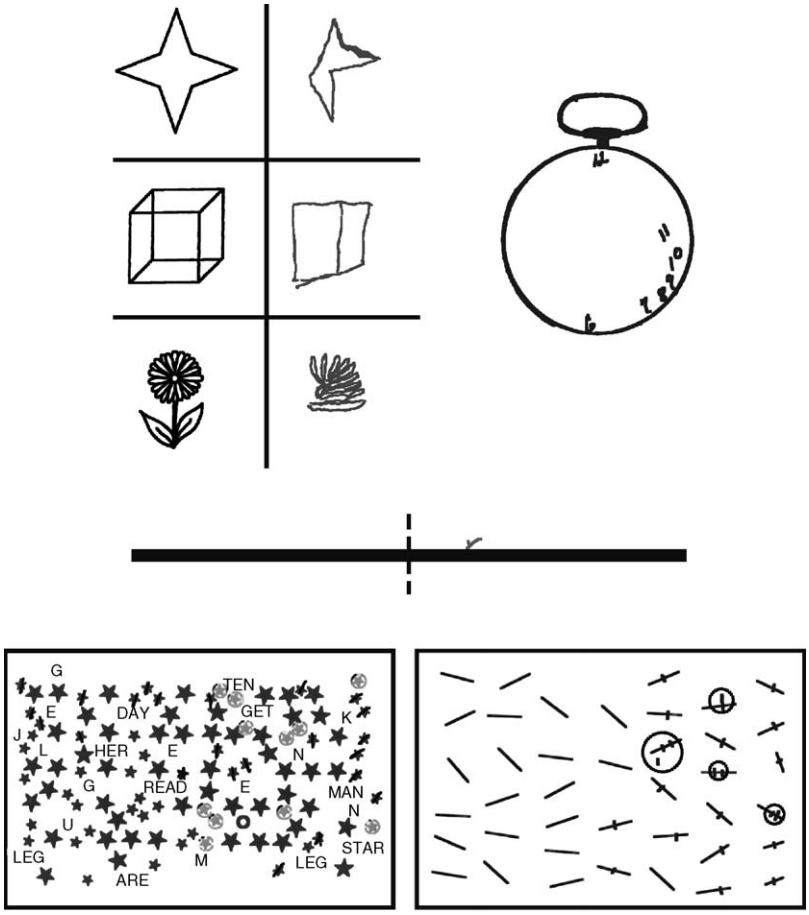

Fig. 1. Examples of neglect behaviour on clinical tests of the disorder. Top left is a copying task in which the patient has distorted or neglected the left side of the figures to be copied (model to be copied is to the left with the patient's copy to the right). Top right is a clock drawing task in 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 organisational 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 grey) is to the right of the true centre (as indicated by the dashed line). Lower panels show 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 black 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 example on the right of the page (Albert's line cancellation) demonstrates 'revisiting' behaviour. That is, the black marks indicate the patient's initial cancellations of the lines, while the circles highlight instances in which a second cancellation on an already marked line was made by the patient suggesting that he was treating the 'old' target as if it were 'new'.

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 Milner, 1997 and Rafal, 1996 for review). It is important to note here that the disorders of extinction and neglect are generally considered to be distinct for two important reasons. First, the lesion locus for extinction in the parietal cortex tends to be more superior than the focus for neglect (Karnath, Himmelbach & Küker, 2003; Milner, 1997). Second, extinction has been demonstrated to be equally common following left and right hemisphere lesions (Milner, 1997), while neglect is far more common following right hemisphere lesions (Driver & Mattingley, 1998;Karnath, Ferber, & Himmelbach, 2001). Nevertheless, throughout this review we will discuss work from patients with extinction where they are deemed to be relevant to the arguments we posit (on such occasions we will indicate whether the work being discussed concerns patients only with extinction and whether similar results have been observed in patients with neglect).

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 (see Kerkhoff, 2001 for a review of neglect models based on the ideas of distorted reference frames or impaired motor control). 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 et al., 2001). When directly tested, their judgment 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 the 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 et al., 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 (Redding & Wallace, in press). Also, reaction times to visual stimuli were found to be fastest at a mid-periphery location on the ipsilesional side (Smania et al., 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, Bradshaw, & Phillips, 1992; Mattingley, Husain, Rorden, Kennard, & Driver, 1998; 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 trajectory.[1] While 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 (Husain et al., 2000; Mattingley, Bradshaw, Nettleton, & Bradshaw, 1994; Mattingley, Phillips, et al., 1994).

A very different picture is observed when patients with neglect are asked to explore space, either with their eyes or via hand movements (Himmelbach & Karnath, 2003; Karnath & Niemeier, 2002). 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 (Himmelbach & Karnath, 2003; Karnath & Niemeier, 2002). 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

[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.

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; Posner, Walker, Friedrich, & Rafal, 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 et al. (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, 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, Eglin, Robertson, and Knight (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 numbers 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 within 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 judgment' (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 left 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

[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.

[3] Note that the Rorden et al. (1997) study involved patients who demonstrated extinction to double simultaneous stimulation without demonstrating neglect.

potential 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 syndrome. Indeed, if patients demonstrating the characteristic impairment on the TOJ task are presented with spatially non-predictive auditory cues prior to making their judgments, 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. In contrast to this, a recent study examining spontaneous recovery from neglect symptoms found that patients showed little or no spontaneous improvement in tasks of sustained attention that have previously been associated with general levels of arousal (Farné et al., 2004). Furthermore, patients with neglect did not differ in their degree of impairment on these tasks of sustained attention when contrasted with patients without neglect (Farné et al., 2004). Despite this, the question remains as to 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 unspeeded 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 (Fig. 1). 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; 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 et al., 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, Berberovic, and Mattingley (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 (Colby & Goldberg, 1999; Duhamel, Goldberg, Fitzgibbon, Sirigu, & Grafman, 1992). 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, akin to the stimulus arrays used in 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, Colby, & Goldberg, 1992; Duhamel, Goldberg, et al., 1992) 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 one neglect patient with a large right hemisphere fronto-parietal lesion (Duhamel, Goldberg, et al., 1992). 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, Colby, et al., 1992; Duhamel, Goldberg, et al., 1992; Heide et al., 1995). Furthermore, they contend that this account explains the 'revisiting' behaviour of neglect patients on cancellation tasks (Fig. 1). 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 required and thus the need for horizontal remapping. With this model in mind we examined the performance of four neglect patients on a spatial working memory task that should place low demands on processes of spatial remapping (see Ferber & Danckert, in press for full details of this study). 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., 2001, 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 at encoding which may then interfere with the ability to accurately keep those locations in mind—regardless of remapping problems, which may arise *after* encoding. 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 (Fig. 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 s of inspection time the squares were replaced by a blank screen for a 3 s 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 (Fig. 2; see Ferber & Danckert, in press).

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 lateralized 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 (Fig. 1). 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

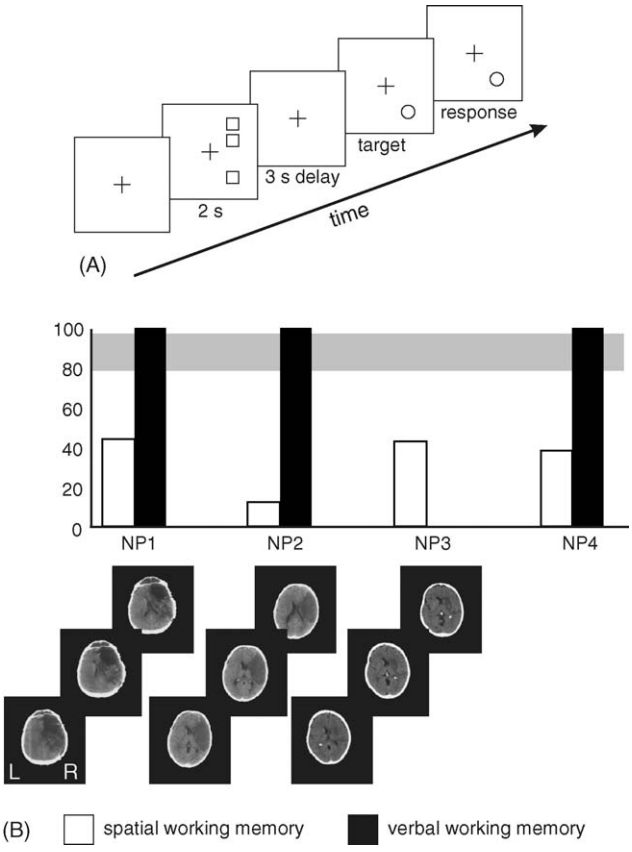

Fig. 2. (Panel A) Schematic representation of the spatial working memory task. Patients saw three squares to the right of a fixation point that were vertically aligned. After 2 s the squares were replaced with a blank screen for a delay period of 3 s. A target circle then appeared and the patient was 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'). The target remained present on the screen until a response was made. For the verbal working memory control task three numerals were presented 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 s before a 3 s delay. After the delay a single numeral was presented in the same vertical column to the right of fixation. The patient had to indicate whether or not this number was among the three numbers presented earlier. Data from four neglect patients (with representative CT images of each patient below the figure; note, scans were not available for NP4 and patient NP3 did not complete this version of the verbal working memory task. In a different verbal working memory task (a one-back task in which the patient had to press a button if the current number presented centrally on the screen matched the number presented just previously), the patient's performance was far superior to her spatial working memory performance) are presented in (Panel B) (spatial working memory performance is indicated in open bars with verbal working memory presented in filled bars; all patients were at ceiling for the verbal working memory task). For both tasks, 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; or that the probe number was among one of the target numbers) 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; similarly, for the verbal task a false alarm occurred when the patient indicated the probe was one of the target numbers when it was not) represented as a percentage of total trials. Control data from four right hemisphere lesioned patients without neglect for the spatial working memory task are indicated in the grey bar. This represents the 95% confidence interval for this group's performance, which is well above that of all four neglect patients (adapted from Ferber & Danckert, in press).

above (Fig. 2) and the strong biases in spatial attention and exploratory motor control discussed earlier. Before discussing in more detail our hypotheses regarding how such a constellation of deficits may lead to neglect, we first examine findings from a novel rehabilitation technique for neglect in an attempt to see how this technique may influence these various aspects of the neglect syndrome.

5. Prism adaptation and neglect

A small percentage of patients who present with neglect in the acute stages post stroke do spontaneously recover from the disorder (according to a recent study around 9% completely recover, while around 43% show some improvement in a 2 week period post stroke; Farné et al., 2004). This leaves a large percentage of patients with debilitating symptoms. 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, Christ, & Hartje, 1993; Karnath, Fetter, & Dichgans, 1996; Rubens, 1985). More recently, Rossetti et al. (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 min. 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 judgments 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 Fig. 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 (see Redding & Wallace, in press and Redding, Rossetti, & Wallace, 2005 for reviews). 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 2 h 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

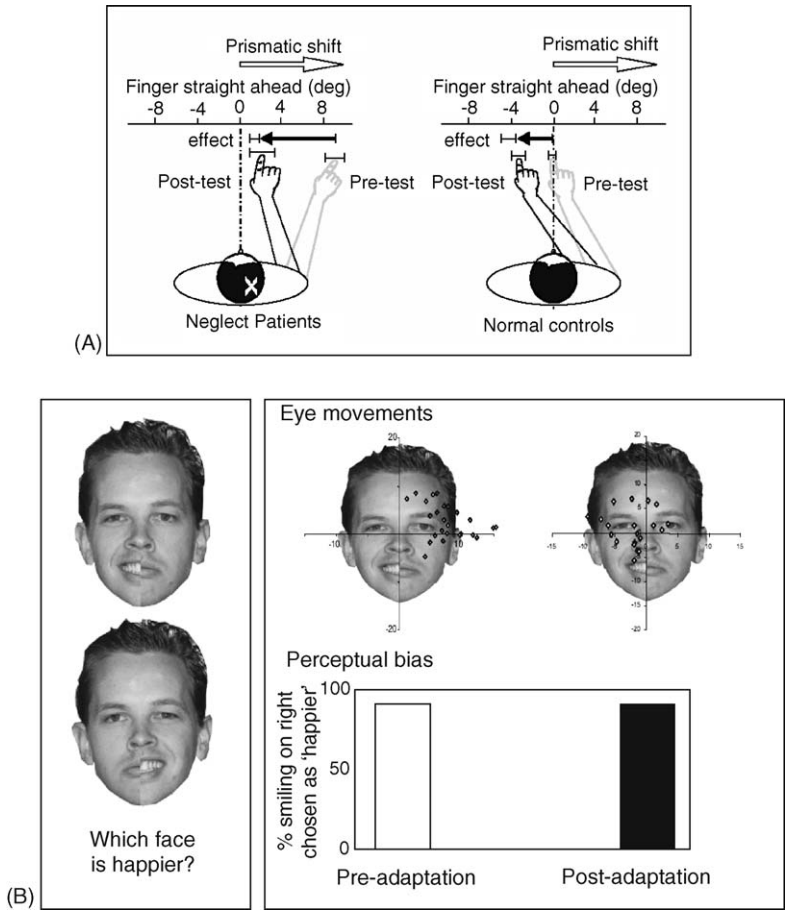

Fig. 3. (Panel A) Straight-ahead pointing data in patients with neglect (left) and healthy controls (right) prior to (grey arm) and after (black arm) a period of adaptation to rightward shifting prisms (adapted from Rossetti et al., 1998). Prior to prism adaptation patients with neglect indicated that 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 with 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 J.D.) appears to be happier. Eye movement data (upper section, fixations depicted as diamonds) 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 strong rightward perceptual bias for choosing which of the two chimaeric faces appeared to be happier (lower section of figure).

in postural balance, visual imagery and exploratory eye movements have been observed post prisms (Ferber et al., 2003; Rode, Rossetti, & Boisson, 2001; Rode, Rossetti, Li, & Boisson, 1998; Tilikete et al., 2001). Thus, the procedure seems to alter higher level internal spatial representations and is not limited to influencing only those visuomotor networks subserving the effector used when executing the pointing movements made during the adaptation procedure itself.

In a recent review of prism adaptation in neglect, Redding and Wallace (in press) suggest that neglect can be considered (at least in part) a result of dysfunctional calibration—one of the components involved in adaptation to prismatic shifts. That is, various frames of reference, for example, eye-in-head or head–hand reference frames, are normally calibrated to suit various task demands. Two aspects of such calibration processes appear to be impaired in neglect; first, the subjective notion of straight ahead is shifted to the right and second, the normal 'work space' – or region of space within which attention and motor behaviours are directed relevant to a given task – is constricted relative to healthy individuals. This fits well with our discussion above of the impairments of attentional orienting and exploratory motor behaviour in neglect (see Sections 2 and 3). When individuals wear prismatic lenses a form of recalibration is required such that reaching movements towards the visually displaced target locations are recalibrated to correct for the error induced by the prisms. This is sometimes referred to as the 'direct' effects of prisms (Redding & Wallace, 1996, in press). Over the course of the adaptation procedure spatial reference frames may then become realigned with the newly calibrated sense of straight ahead. Such a realignment is thought to be responsible for the commonly observed after effects of prisms such that once prisms are removed the individual's subjective notion of straight ahead is shifted in the direction opposite to that of the prismatic shift (among many other after effects). Redding and Wallace (in press) suggest that prisms exert their influence on neglect by recalibrating the patient's subjective notion of straight-ahead to coincide more closely with an objective straight ahead position. Importantly, they claim that the realignment of spatial reference frames

ameliorates the normally dysfunctional calibration evident in neglect (i.e., the shifted sense of straight-ahead) but does not alter constriction of what they call 'task work space'.[4]

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). In addition, the role played by the cerebellum may be in the initial recalibration of reference frames in order to adjust to the displacement of visually perceived targets induced by the prisms (Blakemore, Frith, & Wolpert, 2001; Redding & Wallace, in press). Without showing any adaptation to prismatic displacement in the first place (Thach et al., 1992) it is difficult to know whether this region plays any role in the realignment of spatial reference frames that typically leads to after effects. 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 during adaptation, and 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 (but see Redding et al., 2005; Redding & Wallace, in press for excellent reviews of just such an approach).

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 unaltered behaviours that rely on more consciously mediated perceptual processing. 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 explicit 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 (Fig. 3B). Two of these chimaerics 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, et al., 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 (Fig. 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 judgment of straight-ahead.

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 explicit perceptual bias for the right side remained unaltered (see also Dijkerman et al., 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 commonly observed by clinicians and researchers using prisms, is the complete lack of awareness of the shift caused by the prisms (Rossetti, Rode, & Goldenberg, 2005). These authors report that "none of the neglect patients we have examined so far (even when tested up to 28 years after the stroke) noticed anything special when wearing the prism goggles" (Rossetti et al., 2005, p. 486). This is true in our own experience such that even when patients are asked directly, they will report that there is nothing unusual about the prisms they wore during adaptation despite the fact that they caused a 10–15° 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.

One aspect of the case study reported above has received some criticism (Redding & Wallace, in press). Although our

[4] Redding and Wallace (in press) do suggest that some amount alteration of the constricted task work space may occur but that this is a consequence of task-specific relearning and as such is not a pure effect of prism adaptation.

patient demonstrated a dramatic overcompensation for the shift induced by the prisms (i.e., the change in straight ahead pointing amounted to a 143% overcompensation for the 10° shift of the prisms) this was still not enough to shift his subjective notion of straight-ahead into contralesional space. Nor was it proportional to the amount of shift in exploratory eye movements (Ferber et al., 2003). While this may suggest that the shift induced by the prisms was "not sufficiently great enough to ameliorate the extreme neglect for this patient" (Redding & Wallace, in press, no page number available) the dissociation between the change in eye movements and the unaltered explicit perceptual bias for choosing right-sided chimaerics as appearing happier remains. Indeed, even in patients for whom prisms have shifted their notion of straight ahead sufficiently to presumably alter their 'extreme neglect', an argument can be made that only some aspects of their behaviour have been altered. For example, inspection of the figure copying performance of one of the patient's tested in the original work by Rossetti et al. (1998) demonstrates that while more of the left sided figures are copied by the patient post adaptation, left sided distortions in those copies are still evident. This observation is very much in line with our finding that while the field of exploration is shifted after prism adaptation, the actual neglect for elements on the left side of the stimulus display remains unaltered. In the context of the model proposed by Redding and Wallace (in press) for the effects of prisms in neglect, we would suggest that not only do prisms fail to alter the constricted work space evident in neglect patients (see Section 2), but they also fail to alter perceptual biases that may remain regardless of where the patient now calibrates their subjective notion of straight-ahead to be (i.e., to the right of objective straight-ahead pre-prisms and closer to objective straight-ahead post prisms). In other words, while prisms may shift (i.e., recalibrate) a patient's notion of straight-ahead and with it their 'task work space' (albeit a constricted task work space), this recalibration does little to change perceptual biases inherent to the patient's behaviour. In the context of the chimaeric faces task this dissociation may be due to salience of the task stimuli. That is, the patient (pathologically) considers the right side of the faces to be more salient than the left. This bias appears in the context of a shifted and constricted task work space prior to prism adaptation and remains unchanged after adaptation has shifted the task work space towards contralesional space. This interpretation of the limited effects of prism adaptation is also supported by a recent finding of ours in which we showed that the leftward perceptual bias on the chimaeric faces task commonly observed in healthy subjects also remains unchanged after prism adaptation despite the fact that eye movements were shifted towards the other side (Ferber & Murray, 2005).

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 et al. (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 et al. (2004) showed a reduced bias in temporal order judgments 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.

The dissociation between motor behaviour and perceptual report described above in the context of prism adaptation has also been reported independent of such a manipulation (Bisiach & Rusconi, 1990; Young, de Haan, Newcombe, & Hay, 1990; Young et al., 1992). Bisiach and Rusconi (1990) attempted to replicate Marshall and Halligan (1988) finding in one neglect patient who when asked to determine whether two vertically aligned drawings of houses were the same or different consistently neglected the left half of one of the drawings showing the house to be on fire. Despite this explicit perceptual bias, when asked which house the patient would prefer to live in she chose the house without flames at a greater than chance level (Marshall & Halligan, 1988). This seminal paper demonstrates that neglected information can nevertheless be implicitly processed to such an extent that it will influence the patient's behaviour. In their replication, Bisiach and Rusconi (1990) also asked patients to manually trace the outline of the objects they were required to make same/different judgments about. Two findings of note are relevant here; first, some patients would trace the object perfectly well but still fail to detect the fact that one of the objects was different than the other on the left hand side. This is reminiscent of the shift in exploratory eye movements in our patient that failed to alter his perceptual bias (Ferber et al., 2003). Second, some patients would trace a line through the left sided abnormality as if they were perceptually ignoring it. For example, when presented with two vertically aligned wine glasses one of which was depicted as being broken on the left half, the patient would trace the left half of the glass as if it were intact (Bisiach & Rusconi, 1990). This is reminiscent of another patient who when presented with chimaeric objects would not only claim that she perceived the right half of the chimaeric as an entire object, but would also manually trace an outline indicative of that bias. For example, when presented with a chimaeric consisting of a bowl on the right side and a football on the left the patient would claim she saw a bowl and would then trace an outline of a bowl that ignored the shape of the football that was the actual percept present (Young et al., 1992). What these examples highlight is that motor behaviours can be dissociated from explicit, consciously mediated perceptual representations relevant to those same objects that were explored motorically. We would suggest that such dissociations remain unaffected by prism adaptation.

Why might perceptual biases persist even in the face of motor behaviour (either prior to or after prism adaptation) that clearly encompasses the left side of space (or at the very least, the half of an object)? One possibility is that there is a breakdown in the communication between the dorsal 'action' pathway, which runs from area V1 to superior, posterior parietal cortex and the ventral 'perception' pathway, which runs from V1 to inferotemporal cortex (Goodale & Milner, 1992; Milner & Goodale, 1995). These two pathways are thought to subserve the control of visually guided actions and processes of object and scene perception, respectively. In order for a left sided object (or the left half of a single object) to reach consciousness there may need to be direct communication between the areas processing that object in both a motoric and a perceptual sense. There is certainly evidence to suggest a role for the inferior parietal cortex in motor planning, necessitating communication between this region and the more superiorly located dorsal stream (Mattingley et al., 1998; see also Rizzolatti & Matelli, 2003). The inferior parietal lobule (and perhaps also the superior temporal gyrus) is certainly ideally placed to integrate information necessary for both visuomotor control and visually dependent cognition. The suggestion here is that without that integration motor behaviour alone is no guarantee that an object will reach consciousness. Similarly, processing within the ventral stream alone does not guarantee that an object will reach awareness. Indeed there are many demonstrations of implicit processing of neglected stimuli indicative of continued processing of object properties despite a lack of awareness for those same properties (Berti et al., 1992; Bisiach & Rusconi, 1990; Danckert, Maruff, Kinsella, de Graaff, & Currie, 1999; Marshall & Halligan, 1988[5]). That is, despite being unaware of left sided stimuli patients may demonstrate alterations in reaction time or accuracy for stimuli they can report being aware of that is a direct consequence of the neglected stimuli. This suggests that ventral stream processing in and of itself is also not sufficient to lead to a conscious percept. We would go further here to suggest that while prisms may alter motoric behaviour carried out by the dorsal visual stream, they do not alter the processing of information in the ventral 'perception' stream. In addition, the traditional conception of processing within the ventral stream suggests that it uses allocentric, scene-based reference frames as opposed to the effector dependent, egocentric reference frames used by the dorsal stream (Goodale & Milner, 1992; Milner & Goodale, 1995). Given that prisms explicitly influence effector dependent, egocentric reference frames and the calibration of those reference frames to one another (i.e., eye-in-head needs to be calibrated to head-hand reference frames; Redding & Wallace, in press) it may not be surprising that prisms exert little influence on processing within a visual pathway that does not rely on those same reference frames. Further research that directly explores biases in spatially directed behaviours (e.g., eye and hand movements) and explicit perceptual judgments of the kind explored in the chimaeric faces task are needed to determine the extent to which these distinct impairments characteristic of the neglect syndrome can be altered by prism adaptation.

[5] Note, the Berti et al. (1992) reference investigated a patient with extinction.

This necessarily brief discussion of implicit processing in neglect and the role played by the inferior parietal cortex and/or superior temporal gyrus in communicating between the dorsal and ventral streams highlights the functional complexity of the parietal cortex and surrounding multimodal regions. This leads us into a discussion of particular regions within the parietal cortex and surrounding areas with an emphasis on how the functions subserved by these regions may contribute to deficits in attentional orienting, motor control and spatial working memory that in combination produce the characteristic loss of awareness for contralesional stimuli in neglect patients.

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

The earliest descriptions of unilateral neglect that were able to localize 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 et al., 2001; Karnath, Fruhmann-Berger, Kuker, & Rorden, 2004; Mort et al., 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 (1944) case 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).

While it is not feasible in a review of this kind to provide a definitive answer to the current controversy surrounding the critical lesion site for neglect, we feel it is important to examine the issue in light of the proposed deficits discussed above. 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, 2004) or the temporo-parietal junction (TPJ; Mort et al., 2003; see Fig. 4A for a schematic of these regions).[6]

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. In addition, the selection criteria for patients in these studies may have influenced which region the authors considered to be critical to the neglect

[6] Early studies using computerised tomography (CT) scans indicated substantial overlap in the inferior parietal lobule (IPL) proximal to the temporo-parietal junction (Vallar & Perani, 1986). Another recent study using CT and MRI scans found that the angular gyrus was the common region of overlap in neglect patients who failed to spontaneously recover from the disorder (Farné et al., 2004).

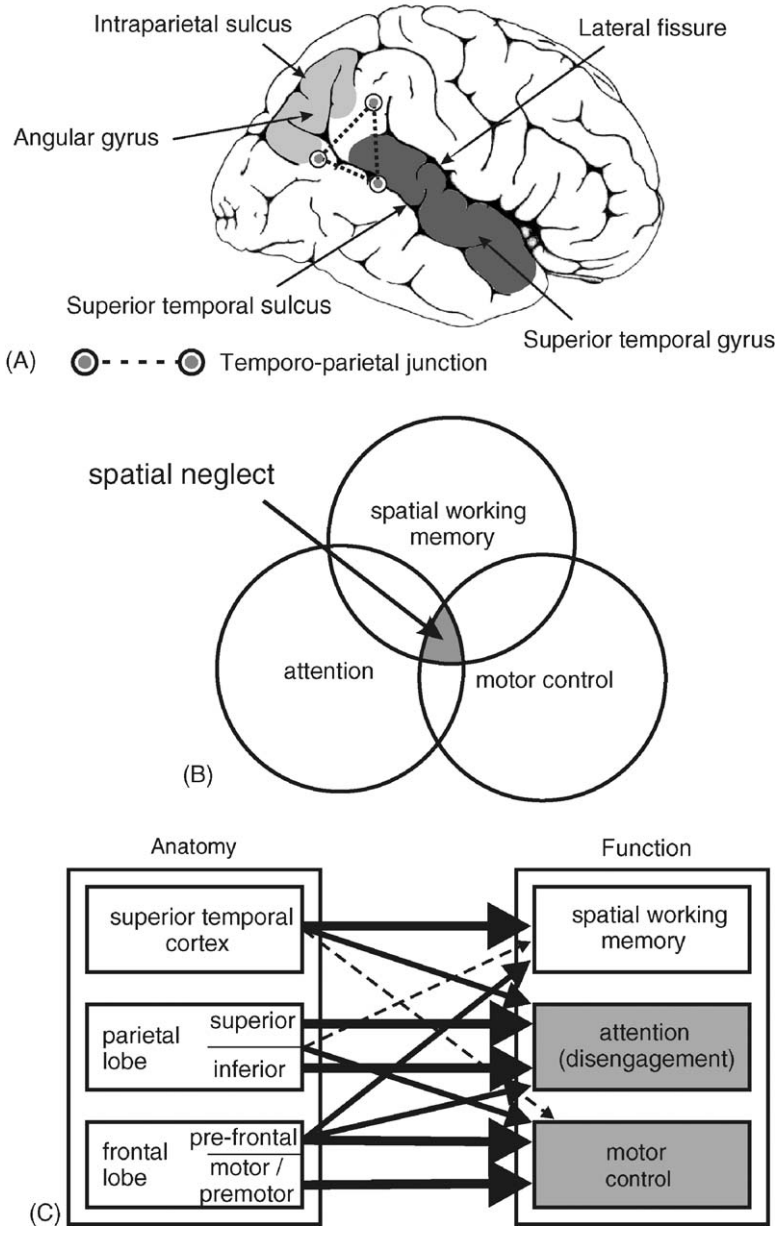

Fig. 4. (Panel A) Schematic outlining the regions of parietal and temporal association cortex commonly involved in the neglect syndrome (figure adapted from a template in Duvernoy, 1999). The region of the angular gyrus is outlined in light grey, while the superior temporal gyrus is outlined in dark grey (the posterior portion of which is commonly lesioned in neglect patients). The region of the temporo-parietal junction is indicated by the circles joined by a dotted line (see Mort et al., 2003 for a more detailed description of demarcating this region). (Panel B) Schematic representation of the constellation of impairments hypothesised to be necessary for producing the neglect syndrome. That is, the confluence of these three distinct deficits will produce the characteristic loss of awareness for contralesional stimuli. (Panel C) Hypothetical connections between anatomical regions commonly lesioned in neglect (see Panel A) and the functional attributes subserved by each region (see Panel B). While we would not suggest that a single region (e.g., the angular gyrus) is responsible for a discrete function (e.g., disengaging attention), each region is likely to have a more substantive contribution to a specific process. The different thicknesses of arrows joining anatomy to function are meant to indicate the differential contributions each region is likely to play in subserving each distinct function (i.e., a thicker arrow indicates a greater contribution from that region to controlling the function indicated). This is necessarily a hypothetical schematic requiring further research. The grey boxes in the functional column are meant to indicate those functions we hypothesise are influenced by adaptation to prismatic lenses.

syndrome. Karnath et al. (2001) assessed lesions in a group of 25 neglect patients without any evidence of visual field defects while Mort et al. (2003) included several patients with field cuts. This may have also been due to the fact that this latter study included patients suffering from posterior cerebral artery (PCA) strokes, as well as patients suffering middle cerebral artery (MCA) strokes (with the PCA group more likely to demonstrate field cuts; Mort et al., 2003). Finally, the tests used to determine the presence of neglect also differed between the two studies with Karnath et al. (2001) using several cancellation tasks plus figure copying and the baking tray test, while Mort and colleagues used a single cancellation test and a line bisection test known to be less sensitive to the presence of neglect (Ferber & Karnath, 2001).

The seemingly subtle differences in selection criteria described above may have had profound influences on the conclusions drawn in each of these studies. Arguably, the criteria chosen by Karnath et al. (2001) are somewhat stricter than those chosen by Mort et al. (2003). It is not our intention to resolve this controversy here, but it is important to point out the potential influence of the chosen methodologies. 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 explore the functions of each region – perhaps through human fMRI or monkey neurophysiology, which can both provide greater spatial precision than human 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 inferior parietal lobe, 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 (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. Inferior parietal lobe

From the very earliest studies of neglect the angular gyrus became the prime suspect as the critical lesion site needed to produce the disorder (Mattingley, 1996; Paterson & Zangwill, 1944). While more recent studies have cast some doubt over how critical this region is to neglect, evidence from trans-cranial magnetic stimulation (TMS) studies of healthy individuals is providing some insight into the role this region may play in the disorder. In addition, information from more disparate sources including psychiatric disorders such as schizophrenia, and from studies examining out-of-body experiences (OBEs), may shed a different light on what the specific role of the angular gyrus may be (Blanke, Ortigue, Landis, & Seeck, 2002; Danckert, Saoud, & Maruff, 2004; Spence et al., 1997; Tong, 2003).

Just as lesion studies in primates are capable of providing insights into specific structure–function relationships, 'virtual lesions' created by TMS can address similar questions in humans. Indeed, virtual lesions over frontal and parietal cortex have been shown to produce neglect-like response biases in healthy individuals in a line bisection task (Brighina et al., 2002; although see Fierro, Brighina, Piazza, Oliveri, & Bisach, 2001 for a discussion of similar behavioural results arising only from right parietal TMS). In addition, TMS over the parietal cortex in healthy individuals leads to a reduction in target detection for contralateral stimuli particularly when an ipsilateral stimulus is also present (Hilgetag, Théoret, & Pascual-Leone, 2001; see Nager, Wolters, Münte, & Johannes, 2004 for a similar result using somatosensory stimuli). This result is reminiscent of an extinction-like pattern often observed following parietal injury. In addition, one study found an enhancement of ipsilateral target detection following TMS over the parietal cortex (Hilgetag et al., 2001). Although a trend was evident after TMS to either left or right parietal cortex it was only significant for right parietal stimulation again mimicking the ipsilateral attentional bias evident in neglect patients (Hilgetag et al., 2001). Although not precisely indicated, it appears as though the stimulation site for this study was in the intraparietal sulcus, proximal to the angular gyrus. As well as being implicated in visually guided motor behaviour (Goodale & Milner, 1992) this region may also play a critical role in the voluntary allocation of attention throughout the visual field (Corbetta & Shulman, 2002). In a more recent study, examining the effects of TMS during either the cue period or target period of a covert orienting task, results demonstrated that voluntary orienting was disrupted only after TMS to the supramarginal gyrus during the cue period of the task (Chambers, Stokes, & Mattingley, 2004). This was the case for visual but not somatosensory cues suggesting that this region of the inferior parietal lobule is not only crucial for voluntary or strategic allocation of attention but that it is also modality specific (although see Nager et al., 2004).

In another study, examining the effects of TMS on the ability to redirect attention after an invalid cue, Chambers, Payne, Stokes, and Mattingley (2004) and Chambers, Stokes, et al. (2004) found that stimulation of the right angular gyrus impaired performance at two distinct time periods post target onset at around 90–120 and 210–240 ms. The authors suggest that this biphasic effect of TMS on the right angular gyrus reflects the fact that this region receives input from both the fast-acting retinofugal pathway (i.e., from the superior colliculus to parietal cortex via the pulvinar nucleus of the thalamus) and the slower acting geniculostriate pathway (i.e., from the LGN to V1 and from there to extrastriate and posterior parietal cortex). They go further to suggest that the early processing, presumably dependent on input from the retinofugal pathway, may be crucial for disengaging attention from its current focus, while the later processing reliant on geniculostriate input, is required for the more complicated aspects of target discrimination at the new location (Chambers, Payne, et al., 2004; Chambers, Stokes, et al., 2004). Differential effects of TMS on the functioning of the angular and supramarginal gyri have also been demonstrated using a similar cuing paradigm (Rushworth, Ellioson, & Walsh, 2001). In this study, subjects detected the presence of a target in two conditions; in the first condition four possible locations surrounded a central fixation point with simple target detection requiring an identical motor response (a single button press). In the second condition, two potential target locations above and below fixation were used that each required a distinct motor response. For simple orienting (i.e., the same motor response regardless of target location) TMS over the right angular gyrus increased reaction time to invalidly cued targets. In contrast, for what the authors referred to as 'motor attention' (i.e., distinct motor responses to targets above and below fixation) TMS over the left supramarginal gyrus increased reaction time to invalidly cued targets regardless of which hand was used to respond (Rushworth et al., 2001). Taken together with Posner and colleagues' observation that patients with parietal lesions show long reaction times to invalidly cued targets, the results discussed above provide converging evidence that the right inferior parietal lobe plays a

crucial role in disengaging attention from its current focus and reorienting towards a new location (see Corbetta & Shulman, 2002 for review). Such a 'disengage' and reorienting function is likely to play an important role in the behaviour of neglect patients in that it may be in part responsible for the ipsilesional attentional bias commonly observed on clinical and experimental tasks (see Section 3). As we pointed out earlier, however, this kind of impairment is not sufficient to explain many of the other characteristics of neglect patients including their failure to attend to ipsilesional stimuli (see Section 4). To be more explicit then, the crucial role played by damage to the inferior parietal cortex in our model is to bias attention towards ipsilesional space (Fig. 4C).

In addition to the classical understanding of the inferior parietal lobe as subserving shifts of visual attention, recent research has revealed some interesting insights into distinct functions of this brain area which we believe are linked to the neglect syndrome: a cognitive supervisor role for comparing the outcomes of intended versus executed actions and attention to bodily or corporeal awareness. For some time now researchers interested in schizophrenia have suggested the disorder affects association cortex in the parietal lobes (Frith, 1992; Pearlson, 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 (Pearlson, 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, Frith, & Wolpert, 1999; Blakemore, Smith, Steel, Johnstone, & Frith, 2000; 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 et al., 2002, 2004; Sirigu et al., 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). In addition, one recent study found reduced grey matter volume in the right inferior parietal cortex of schizophrenia patients with passivity phenomena (Maruff et al., 2005). This suggests that the right inferior parietal cortex 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). The work discussed above suggests another hypothesis for the role of the inferior parietal cortex in neglect. In addition to a disengage deficit, faulty use of forward models of intended actions may be at the heart of what Redding and Wallace (in press) refer to as the constricted task work space, common to neglect patients. That is, not only are patients with neglect biased towards attending to ipsilesional space, impaired inferior parietal function may also lead to a constriction of the region of space to which their exploratory motor behaviours are directed. Further research would be needed to explore this hypothesis.

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. That is, patients with neglect may generate faulty forward models of intended actions that are particularly impaired for contralesional space as a direct consequence of the fact that attention is already biased towards ipsilesional space (Pisella & Mattingley, 2004). Critically, this impairment may be independent of the effector used (see Danckert et al., 2002 for a description of impaired motor imagery for left and right hand movements in one neglect patient). Thus, lesions of the right inferior parietal lobule are likely to contribute to several of the characteristic impairments evident in the neglect syndrome. While the primary impairment associated with this region may involve the ability to disengage attention and reorient towards contralesional space, disruptions to exploratory and goal-directed motor behaviour, as well as disturbances in corporeal awareness, are also likely to be evident (Fig. 4C).

6.2. *The temporo-parietal junction*

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, Ollinger, McAvoy, & Shulman, 2000; Corbetta, Kincade, & Shulman, 2002; Yantis et al., 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 (Downar, Crawley, Mikulis, & Davis, 2001). Taken together, these imaging results would suggest that the right TPJ is acti-

vated when attention must be directed towards behaviourally relevant stimuli regardless of their physical location in space (Corbetta & 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 dependent 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). Distinguishing the functions of the TPJ from that of inferior parietal cortex discussed above may prove to be difficult due to the close anatomical proximity of the regions (Fig. 4A). Indeed, the most inferior portions of the inferior parietal cortex are considered part of the TPJ. One potential distinction, although necessarily speculative at this stage, may involve the direct link between the locus of attention and motor actions on one hand – presumably subserved by portions of the parietal cortex lying along the intraparietal sulcus and including the angular gyrus, and the need to interrupt the focus of attention in response to behaviourally relevant stimuli on the other – a process presumably more dependent on TPJ function and perhaps independent of specific motor actions associated with the task at hand. This contention would obviously require a great deal of further research.

6.3. The superior temporal gyrus

Above we have discussed results from human studies suggesting that the inferior parietal lobe and the TPJ subserve attentional processes relevant to the neglect syndrome. Interestingly, a lesion to the parietal lobe in the monkey brain does not lead to neglect. Instead, Watson, Valenstein, Day, and Heilman (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, Butter, and Buchtel (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, Scalaidhe, Albright, Rodman, and Gross (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, Desimore, and Gross (1981) virtually all STP neurons are visually responsive and about one-half of these neurons also respond to auditory stimuli. This multimodal character of STP neurons is in contrast to some of the TMS results in humans discussed above in which stimulation of the supramarginal gyrus impaired orienting only to visual but not somatosensory cues (Chambers, Payne, et al., 2004; Chambers, Stokes, et al., 2004). 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. While these polysensory neurons were found in more anterior regions of the macaque superior temporal sulcus, it is possible that an evolutionary shift 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, Egly, Rafal, and Beck (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 inferior parietal cortex (including 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. The strong suggestion here is that any dysfunction of such an integrative function subserved by the STG would be crucial for producing the loss of awareness characteristic of the neglect syndrome. As we have argued above, neglect cannot solely be attributed to ipsilateral biases in the deployment of attention or a constriction of explored space. An impairment in the ability to maintain an accurate representation of the spatial layout of the environment in memory (Fig. 2), together with these attentional and motor biases, will ensure that contralesional stimuli are highly unlikely to reach conscious awareness. We are suggesting that the STG is crucial in maintaining a task-relevant spatial memory within an ever changing environment (Fig. 4C).

As we have mentioned earlier, it is beyond the scope of this review to conclusively resolve the debate regarding the critical lesion site in neglect. Furthermore, just as a lesion in one discrete portion of the inferior parietal or superior temporal cortex is unlikely to be sufficient to produce neglect, nor is one specific impairment likely to adequately account for the disorder. What we are proposing here is that the confluence of impairments described above is required to produce neglect (Fig. 4B). In addition, while the subregions of anatomy discussed above are likely to interact in the control of attention, motor behaviour and spatial working memory, it may be the case that specific regions place greater weight on specific functions (Fig. 4C). Careful analysis of lesion overlap and impairment profiles in neglect and non-neglect patients using path analysis may go some way to addressing the hypothetical 'weightings' outlined in Fig. 4C. For reasons of scope we have chosen not to address the potential contribution of frontal cortex to the neglect syndrome in this review. The obvious role of the premotor and prefrontal cortex in motor planning and execution are outlined in Fig. 4C as is a potential role for frontal cortex (perhaps dorsolateral prefrontal regions in this case) in processes of working memory. Given that lesions are typically large and impairment profiles are heterogeneous, it may prove more fruitful to address these possibilities using functional neuroimaging.

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 one critical impairment 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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