Attention, Consciousness, and the Damaged Brain: Insights From Parietal Neglect and Extinction

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ABSTRACT: Milner and Goodale's (1995) model of the primate cortical visual system has been justly influential in shaping recent empirical and theoretical work on the neural basis of conscious vision. In this commentary I examine the extent to which their model accounts for recent neuropsychological findings from patients with visual neglect and extinction, two profound disorders of visual consciousness that arise after unilateral brain damage. I begin by outlining two key claims from their model: first, that the characteristic loss of awareness for contralesional sensory inputs in neglect reflects disruption of ventral, object-recognition processes, rather than dorsal processes as has commonly been thought; and second, that extinction of the more contralesional of two concurrent stimulus events is primarily a disorder of orienting and action-related attention arising from damage to the dorsal, visuomotor stream. I then present recent findings that cast some doubt on these claims. Visual neglect arising from damage to the inferior parietal lobe can involve a significant visuomotor impairment, independent of any perceptual deficit. Moreover, visual extinction can be modulated by perceptual factors that are likely to call upon the ventral object-recognition stream. These findings suggest

that neglect and extinction, two relatively common and striking disorders of consciousness, are not readily accommodated within Milner and Goodale's two visual streams model.

1. Introduction

Rarely in neuroscience does one encounter a model of brain functioning that is derived from such diverse sources as neuroanatomy, single-cell neurophysiology, normal human performance, and the effects of brain lesions on the behaviour of humans and other primates. It is rarer still to encounter a model that integrates findings across the domains of perceptual, cognitive and motor processes, in a manner that is broadly consistent with the available evidence. Yet this is precisely what Milner and Goodale's (1995) influential model of the primate cortical visual system does. Like Schneider (1969) and Ungerleider and Mishkin (1982) before them, Milner and Goodale suggest that visual information processing in the mammalian brain can be divided into two main pathways or "streams", each with a functionally distinct role. But they go further than their predecessors by proposing that the division of labour is determined by the *use* to which visual information is to be put, once it has reached the striate cortex. They suggest that a ventral stream, terminating in the inferotemporal cortex, is involved in maintaining an enduring, viewpoint-independent, representation of objects and their behavioural significance (the so-called "what" pathway). In contrast, they suggest that a dorsal stream, terminating in the posterior parietal cortex, is involved in providing an egocentric representation of objects toward which goal directed actions are planned (the so-called "how" pathway).

One of the great strengths of Milner and Goodale's (1995) monograph is that it throws down the gauntlet to those who would seek to refute the model. It contains a number of explicit predictions, particularly with respect to the consequences of damage to either stream in the human brain, and challenges us to test them empirically. As Milner and Goodale themselves hint, a model stands or falls on its details. Indeed, their own reinterpretation of the evidence used to support Ungerleider and Mishkin's (1982) original "what" versus "where" distinction is persuasive precisely because it challenges the minutiae of that model, rather than the grand facade. The rather modest aim of the present article is to make a start at examining the details of Milner and Goodale's model. Due to constraints of space, I shall limit this enterprise to a consideration of the visual and motor anomalies that arise from damage to the human posterior parietal cortex, a region that lies at the interface of the dorsal and ventral streams, and that may combine aspects of functioning that are characteristic of both. I shall present some recent data from our laboratory that bear upon some of Milner and Goodale's predictions directly, and that challenge some of the central assumptions of their model.

2. Visual Disorders After Human Parietal Damage

Lesions of the right hemisphere in humans commonly give rise to the disorder of unilateral neglect, in which patients lose awareness for visual events on the contralesional (left) side of space. Clinically patients may leave food uneaten on the left side of their plate, fail to groom the left side of their face, or miss words from the left of a printed page while reading (see Robertson & Marshall, 1993). Neglect may affect awareness for contralesional inputs in other sensory modalities as well (e.g. audition, touch), but most research has focused on the visual deficits. Although neglect has been observed after damage to a number of brain areas, the region most commonly affected is the inferior parietal lobe (IPL) (Vallar & Perani, 1986). The parietal lesion that most often results in neglect may spare the geniculostriate pathway, thereby permitting afferent transmission of visual information to the occipital lobe. Thus it seems reasonable to assume that neglected visual inputs must be processed to some extent by the preserved occipital regions. In fact there is good evidence that colour and form information is extracted for neglected stimuli (see Driver & Mattingley, 1998), and that in some cases early image-segmentation and completion processes also proceed normally (Mattingley, Davis & Driver, 1997). Several experiments have also shown that the identity and meaning of neglected visual stimuli can be activated unconsciously (e.g., McGlinchey-Berroth et al., 1993). Neglect is thus a rather paradoxical disorder in the sense that patients remain unaware of contralesional stimuli despite having well preserved mechanisms for processing their physical and semantic properties. All that is missing, apparently, is awareness.

The relative preservation of contralesional sensory inputs in neglect is further illustrated by the fact that some parietal patients do not show any deficit at all for an isolated visual event on the affected side. Their deficit only emerges when stimuli are presented concurrently on the left and right sides, in which case the more contralesional event now goes undetected. This phenomenon is known as "extinction" (Bender, 1952), and it has been taken as evidence that parietal damage causes a problem with selective attention rather than sensory processing (Driver, Mattingley, Rorden & Davis, 1997). Whereas single inputs can be attended normally, thereby entering awareness, bilateral simultaneous stimuli compete for limited attentional capacity, with the more ipsilesional of the two inputs winning the competition and gaining exclusive access to awareness (Ward, Goodrich & Driver, 1994).

In their monograph, Milner and Goodale make much of the performances of neurological patients, including those with visual neglect and extinction, to support their model of the dorsal versus ventral stream distinction. For example the disorder of optic ataxia (misreaching to targets under visual guidance), which follows damage to the superior parietal lobule in the dorsal stream, involves a deficit in performing goal directed actions to visual targets, but with relatively normal visual perception (Perenin & Vighetto, 1988). In contrast the visual form agnosic D.F., whose damage encompasses ventral stream regions of the temporal lobe, has problems in recognising objects visually, but can still use visual information to guide her movements (Goodale, Milner, Jakobson & Carey, 1991). These two disorders clearly fit with Milner and Goodale's formulation of the two visual streams hypothesis, and a significant portion of their book is devoted to showing how they do so. In contrast, the disorders of neglect and extinction fit somewhat less

comfortably within their scheme, despite the fact that there are probably more relevant data on these disorders than on optic ataxia and visual form agnosia combined. There are at least two reasons for this. First, the relevant homologies between monkey and human parietal lobe have not yet been established, so it is unclear whether the human IPL (the structure most frequently damaged in severe neglect) is part of the dorsal or ventral stream, or a functional conglomerate of both. Second, the disorder of neglect itself seems likely to be composed of several more fundamental deficits which may or may not be present in a given patient (Halligan & Marshall, 1994), thereby rendering any strict categorisation in terms of dorsal or ventral dysfunction rather difficult.

3. Neglect and Extinction: Disorders of Dorsal or Ventral Stream Processing?

Traditional textbook accounts of neglect and extinction have assumed that they are distinctly parietal impairments, characterised by an impairment of visual attention for objects and events on the contralesional side of space (Heilman, Watson & Valenstein, 1985). Such accounts are consistent with Ungerleider and Mishkin's view that the parietal cortex is part of the "where" stream, and that damage there leads to problems in attending to contralesional stimuli and in forming enduring representations of their spatial locations. Thus, disorders such as neglect, extinction, and optic ataxia, have been viewed as different manifestations of the same basic impairment of the "where" stream following parietal lobe damage.

In contrast, Milner and Goodale suggest that neglect, extinction, and optic ataxia are dissociable disorders that reflect damage to different areas of the parietal cortex. They suggest that the human parietal lobe in fact consists of two functional subcomponents; the superior parietal lobe (SPL), which they argue is the termination of the dorsal visuomotor stream; and the IPL, which they suggest, contrary to Ungerleider and Mishkin, is part of the *ventral* perceptual stream, but with some evolutionarily new capacities, such as the ability to manipulate viewer-centered representations for tasks like mental rotation and map reading. They suggest that these latter mechanisms, which are uniquely human, may have been co-opted from those originally evolved in the dorsal stream for the visual guidance of movement. This division of the parietal cortex into a dorsal visuomotor region (SPL) and a ventral perceptual region (IPL) generates a number of testable predictions with respect to the consequences of circumscribed damage to them. For instance, damage to the SPL should lead to problems in visually guided action in the context of intact perception and awareness, whereas damage to the IPL should produce deficits in visual perception and awareness with relatively preserved action. To what extent do the available patient data support Milner and Goodale's division of the parietal lobe into dorsal and ventral regions?

There is considerable evidence that damage to IPL, more than any other brain region, produces the classic symptoms of unilateral neglect (e.g., Vallar & Perani, 1986),

including loss of awareness for the contralesional sides of individual objects, regardless of their locations in egocentric coordinates, as well as perceptual problems in judging the size of visual stimuli in the contralesional hemispace, and even difficulties in constructing mental representations of visual arrays in the mind's eye (See Robertson & Marshall, 1993). The pathognomonic loss of awareness for contralesional stimuli in neglect also lends indirect support to Milner and Goodale's notion that only ventral stream representations, such as those mediated by IPL, are available to consciousness.

In contrast, damage to SPL produces optic ataxia, a disorder characterised by misreaching to targets under visual guidance, in the absence of visuoperceptual disturbance, and with preserved awareness for contralesional events (Perenin & Vighetto, 1988). This evidence is consistent with the view that SPL is part of the dorsal stream and that damage there causes deficits in visuomotor control. Milner and Goodale also suggest that visual extinction, the selective loss of awareness for contralesional stimuli that compete for selection with simultaneous ipsilesional events, reflects dorsal stream damage. They cite several studies that have claimed to show a double dissociation between neglect and extinction (e.g., Barbieri & De Renzi, 1989) as well as work by Posner and his colleagues (Posner, Walker, Friedrich & Rafal, 1984) in which patients with superior parietal damage exhibited extinction-like patterns in manual responses to lateralised visual targets. Thus, contrary to the traditional view that extinction is merely a mild form of neglect, Milner and Goodale suggest that visual extinction reflects biased activity of dorsal-stream neurons that subserve "orienting and action-related attention".

We have recently obtained evidence from studies of parietal patients which appear to challenge Milner and Goodale's conceptualisation of neglect and extinction as disorders of ventral and dorsal stream processes, respectively. Here I shall focus on data from just two such studies. For further details of related studies of parietal patients the reader is referred to Driver and Mattingley (1998).

4. A Motor Role For Human Inferior Parietal Cortex

In clinical tests for neglect (e.g., cancellation of visual stimuli scattered on a page), patients are required to make manual responses toward target items. Patients' failures to respond to contralesional items are typically taken as evidence for a spatial deficit in perceptual awareness, but it is also logically possible that such problems arise from the patients' failure to enact appropriate motor responses toward contralesional stimuli, with perception remaining relatively intact. The fact that neglect arises even in tasks that involve simple verbal responses suggests that a contralesional motor deficit cannot be the *sole* underlying cause of neglect behaviour. But the question of whether neglect patients may nevertheless exhibit some motor biases in addition to their clear perceptual deficits has been the focus of considerable recent debate. Certainly there is strong neurophysiological evidence that cells in the monkey posterior parietal cortex (the homologue of human IPL) are selectively tuned to the kind of motor response (saccade versus reach) being planned toward a visual target (Snyder, Batista & Andersen, 1997).

There is also some evidence suggesting that neglect patients are impaired when initiating and executing movements (even with the ipsilesional hand) toward contralesional targets (Mattingley & Driver, 1997). However, in most cases those neglect patients with motor biases have had large frontal and/or striatal lesions, rather than the posterior damage that is normally associated with neglect. These findings have clear implications for Milner and Goodale's hypothesis that neglect after inferior parietal damage is a purely perceptual disorder, and that visuomotor deficits (such as optic ataxia) arise only after dorsal stream damage to SPL. They also speak to their suggestion that directional motor impairments in neglect reflect damage to more anteriorly located "output" regions, rather than superior parietal structures at the "visuomotor interface".

Several studies have reported an association between frontal and/or striatal damage and motor biases in patients with clinical neglect (Bisiach, Geminiani, Berti & Rusconi, 1990; Mattingley, Bradshaw & Phillips, 1992; Tegnér & Levander, 1991). Unfortunately the data from many of these studies are ambiguous, either because they can be explained in terms of purely perceptual impairments, or because they relied on methods that pit perceptual and motor demands against one another, resulting in a high degree of spatial incompatibility between stimuli and responses. The apparent association of motor biases with frontal neglect (as noted by Milner and Goodale) may therefore have arisen simply because such individuals are likely to experience difficulties with highly incompatible tasks, rather than any specific problem in responding to contralesional visual targets. We therefore devised a visuomotor task that allowed us to separate the potential contributions of perceptual and motor biases, while preserving the natural visuomotor correspondences involved in reaching to visual targets. Our aim was to determine whether motor biases occur in patients with neglect, independent of any perceptual biases. We tested left neglect patients whose damage was restricted to the right inferior frontal cortex, to test Milner and Goodale's prediction that motor biases are associated exclusively with frontal damage. We also tested a group of left neglect patients with circumscribed right IPL lesions to test the hypothesis that this region is part of the ventral stream, and consequently has no role in visuomotor control (see Mattingley, Husain, Rorden & Driver, 1998).

The basic task required patients to reach with their ipsilesional (non-paretic) right hand from a start-key toward a target LED that appeared unpredictably to the left or right of a central fixation point (see Figure 1). The rationale behind the task was to keep sensory information regarding target location constant, while varying the direction in which the patient had to reach by changing the start position of the responding hand. Thus at the beginning of each trial the visual events always occurred at the same retinal location, regardless of where the responding hand was positioned. We predicted that if neglect arises from a perceptual bias alone, our manipulation of hand-start position should have no effect on the time required by patients to initiate motor responses to visual target events. On the other hand, if patients suffered from a motor bias in initiating directional reaches, their initiation times should have been differentially affected by the changes in reach direction induced by varying the start position of the hand. Patients were tested in three start positions: a central position, which was located midway between the left and right LEDs; an extreme left position, in which both target LEDs were located to the right

of the hand; and a corresponding position on the extreme right, such that both target LEDs were to the left of the hand.

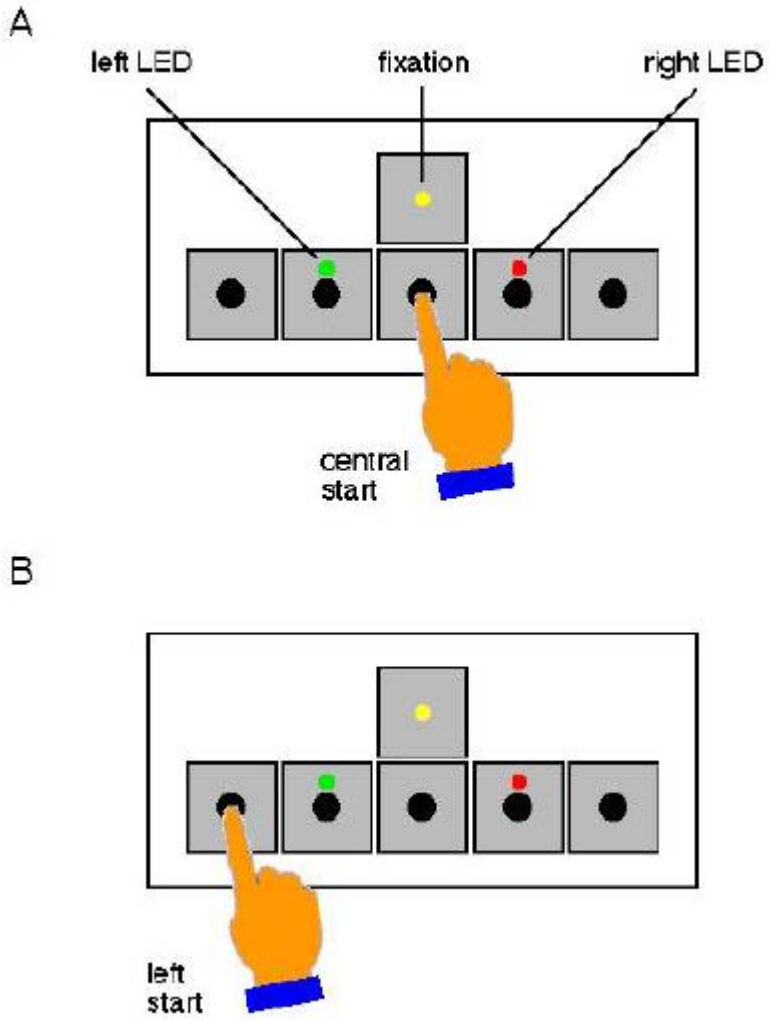

Figure 1

Schematic illustration of the reaching task used by Mattingley et al. (1998) to measure directional motor impairments in neglect patients with right inferior parietal damage. Patients fixated the central yellow LED and then made a speeded reach (using their right hand) to push a button beneath a peripheral green LED, ignoring any red distractor LED. The figure shows a trial in which the target appeared on the left side, although it could occur with equal probability on the right. (A) From a central-start position left targets require a leftward movement and right targets a rightward movement. Reaches to the left target were initiated very slowly by the inferior parietal patients, consistent with their left neglect. (B) From a left-start position both right and left targets require a rightward movement. Reaches to the left target were significantly faster in this condition relative to that illustrated in (A), suggesting that part of the problem in responding to left targets from a central start is due to motoric slowing in the initiation of leftward reaches. Changes in reach initiation times as a function of the hand's start position were not found for neglect patients with right frontal damage.

Both the frontal and parietal patients were slower to react to left-sided targets than to right-sided targets when their hand began in the central position. The result is ambiguous, however, in the sense that it is consistent with a perceptual bias, a motor bias, or both. The crucial data were obtained from the condition in which the patients initiated their reaches from the extreme left position, so that either target would require a reach toward the right (ipsilesional) side. Neglect patients with IPL damage showed a dramatic reduction in the time taken to initiate reaches to a left target, compared with the time taken to initiate reaches to the same target from a central start (which required a leftward rather than a rightward movement). Since the visual locations of potential targets were the same at the beginning of each trial, the significant reduction in the IPL patients' movement-initiation times to left-side targets can be attributed unambiguously to a motor bias favouring rightward over leftward reaches. Crucially, although neglect patients with inferior frontal damage were slower to respond to left-side than right-side targets, they did not show any modulation of this asymmetry as a function of hand-start position, indicating an *absence* of a motor bias in these individuals.

In a control experiment we tested whether the benefit for left-side targets from a left start position may have been due to attentional cueing from the responding hand as it rested on the patients' neglected side. The task was identical to the initial reaching study, except that patients now pressed the start key itself (which was located centrally, or to the left or right, as before) to indicate when they had detected a target. The aim was to replicate the visual events and hand positions of the initial experiment, while eliminating the requirement for a directional motor response. In this "no-reach" task, both patient groups were again slower to respond to left- versus right-side targets from a central start position, indicating a perceptual deficit for left targets. More importantly, however, there was no longer a significant reduction in IPL patients' response times to left-side targets with a left-hand start, compared with central- and right-start positions. It is therefore the *direction* of the required reach, rather than any attentional cueing from the hand at the beginning of the trial, that underlies the parietal patients' performances in the reaching task. The frontal patients were again unaffected by the hand-start manipulation, confirming the absence of a motor bias in these individuals.

The results of this study provide the first unambiguous evidence that neglect patients with IPL damage may exhibit a specific impairment in initiating contralesional movements. The findings seem contrary to the predictions of Milner and Goodale, who suggest that the human IPL is part of the ventral stream, with no role in overt visuomotor control. Of course it remains possible that the IPL in humans forms part of a "third" visual stream, in which synergies arise between dorsal and ventral mechanisms. Milner and Goodale themselves hint at this in their suggestion that "new functional areas may have evolved ventrally within the human parietal lobe which can co-opt some of the transformational algorithms that originally evolved for the control of movement" (1995, p.111). But they suggest that such "transformational algorithms" probably evolved for manipulating viewer-based visual representations which are called into play during such tasks as mental rotation and map reading. These conscious, imagery-based abilities seem a far cry from the rapid, ephemeral, unconscious representations that are evidently crucial for such abilities as visually guided reaching.

The parietal patients in our reaching study had no evidence of optic ataxia (spatial errors in visually guided manual responses), nor did their lesions extend into SPL, the region that Milner and Goodale hypothesise is the termination of the human dorsal stream. Our findings also challenge the widely held view (advocated by Milner and Goodale) that motor deficits in neglect arise from anterior lesions, with posterior damage producing only perceptual biases. In contrast, our findings are entirely consistent with single-unit studies of posterior parietal cortex in monkeys (Snyder et al., 1997) showing that neurons there modify their firing patterns to visual stimuli in accordance with the specific motor actions that are planned towards them. Taken together, the recent patient and monkey data suggest that the IPL has a key role in the early stages of motor planning. This proposal is particularly significant in the context of Crick and Koch's (1995) recent suggestion that visual awareness may be closely tied to the initial planning of motor responses. Thus, although I agree with Milner and Goodale that the ventral stream, and the IPL in particular, is critical for visual awareness, I believe the reason for this may lie in its role in forming intentions to act toward particular locations in space.

5. Visual Extinction: A Failure of Orienting and Action-Related Attention in the Dorsal Stream?

As noted above, Milner and Goodale argue that neglect and extinction are distinct neuropsychological disorders that reflect damage to the ventral and dorsal streams, respectively. Although this idea is contrary to the textbook story that extinction is simply a more subtle manifestation of neglect (Heilman et al., 1985), it is strongly supported by observations that individual patients may occasionally show neglect without extinction (Barbieri & DeRenzi, 1989; Driver et al., 1997), as well as the more familiar pattern of extinction without neglect . Unfortunately, drawing clear distinctions between the two disorders is complicated by the fact that extinction can be *operationally defined* (i.e., poorer performance for contralesional events under bilateral versus unilateral stimulation), whereas neglect has only a rather vague clinical definition (a failure to orient toward or respond to contralesional events). The difficulty in providing a definition of neglect that is *independent* of the task used to measure it gives a clue that it may not in fact be a single entity, but a conglomeration of distinct impairments that merely co-occur after parietal damage, as Milner and Goodale suggest.

As we saw for their predictions regarding the roles of the inferior and superior parietal cortex in motor behaviour, however, it is in the details that Milner and Goodale's conceptualisation of extinction as a dorsal stream phenomenon breaks down. There are two lines of evidence that run contrary to the strict association of extinction with dorsal stream damage, one anatomical and the other perceptual. Recall that Milner and Goodale suggest that extinction arises predominantly from damage to the superior parietal cortex in humans. In support of their idea, they cite findings from positron emission tomography (PET) studies showing that the superior parietal cortex is selectively activated when normal subjects shift their attention covertly in order to track a visual target on a

computer display (Corbetta, Miezin, Shulman, & Petersen, 1993). They also cite the classic study by Posner et al. (1984) in which superior parietal damage produced an extinction-like reaction time pattern in right hemisphere patients, whereas more inferior parietal damage had no such effect.

The first problem for Milner and Goodale's account is that there is an extensive literature demonstrating that extinction is a common outcome of virtually any unilateral lesion (Berti, Allport, Driver, Dienes, Oxbury & Oxbury, 1992; Vallar, Rusconi, Bignamini, Geminiani & Perani, 1994), not just those that affect SPL and related structures within the dorsal stream, as they suggest. Indeed, in his classic treatise on extinction Bender (1952) showed that under appropriate testing conditions extinction could even be elicited in patients with circumscribed *spinal* injuries, with no involvement of the cerebrum whatsoever. The pathognomonic extinction-like reaction time (RT) pattern shown by Posner et al.'s parietal patients has subsequently been observed in cases with a diverse range of cortical and subcortical damage outside the dorsal stream (e.g., Morrow & Ratcliff, 1988). Moreover, a recent study has challenged the original association of severe extinction-like deficits with predominantly SPL damage (Friedrich, Egly, Rafal & Posner, 1998). Using the same Posner cueing paradigm, Friedrich et al. found that patients with discrete lesions of the temporoparietal junction showed a clear extinction-like RT pattern, whereas patients with more superior parietal damage showed no such pattern. Thus there seems little evidence for Milner and Goodale's proposal that extinction is a dorsal stream deficit, and even less to suggest that extinction is principally associated with SPL damage.

A further inconsistency in Milner and Goodale's view that extinction is primarily an impairment of orienting due to superior parietal damage, is that even subtle changes in the nature of the stimulus events tend to have a profound impact on whether patients are able to report them. Thus patients may show extinction for contralesional events in just a single sensory modality (e.g. for pairs of visual stimuli), or in several different modalities (e.g. for pairs of visual, tactile, and auditory stimuli). Recent evidence suggests that extinction may even occur *crossmodally*, such that an ipsilesional stimulus in one modality (e.g., vision) extinguishes patients' awareness of a simultaneous contralesional stimulus in a different modality (e.g., touch) (Mattingley, Driver, Beschin & Robertson, 1997). It seems likely that these heterogeneous manifestations of extinction arise from damage to several attentional processes distributed over a range of brain areas, rather than from a basic (supramodal?) orienting mechanism located in the superior parietal cortex.

Even in cases where extinction is restricted to a single modality, however, the precise nature of the stimuli themselves may exert a profound effect upon whether patients become aware of contralesional targets. As illustrated in Figure 2, several recent studies have found that contralesional visual events can be "rescued from extinction" if they can be grouped together with simultaneous ipsilesional events on such dimensions as contrast polarity, collinearity, connectedness, illusory filling-in, apparent occlusion in depth, and surroundedness (Mattingley et al., 1997; Ward et al., 1994; Gilchrist, Humphreys & Riddoch, 1996). A recent study has also found suggestive evidence that extinction is reduced when contralesional visual events are grouped with ipsilesional events by

common motion (Pierson & Ralley, personal communication; see Figure 2). There is even evidence for modulation of extinction via top-down influences such as familiarity of the grouped configuration (Ward et al., 1994) and object identity (Berti et al., 1992; Ward & Goodrich, 1996). These latter effects do not seem consistent with Milner and Goodale's suggestion that extinction is a deficit of orienting, since in many cases whether a contralesional target will reach awareness depends not on its mere location in space, but on processes of object recognition and extraction of meaning that arise in the ventral stream.

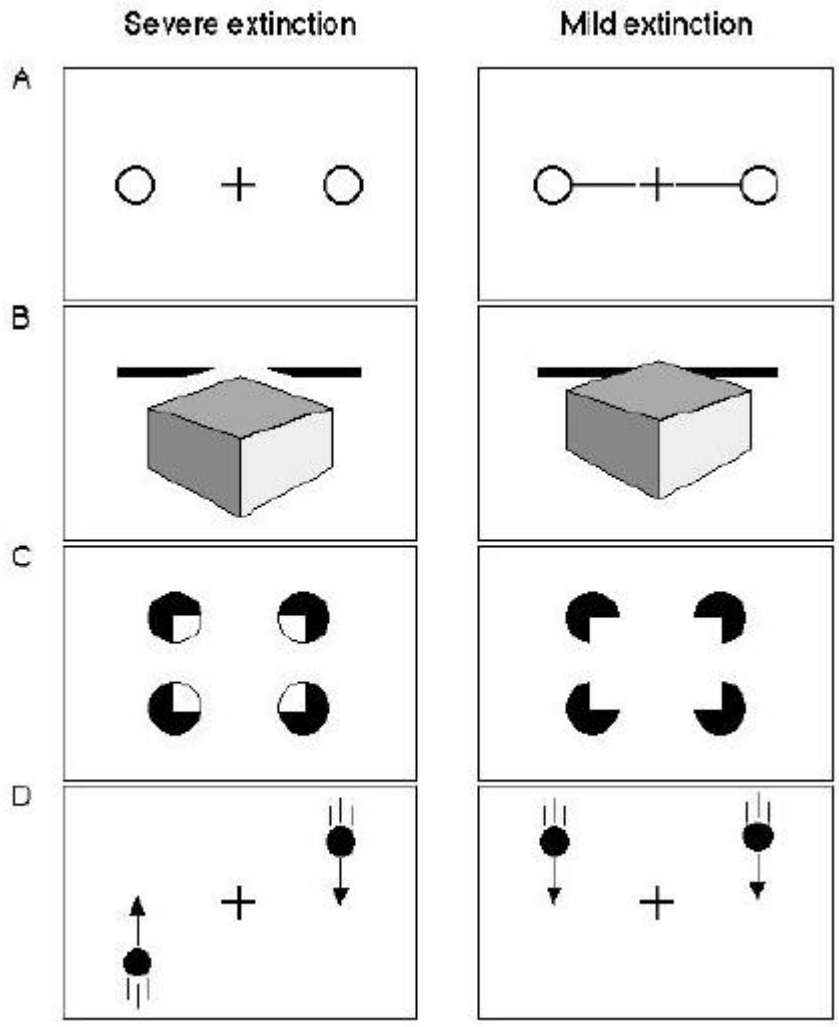

Figure 2

Schematic illustration of conditions in which contralesional visual stimuli can be "rescued" from extinction following unilateral damage. In all examples patients were required to fixate centrally and detect target stimuli flashed briefly on the left or right side alone, or on both sides simultaneously (as shown for all examples here). On "catch" trials no targets were displayed, in order to measure any guessing. Patients indicated their detection of target events by responding "left", "right", "both" or "none". For all display types detection of *single* contralesional targets was close to 100% correct.

Detection of contralesional targets in *bilateral* displays was severely impaired when the events were ungrouped (left panels), but improved significantly when they could be grouped to form a single object (right panels). (A) Target circles in linked vs. unlinked configurations (from Driver, Goodrich, Ward & Rafal, submitted). (B) Target bars as separate objects, or grouped by amodal completion behind an occluding cube (from Mattingley, Davis & Driver, 1997). (C) Targets were quarter segments removed from black circles. The narrow arcs veto any grouping. The display without arcs forms a single subjective rectangle by modal completion (from Mattingley, Davis & Driver, 1997). (D) Target spots moving at 180 out of phase do not group. Spots moving in phase are grouped by common motion.

Clearly the disorder of extinction does not fit neatly into the current Milner and Goodale model. From an anatomical perspective, extinction can clearly arise after damage to a number of cortical and subcortical brain areas, and is not dependent upon dorsal stream damage. Moreover, the disorder itself seems more than just a problem of orienting or "action-related attention"; in some cases it can be modulated by perceptual factors that probably rely more upon object recognition mechanisms in the ventral stream than visuomotor mechanisms in the dorsal stream. But this need not imply that extinction cannot also be modulated by activity within the dorsal action system. Milner and Goodale themselves have suggested that the severity of visual extinction may occasionally be modulated by the hand that is used by the patient to generate a response. More specifically, they predict that a patient's extinction should be *more severe* when the contralesional hand is used to respond than when the ipsilesional hand is used, presumably because the mechanisms for contralesional orienting and for contralesional limb control are both subserved by the patient's damaged superior parietal lobe.

Contrary to Milner and Goodale's prediction, we recently found that self-initiated movements of the contralesional limb dramatically *reduced* the severity of visual extinction in a right hemisphere patient (rather than increasing severity as the theory predicts), even though the limb movements were not visible to the patient, and carried no predictive value with regard to the likely location(s) of visual targets (Mattingley, Robertson & Driver, 1998). Our study was based upon previous research demonstrating the beneficial effects of the so-called "limb-activation" technique in right hemisphere patients with neglect (e.g., Robertson, North & Geggie, 1992). In our study, a right hemisphere patient was required to detect brief visual targets flashed randomly to the left or right of fixation, or bilaterally. In blocks of trials, the patient initiated the onset of each visual presentation by pressing a key located on the left or right side of the display, using either her left or right hand. In a baseline condition, in which the experimenter initiated each trial, the patient showed strong visual extinction (i.e., failure to detect the left-sided member of a bilateral pair, with good detection of the same left-sided target presented alone). When the patient initiated trials with a left-sided keypress, however, she was now aware of significantly more contralesional targets in bilateral displays, a result that held for both hands, although it was stronger for the left hand. Initiating trials with a right-sided keypress had no significant effect on the patient's performance relative to baseline, thereby ruling out any generalised effect of the motor activity on alertness or motivation. Thus, although visual attention evidently *can* be modulated by motor activity, as suggested by Milner and Goodale, it seems that the effect of contralesional limb movement in patients is to *boost* attention for inputs on the affected side, rather than to attenuate them as predicted by Milner and Goodale.

6. Conclusions

The aim of this review has been to examine Milner and Goodale's model of the primate cortical visual system in the light of recent findings from humans with discrete lesions of the parietal cortex. In particular, I have focused upon their claim that the inferior and superior divisions of human parietal cortex belong to the ventral and dorsal streams, respectively. They propose that the inferior parietal cortex is dependent upon inputs from the ventral stream, and that its primary functional role is in constructing and manipulating visual representations of objects. Although they suggest that the human IPL may have "co-opted" some dorsal stream mechanisms, Milner and Goodale clearly deny any role for the IPL in visuomotor control. Our recent experiments on neglect patients with discrete IPL lesions have yielded evidence that challenges this view. When reaching under visual guidance to left-sided targets, our right hemisphere patients exhibited a motor deficit that was independent of their perceptual bias. Thus the human IPL seems to play a key role in early motor planning or "intention", a finding that is supported by recent single-unit recordings in the posterior parietal cortex of the monkey (Snyder et al., 1997). Our patient data are also consistent with recent suggestions that visual awareness itself may arise from initial planning of motor responses towards relevant objects (Crick & Koch, 1995).

Recent data from patients with visual extinction also challenge Milner and Goodale's suggestion that the superior parietal cortex (part of their dorsal stream) is exclusively involved in orienting and action-related attention, and that damage here is the primary cause of extinction. In fact most clinical evidence suggests that extinction, at least as it is currently defined, cannot be attributed to damage to any particular brain structure; rather, it seems to arise after unilateral damage to virtually *any* cortical or subcortical site. The fact that extinction occurs not only for visual stimuli, but also for events in the auditory and tactile modalities (and crossmodally), may imply that it reflects a fairly general form of competitive imbalance between neural circuits in the two hemispheres (Duncan, 1996). It is also clear from recent studies that extinction involves more than a mere failure to orient attention. As outlined earlier, both low-level and high-level properties of visual stimuli can have a profound effect on whether or not a contralesional event reaches the patient's awareness.

The picture that emerges from the clinical literature on extinction seems more consistent with recent models of attention, which suggest that limits in processing capacity arise from competition between concurrent stimuli for the control of distinct neural circuits (Desimone & Duncan, 1995; Duncan, 1996). These circuits represent different aspects of sensory inputs, such as their colour, form, motion, and so forth; but their activity is integrated such that all the properties from a single perceptual object come to dominate activity across all the circuits at one time. According to this view, damage to one hemisphere will give a competitive advantage to one particular class of object over another, depending on which brain area has been affected by the lesion. Conversely, activation of circuits in one hemisphere, via, for example, limb activation, will tend to

increase activity in all related circuits. This model neatly predicts the beneficial effects of contralesional limb activation on detection of contralesional targets in right hemisphere extinction (Mattingley, Robertson & Driver, 1998), a result that is *opposite* to that predicted by Milner and Goodale. A further (as yet untested) prediction of Duncan's integrated competition model is that extinction may be more severe for some classes of stimuli than others, depending on which neural circuits are most affected by the lesion.

Milner and Goodale's model of the two visual systems has been justly influential in shaping current theorising about the functional organisation of the primate visual system. In this brief review I have ignored the very substantial body of evidence that supports their model, focusing instead on data that challenge just one or two of its central assumptions. My arguments have been deliberately provocative in the hope that they may stimulate others to examine more closely some of the specifics of the Milner and Goodale model, particularly as it applies to human parietal lobe function.

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