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Perceptual awareness and its loss in unilateral neglect and extinction

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Abstract

We review recent evidence from studies of patients with unilateral neglect and/or extinction, who suffer from a loss of awareness for stimuli towards the affected side of space. We contrast their deficit with the effects of damage to primary sensory areas, noting that such areas can remain structurally intact in neglect, with lesions typically centred on the right inferior parietal lobe. In keeping with preservation of initial sensory pathways, many recent studies have shown that considerable residual processing can still take place for neglected or extinguished stimuli, yet without reaching the patient's awareness. This ranges from preserved visual grouping processes through to activation of identity, semantics and emotional significance. Similarly to 'preattentive' processing in normals, such residual processing can modulate what will enter the patient's awareness. Recent studies have used measures such as ERPs and fMRI to determine the neural correlates of conscious versus unconscious perception in the patients, which in turn can be related to the anatomy of their lesions. We relate the patient findings to neurophysiological data from areas in the monkey parietal lobe, which indicate that these serve as cross-modal and sensorimotor interfaces highlighting currently relevant locations as targets for intentional action. We speculate on the special role such brain regions may play in perceptual awareness, seeking to explain how damage to a system which appears primarily to code space could eliminate awareness even for non-spatial stimulus properties at affected locations. This may relate to the extreme nature of 'winner-takes-all' functions within the parietal lobe, and their correspondingly strong influence on other brain areas.

Keywords: Spatial neglect; Extinction; Awareness; Parietal cortex; Attention

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1. Introduction

Unilateral spatial neglect is a relatively common and disabling neurological disorder after unilateral brain damage. It is characterized by a lack of awareness for sensory events located towards the contralesional side of space (e.g. towards the left following a right lesion), together with a loss of the orienting behaviours, exploratory search and other actions that would normally be directed toward that side. Neglect patients often behave as if half of their world no longer exists. In daily life, they may be oblivious to objects and people on the neglected side of the room, may eat from only one side of their plate, read from only one end of a newspaper page, and make-up or shave only one side of their face. The spatial bias towards one side can also be apparent in many simple paper-and-pencil tests. When required to search for and mark all target shapes on a page, the patients may cancel only those towards the ipsilesional side. When bisecting a horizontal line, they may err towards that side, and when drawing from memory, or copying a picture, they may omit details from the contralesional side (Fig. 1).

The characteristic spatial bias of neglect patients has been observed in some form for all of the sensory modalities (vision, audition, touch, proprioception, even smell; see Bellas, Novelly, Eskenazi, & Wasserstein, 1988; Heilman, Watson, & Valenstein, 1993; Mesulam, 1981; Vallar, Guariglia, Nico, & Bisiach, 1995). Analogous spatial biases may also be apparent in motor-output systems, as we discuss later (e.g. with eye or hand movements being biased towards the 'good' ipsilesional side; see Bisiach, Geminiani, Berti, & Rusconi, 1990; Coslett, Bowers, Fitzpatrick, Haws, & Heilman, 1990; Heilman, Boweres, Coslett, Whelan, & Watson, 1985). Some patients may neglect their own contralesional limbs, attempting to climb out of bed without moving these, even though they have no primary motor weakness on that side. Others may be paralyzed on the contralesional side, yet remain unaware of this. In general, neglect patients often have little insight into their deficits for the affected side, especially in the acute stage. In the longer term, they may acknowledge that they can 'miss things' on the affected side, yet continue to do so.

Thus, neglect ostensibly involves a dramatic loss of awareness, and of appropriate action, for sensory events towards the affected side. The paradox is that this can arise even though the primary sensory pathways for processing the neglected information may all still be intact. That is, patients may show profound neglect for sights, sounds and touches towards the affected side, even though they are by no means blind, deaf or insensitive on this side. Here we will argue, in keeping with recent advances in neglect research, that this paradox can be resolved to some extent by relating the plight of neglect patients to particular aspects of *normal* cognition. Even neurologically healthy people can fail to see, hear or feel salient stimuli, provided that their selective attention is engaged elsewhere, as we shall describe (see also Merikle, Smilek, & Eastwood in this volume). Perceptual awareness is not determined solely by the stimuli impinging on our senses, but also by which of these stimuli we choose to attend. This choice seems pathologically restricted in neglect patients, with their attention strongly biased towards events on the ipsilesional side.

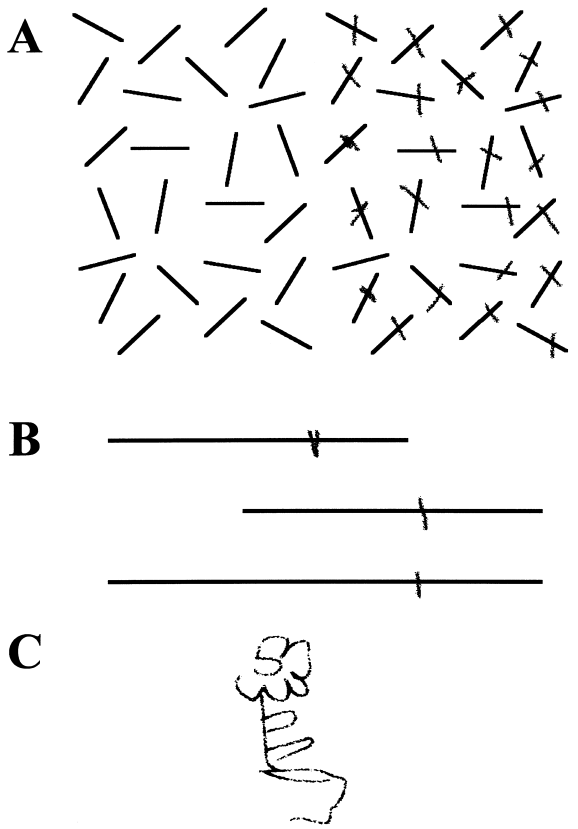


Fig. 1. Examples of deficits found in patients with left spatial neglect on typical clinical tasks. (A) Line cancellation task. The patient has to mark each of many small lines that are spread out on a sheet of paper presented in front of them. Typically, the patient fails to detect some lines on the contralesional side, even when given considerable time to complete the task. More sensitive cancellation tasks may involve the marking of a target letter or shape among distractors. (B) Line bisection task. The patient is asked to mark the midpoint of long horizontal lines and deviates towards the right ipsilesional side, as if neglecting the left contralesional extent. Such an ipsilesional bias is often greater when the lines are positioned more to the left side. It can be partly alleviated when the patient is cued to the contralesional end of the line, for instance by reporting a letter there. (C) Neglect in drawing is characterized by the omissions of all or parts of the elements on the contralesional side. This can occur when the patient is required to draw an object from memory (as here, a flower in a pot) or to copy a drawing template.

2. Basic anatomy of neglect, and multiple components to the clinical syndrome

Unilateral spatial neglect can be observed in some form after various unilateral brain lesions, but is most common and long-lasting in humans when the damage involves the inferior parietal lobe, particularly in the right hemisphere. Studies seeking to determine the critical cortical areas, by looking for overlap in the lesions of different cases, have pointed to the angular and supramarginal gyri (Fig. 2A), corresponding to Brodmann areas 39 and 40 (Heilman et al., 1993; Leibovitch et al.,

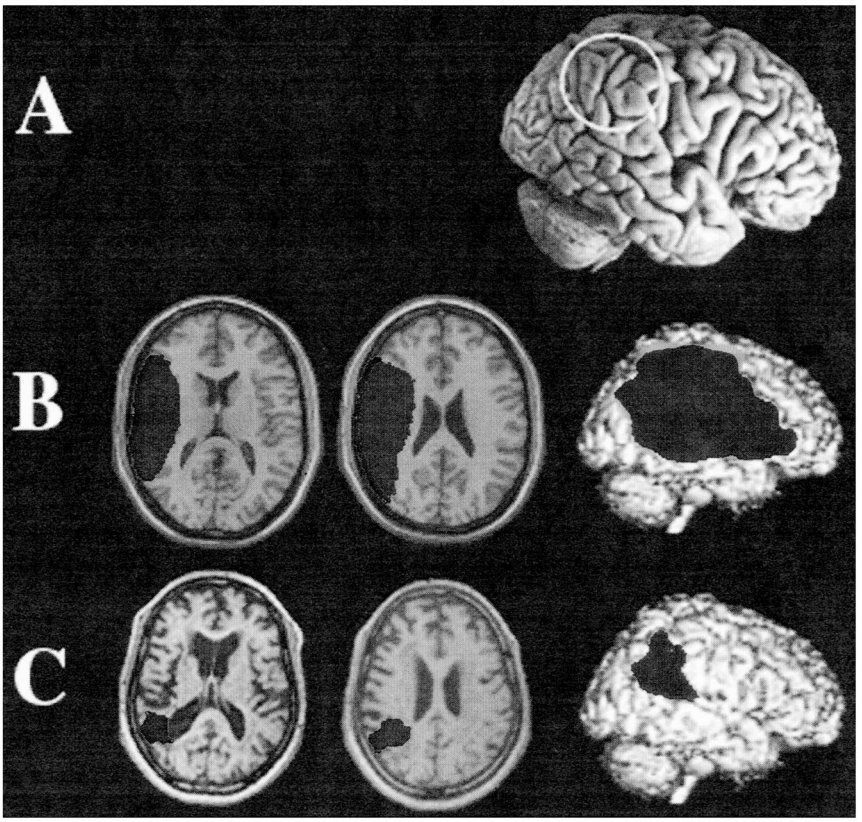


Fig. 2. Topography of parietal damage in unilateral spatial neglect. (A) Lateral view of the right hemisphere in the human brain. The supramarginal and angular gyri in the inferior parietal lobe (circled) are most often involved in severe chronic cases of neglect. Early visual areas in the posterior part of the brain and anterior motor areas are typically spared. Individual patients may have varying extent of damage to other cortical or subcortical regions outside the parietal lobe. While a large lesion in the territory of the middle cerebral artery is very common (B, patient SD), a smaller focal lesion may be sufficient to produce marked visual extinction and neglect (C, patient KG). It is likely that such differences in lesion size may lead to significant differences in clinical signs of neglect, though it is still largely unknown which differences.

1998; Perenin, 1997; Vallar, 1993; Vallar & Perani, 1986). Concomitant damage to white-matter fibre-bundles beneath the parieto-temporo-occipital junction is also common (Leibovitch et al., 1998; Samuelsson, Jensen, Ekholm, Naver, & Blomstrand, 1997), and may result in a larger functional lesion than that implied by considering only the grey-matter damage (e.g. see Gaffan & Hornak, 1997).

It is controversial whether parietal damage in monkeys can produce an equivalent syndrome to spatial neglect in humans. The exact homology between particular human parietal areas and monkey parietal areas is also debated. Architectonic studies suggest that human areas 39 and 40 may correspond to monkey areas 7a

and 7b, respectively located caudally and rostrally below the intraparietal sulcus (Eidelberg & Galaburda, 1984). However, while unilateral excision of these areas in the monkey can produce some spatial biases in perception and behaviour (Deuel & Regan, 1985; Heilman, Pandya, & Geschwind, 1970; Mesulam, 1981), these deficits are usually considered milder and more transient than human neglect. It has been argued that more inferior excisions, extending into the superior temporal sulcus, produce a stronger spatial bias in monkeys (Luh, Butter, & Buchtel, 1986; Watson, Day, Valenstein, & Heilman, 1994), as does complete parietal ablation combined with hemispheric disconnection (Gaffan & Hornak, 1997). One difficulty in any monkey–human comparison for neglect is that comparable tasks have rarely been used across the species. It seems clear that spatial biases towards the ipsilesional side can be produced by several lesions in the monkey, including those brain areas implicated in human neglect, but these deficits are usually more specific and transient than for human neglect. Moreover, the right-hemisphere specialization so apparent in humans is not evident in monkeys.

The lesions in human neglect patients are typically much larger and more diffuse than the focal surgical lesions used in monkey studies, being caused by natural accidents such as strokes. Although it has become common to refer to 'parietal patients' in the neglect literature, in fact severe cases often have a very extensive lesion that will encompasses part of the frontal and temporal lobes, plus subcortical structures, in addition to the parietal lobe (e.g. see Fig. 2B). Patients with lesions of this kind will clearly suffer from more than one problem. Clinical neglect often involves a combination of deficits, each of which may exacerbate the others. Since the anatomical extent of brain damage varies from one case to another, different patients can also exhibit somewhat different combinations of deficits. As noted earlier, neglect can involve not only spatial biases in perceptual awareness, but also in motor output (Bisiach et al., 1990; Coslett et al., 1990; Heilman et al., 1985; Mattingley, Bradshaw, & Phillips, 1992; Mattingley, Husain, Rorden, Kennard, & Driver, 1997). Famously, spatial neglect can also be apparent in visual imagery and memory for some patients (Bisiach & Luzzatti, 1978; Meador, Loring, Bowers, & Heilman, 1987). Recent studies suggest that imaginal neglect can dissociate from perceptual neglect, and may have a more anterior anatomical substrate (Coslett, 1997; Guariglia, Padovani, Pantano, & Pizzamiglio, 1993).

The spatial biases towards one side that are the primary cause of neglect may also be exacerbated by some further deficits which do affect both sides of space. For instance, cortical damage around the right temporal-parietal junction biases visual perception towards fine local details of visual scenes, rather than the more global properties apparent at larger spatial scales (Lamb, Robertson, & Knight, 1990; Robertson, Lamb, & Knight, 1988). Since this particular brain area is typically included in the large strokes that produce neglect (e.g. see Fig. 2B), many neglect patients will have a local bias in addition to their bias towards one side. This may be an important element in some aspects of the neglect syndrome (Halligan & Marshall, 1991a, 1993), and in the clinical tests used to diagnose it. For instance, both drawing and cancellation tests may reflect the patients' tendency to lock onto small details (see Fig. 1).

Neglect may also be exacerbated by sensory loss or paralysis in those patients who suffer from these additional deficits. While blindness and paralysis are not primary causes of neglect (see below), a field cut which completely wipes out input from the left visual field may exaggerate any existing tendency to neglect that side of space (e.g. Doricchi & Angelelli, 1999); similarly, an inability to move the left of the body may exaggerate any tendency to respond only to right events.

Thus, each patient will have a particular constellation of deficits and anatomical damage. Nevertheless, there are many commonalities between neglect patients, as we emphasize here. Likewise, we shall tend to focus anatomically on the effects of (right) inferior parietal damage, as this is the most common denominator across patients with severe neglect. Human neglect can occasionally be seen after lesions restricted to subcortical (Bogousslavsky et al., 1988; Rafal & Posner, 1987; Vallar, 1993; Vallar & Perani, 1987; Watson & Heilman, 1979) or frontal areas (Damasio, Damasio, & Chui, 1987; Heilman & Valenstein, 1972; Husain & Kennard, 1996, 1997), with the latter apparently involving ventral lateral cortex (Husain & Kennard, 1996) rather than dorsal superior regions as formerly thought (Mesulam, 1981). However, such neglect is usually more transient than that following inferior parietal damage, and may involve remote metabolic effects on parietal structures in the chronic state (Perani, Vallar, Cappa, Massa, & Fazio, 1987). The full anatomical picture that emerges for neglect is thus of an extended network (Heilman et al., 1993; Mesulam, 1981), involving subcortical, frontal, cingulate and superior temporal structures, but with the inferior parietal lobe as the major hub. Although many neglect patients have large lesions, severe and prolonged neglect can still be observed in cases with a more focal lesion centred on the right inferior parietal lobe (e.g. Fig. 2C).

3. Neglect as a window on the neural basis of awareness, and the contrast with blindsight

Clinical descriptions of neglect were documented by German neurologists a century ago (e.g. Loeb, 1885; Oppenheim, 1885; Poppelreuter, 1917; Zingerle, 1913). However, the syndrome subsequently received less systematic attention than other classical neurological syndromes (such as aphasia or agnosia), perhaps because of the paucity of suitable theoretical ideas or analogies for grappling with it. Moreover, despite the dramatic loss of awareness for one side, neglect was rarely considered in discussions of the neural basis of conscious perceptual experience until recently. Most discussions tended to focus on the neuropsychological syndrome of 'blindsight' instead. We shall contrast neglect with blindsight here. While some similar issues arise, there are fundamental differences.

The excitement about blindsight stemmed from reports of unconscious residual vision (Pöppel, Held, & Frost, 1973; Weiskrantz, 1986; Weiskrantz, Warrington, Sanders, & Marshall, 1974) in patients who were consciously blind for a region of the visual field, following a lesion to a corresponding part of primary visual cortex in the occipital lobe. This area provides a complete retinotopic map of the visual field

(as do many subsequent areas of visual cortex). It has been known since early this century that damage to parts of this map leads to blindness for corresponding parts of the visual field (a 'field cut' or 'hemianopia'). So nobody was surprised by the loss of conscious vision in the Pöppel et al. (1973) or Weiskrantz et al. (1974) patients. What was surprising was the report that some visual functions (e.g. the direction of eye movements or pointing) could still take place for stimuli presented within the retinotopically blind region, in an apparently unconscious fashion. The challenge of blindsight ever since has been to explain this residual function, *not* the loss of awareness. It was simply taken as read that removing primary visual cortex (and thus the input to many subsequent visual cortical areas also) would remove visual awareness.

Some similar issues arise for neglect, in the sense that there is a spatially-specific loss of awareness, and also (as we will show) considerable residual but unconscious processing for the information which escapes awareness. However, in other respects the situation could not be more different. The challenge in neglect is very much to explain the loss of awareness itself, rather than the residual processing, because so many of the neural pathways conventionally associated with conscious perception (including primary sensory areas) remain intact in many neglect patients. For instance, some neglect patients have no visual field cut whatsoever. Unlike an occipital blindsight patient, they are able consciously to report an isolated light, wherever it appears. Yet they still show severe visual neglect in daily life, where visual events can occur on all sides at once, as we emphasize below. Conversely, many patients with field cuts on one side, due to damage in primary visual cortex for one hemisphere, will exhibit no visual neglect whatsoever, even though they are consciously blind for isolated lights presented on one side of their retinae. Clearly, neglect is not equivalent to retinotopic blindness, and the effects of parietal damage are very different from those of occipital damage, even though we have no reason to doubt the parietal neglect patient's insistence that they do not consciously see the information they neglect, any more than for the occipital blindsight patient's reports of seeing nothing in their field cut.

4. The spatial nature of neglect, and further contrasts with primary sensory loss

The loss of awareness in neglect differs from that in blindsight in its spatial nature also. The visual field cut of an occipital patient is absolutely tied to a region on the retina, in accordance with the damage to the retinotopic map in primary visual cortex. Usually there is a fairly sharp demarcation between the affected region and the intact region, often corresponding to one sensory hemifield versus the other in patients with unilateral damage.

This is very different from the spatial nature of the loss in conscious perception for neglect patients. First, there is rarely a sharp divide in performance at some anatomical midline. Instead, there is more typically a *gradient* of impairment, with performance gradually declining for stimulus locations that are further in the affected direction (Kinsbourne, 1987; Ladavas, Petronio, & Umilta, 1990; Smania

et al., 1998). Second, in neglect patients, performance for a given stimulus at the eye, ear or skin can depend strongly on whether any other stimulation is provided at the same time. An occipital patient with a left visual field cut will not consciously detect a light on the left even if presented in complete isolation, in an otherwise dark room. But many patients with left neglect would detect such a light with relative ease. Their deficit would only become apparent if two lights were presented simultaneously, in which case they would typically miss whichever light was presented further to the left (Bender & Teuber, 1946; Critchley, 1953; Loeb, 1885; Oppenheim, 1885; Wortis, Bender, & Teuber, 1948). This deficit during double simultaneous stimulation is known as 'extinction', as the right event is said to 'extinguish' the other event from awareness. We later discuss this phenomenon and its boundary conditions at length.

A further difference between primary sensory loss and neglect is that the spatial deficit in neglect can depend strongly on the current posture of the patient, because it reflects an impairment at a higher level of spatial representation than provided in primary sensory areas. In patients with damage to primary visual cortex, a blind retinal hemifield will remain blind irrespective of current eye-in-orbit posture, moving with any change in gaze direction. But in neglect patients, the same visual stimulus at a fixed retinal position may be neglected or detected depending on the current orbital position of the eye (Kooistra & Heilman, 1989; Vuilleumier, Valenza, Perrig, Mayer, & Landis, 1999), or of the head on the neck (Karnath, Schenkel, & Fischer, 1991; Vuilleumier et al., 1999). For instance, a left visual field stimulus that was neglected with the head and eyes directed forwards may be detected when presented at the same point on the retina but with the eyes and/or head turned right (see Fig. 3). Passively twisting the trunk towards the left, while leaving the eyes and head facing straight ahead, can also bring an otherwise neglected left visual field stimulus back into awareness (Karnath et al., 1991), again suggesting that extraretinal factors can influence visual neglect. This is a remarkable finding; whether the patient sees a stimulus depends not only on the visual information entering the eyes, but on body posture also.

Neglect within touch can also be affected by postural changes that would have no influence on a primary (somatotopic) loss. Tactile stimuli that are not felt on the contralesional arm may eventually be perceived if the arm is placed further towards the ipsilesional side of the trunk (Aglioti, Smania, & Peru, 1999; Smania & Aglioti, 1995). By contrast, damage to primary somatosensory cortex would cause a loss of sensation for the corresponding body part no matter where it is placed.

When facing forwards in a standard upright posture (as in many clinical examinations and experiments), numerous potential frames of reference are usually aligned, and thus potentially confounded (e.g. egocentric co-ordinate systems such as those centred on the retina, head, or trunk are all aligned, along with more allocentric co-ordinates such as those centred on the computer screen or testing page, the objects appearing upon these, the table and the room, etc). A number of neglect studies have now attempted to uncouple some of these spatial frames of reference by various manipulations such as tilting the patient or the display (e.g. see Bisiach, 1997, for a review) in a complex literature that we cannot describe fully

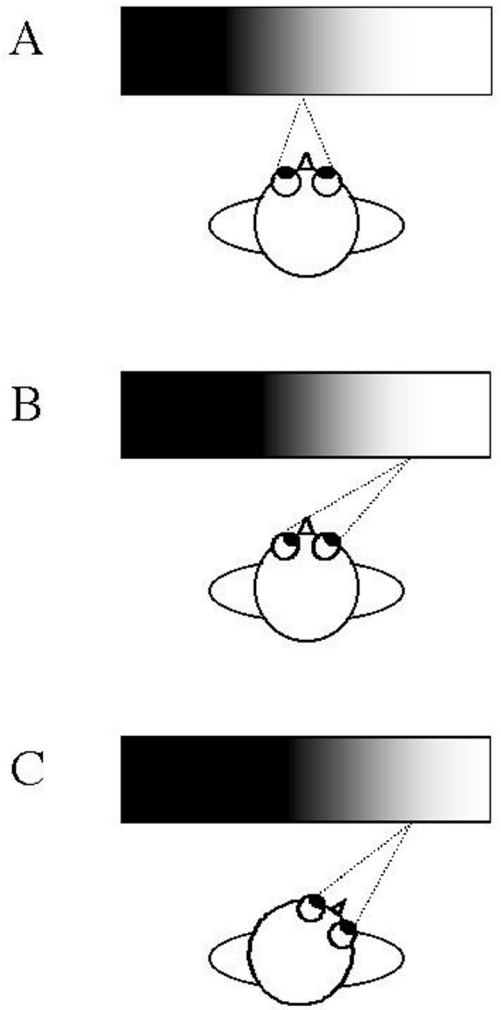


Fig. 3. Schematic illustration of varying spatial co-ordinates in unilateral neglect and effects of posture. A hypothetical patient with right-brain damage and left spatial neglect is depicted as seen from above the head when facing a hypothetical visual scene. Perception of visual stimuli generally improves as a gradient from the contralesional side (black area, complete unawareness) to the ipsilesional side (white area, normal awareness). This spatial gradient could in principle be defined with respect to the retina (line of gaze, depicted by dashed lines), head, or body, which are often aligned in experiments. If the patient keeps his gaze straight ahead (A), he may fail to detect stimuli falling on the contralesional hemiretina. But if the patient directs his gaze to the right while keeping the head straight (B), or if he turns both gaze and head to the right (C), stimuli falling on the contralateral hemiretina may now be perceived. Such effects demonstrate that spatial neglect differs from hemianopic visual field cuts. Also, they indicate that neglect is partly determined by the head and/or trunk position in addition to the primary retinal afferents, and that posture signals can modulate awareness of contralesional stimuli. Adapted from Vuilleumier et al. (1999).

here. Suffice to say that these studies demonstrate there is more to the spatial nature of neglect than mere primary sensory loss within fixed retinal or somatotopic co-ordinates. They imply that neglect can arise at higher levels of spatial representation, some of which involve the integration of different sources of spatial information (e.g. retinal location together with postural signals about the current dispositions of the eye in its orbit, and/or the head on the neck, etc; see Pouget, Fisher, & Sejnovski, 1993; Pouget & Sejnovski, 1997). In a later section, we try to relate this aspect of neglect to current findings on such spatial integration at the level of single neurons in the monkey parietal lobe.

Another complexity to the spatial nature of neglect, which again differentiates it from primary sensory loss, is that visual neglect for one side of *far* space (well out of the patient's reach) has been reported to dissociate from neglect for one side of *near* space (within reach). This was in fact predicted for humans on the basis of pioneering monkey-lesion studies (Rizzolatti & Camarda, 1987; Rizzolatti, Matelli, & Pavesi, 1983). After an initial report of one patient who exhibited neglect in near but not far space (Halligan & Marshall, 1991b), a further case was described who showed the opposite pattern, neglecting visual stimuli in far-left space but not near-left space, even when retinal visual angles and response requirements were closely matched (Vuilleumier, Valenza, Mayer, Réverdin, & Landis, 1998). However, it is probably more common to observe similar neglect in near and far space for most patients (Cowey, Small, & Ellis, 1994; Pizzamiglio et al., 1989).

A final difference between neglect and primary sensory loss is that neglect patients more frequently lack insight into their deficit. Patients with visual field cuts may realize that their vision is restricted, and can compensate with eye movements. Neglect patients are typically more disabled, in effect 'neglecting' their own contralesional deficit.

5. Attentional perspectives on neglect

By now we hope to have convinced the reader that there is more to neglect than primary sensory loss. Neglect patients can be unaware of sights, sounds, touches and body parts towards their left in daily life, even though they are not blind, deaf or insensitive on that side. As we foreshadowed earlier, this seeming paradox may be resolved by considering that, in some circumstances, this applies to neurologically healthy individuals also, when their *attention* is engaged elsewhere. We can fail to see something, even when a perfectly clear image is available on our retina, if attending elsewhere (e.g. Mack & Rock, 1998; Rees, Russell, Frith, & Driver, 1999; Rensink, O'Regan, & Clark, 1997; see also Merikle et al. in this volume), or fail to hear something which is no quieter at the ear than the conversation we are currently following. Our perceptual awareness thus depends not only on stimulation of our senses, and on the projection of this information to primary sensory areas of cortex, but also on what we choose to attend.

Of course there are some limits on our freedom of choice here; a particularly intrusive, painful or emotive stimulus can be hard to ignore, as documented in the

extensive literature on normal selective attention (for recent reviews see Egeth & Yantis, 1997; Wells & Matthews, 1994; see also Merikle et al. in this volume). Nevertheless, this literature contains countless examples of the dramatic phenomenal and objective effects that selective attention can exert on perception. Classic experiments on selective listening (Broadbent, 1958; Cherry, 1953) show that when presented with two spoken messages (e.g. one on the left, one on the right) we can choose to follow one or the other, resulting in a rich awareness of the contents for that attended message, but very restricted awareness for the other. Similar phenomena have long been established in vision (e.g. Neisser & Becklen, 1975) even for constant retinal inputs (Rock & Guttman, 1981). There is currently renewed interest in such phenomena under the heading of 'inattentional blindness' (e.g. Mack & Rock, 1998; Rensink et al., 1997; see also Merikle et al. in this volume).

In addition to these textbook effects of selective attention on phenomenology and report, attention can affect many objective psychophysical measures of perceptual performance, including thresholds to detect a stimulus (e.g. Muller & Humphreys, 1991). Finally, data from many different methods in neuroscience (e.g. single-cell recording, ERPs, functional imaging) converge to show that the brain response to a given stimulus can vary substantially, depending on the current attentional state of the observer (see Parasuraman, 1998, for reviews). Thus, a stimulus which is optimal for driving a particular visual neuron will produce greatly attenuated firing if a behaving monkey is instructed to attend elsewhere (Chelazzi, Duncan, Miller, & Desimone, 1998; Gottlieb, Kusunoki, & Goldberg, 1998; Moran & Desimone, 1985; Treue & Maunsell, 1997).

Against the backdrop of these findings on normal selective attention, the dilemma of a neglect patient who remains unaware of sensory information, even though primary sensory projections are demonstrably intact, may seem less paradoxical. It is tempting to suggest that such patients may neglect left-sided information because their attention is pathologically locked onto right-sided information. Proposals of this kind have become increasingly frequent in recent years (e.g. Humphreys & Riddoch, 1993; Posner, Walker, Friedrich, & Rafal, 1984; Rafal, 1994), and can be traced back to suggestions by Kleist (1923) and others (Brain, 1941; Critchley, 1953; Heilman & Valenstein, 1979; Mesulam, 1981) that neglect involves 'inattention'. However, attentional accounts for neglect are not universally popular. Sceptics point out that little explanation is offered until the concept of attention is fleshed out in mechanistic terms. Fortunately, there is now a vast body of knowledge on normal attentional mechanisms within both psychology (e.g. see Pashler, 1998) and neuroscience (e.g. Parasuraman, 1998).

For many years it was conventional to contrast attentional accounts for neglect with those positing a failure to construct internal representations of contralesional space (e.g. Battersby, Bender, Pollack, & Kahn, 1956; Bisiach & Berti, 1987; Bisiach, Luzzatti, & Perani, 1979; Denny-Brown & Banker, 1954; Hécaen, 1972; Paterson & Zangwill, 1944; Scheller & Seidemann, 1931). In our view, this is rather a false dichotomy. On the one hand, many aspects of selective attention operate spatially (e.g. Tsal & Lavie, 1993); hence, attention has spatially-selective repre-

sentational consequences. On the other hand, the ultimate cause of neglect is the loss of neurons which selectively represent certain parts of space for specific functions.

The lesion in many neglect patients is so large (see Fig. 2B) that one may doubt whether it could correspond to a surgical removal of just 'selective attention' and nothing else. As we emphasized earlier, most patients have several concurrent deficits. Nevertheless, it remains useful to think of one primary component of neglect as involving 'inattention', and we justify this later in relation to the damaged neural systems. For now, the usefulness of the analogy between neglect in patients and inattention in normals can be judged by the new questions and answers it has provoked in recent years, as described below.

6. Extinction as a difficulty in attending to multiple targets

The analogy with normal attention seems particularly apt for one aspect of the neglect syndrome that we mentioned earlier, namely *extinction* during double simultaneous stimulation. Recall that many neglect patients can detect a single left-sided event in isolation, missing this only when presented in combination with another event further to its right. Such extinction can actually be found within hemifields as well as between them, but for simplicity we will stick to the example of two events in different fields. Extinction can be observed within vision, hearing or touch, and even between two events in separate sensory modalities (e.g. Mattingley, Driver, Beschin, & Robertson, 1997), as we describe later. The phenomenon suggests that the patients' spatial bias is most detrimental when multiple events compete for attention at the same time, as will usually be the case in the cluttered scenes of daily life.

Although extinction can be seen in some form after various lesions (Vallar, Rusconi, Bignamini, Geminiani, & Perani, 1994), and may represent only one aspect of the heterogeneous neglect syndrome (Liu, Bolton, Price, & Weintraub, 1992), it is often present in patients with focal parietal lesions that leave primary sensory pathways intact (e.g. Fig. 2C). Moreover, a recent study found that neglect as measured clinically (by conventional cancellation scores; see Fig. 1A) correlated directly with the rate of extinction by distracting ipsilesional shapes in a computerized test (Vuilleumier & Rafal, 2000; see also Morrow & Ratcliff, 1987). We take the position that while extinction is by no means the whole story for neglect, it encapsulates a critical general principle that applies for most aspects of neglect, namely that the patients' spatial deficit is most apparent in *competitive* situations, where information further towards the 'good' ipsilesional side comes to dominate information that would otherwise be acknowledged towards the contralesional side (see Dennett in this volume for further discussion about competition in relation to awareness).

Other authors (e.g. Milner, 1997) have argued that extinction may reflect a separable 'attentional' component of neglect, associated with a more superior lesion. However, the anatomical aspect of this argument was based on a single study (Posner et al., 1984), and more recent studies suggest a critical lesion that resembles the typical anatomy of neglect (see Driver, Mattingley, Rorden, & Davis, 1997;

Friedrich, Egly, Rafal, & Beck, 1998, for further discussion). In any case, extinction is one aspect of the neglect syndrome that clearly involves a loss of perceptual awareness, and so is particularly relevant to the theme of this volume.

Extinction may relate to a well-established but often overlooked attentional limitation in neurologically healthy people (Duncan, 1980; Eriksen & Spencer, 1969; Shiffrin & Gardner, 1972). In many situations, we are able to monitor several streams of information for a specified target as efficiently as one stream. This apparently implies that our sensory systems can transduce all the incoming information at once. Yet if several streams happen to each contain a target at the same time (or close together in time, as in so-called 'attentional blink' paradigms; e.g. Raymond, Shapiro, & Arnell, 1992), people will typically miss some of these targets. This shows that we cannot become aware of multiple targets all at once, even if our sensory systems have transduced them. As we have argued at length elsewhere (e.g. Driver et al., 1997; Vuilleumier & Rafal, 2000), this seems analogous to the plight of a patient suffering from extinction, who is able to detect a single target in any location, with a deficit only for multiple concurrent targets. There are of course a few obvious differences between the normal observer and the patient. One can usually predict which target the patient will miss (the one further towards the contralesional side, presumably because the lesion has reduced its competitive strength). Second, the patient has this difficulty even with just two salient suprathreshold stimuli, whereas the normal attentional limitation with multiple targets arises only for very brief or masked stimuli in more cluttered displays. This suggests a non-spatial restriction in capacity for the patients (Duncan et al., in press; Husain, Shapiro, Martin, & Kennard, 1997; Robertson, 1989; Vuilleumier & Rafal, 2000) in addition to their spatial bias. Indeed, 'simultanagnosia' (a restriction in the number of objects than can be concurrently seen; Holmes & Horax, 1919; Rafal, 1997) may be a critical component of the deficit, over and above any bias to one side. Extinction can be regarded as a pathological, spatially-specific exaggeration of the normal difficulty in distributing attention to multiple targets. This leads to several testable new predictions, as described below.

7. Preserved 'preattentive' processing in extinction: grouping effects

Recent patient studies show that considerable processing can still take place prior to the level at which extinction arises. As a first approximation, such processing typically corresponds well with that considered to take place 'preattentively' in the normal system (see Merikle et al. in this volume). As in normals, this processing can determine which information will attract attention and reach awareness in the patients, and which will escape awareness.

In normal vision, the limitation in attending to multiple concurrent targets can be reduced if these are linked into a single object or group by Gestalt principles such as good continuation, closure, and so on (e.g. Baylis & Driver, 1993; Duncan, 1984). This suggests that the retinal image may initially be segmented into separate groups or objects, so that competition for attention acts on the resulting segmented percep-

tual units (Duncan, 1984). If extinction is indeed a pathological exaggeration of the normal difficulty with multiple concurrent targets, then we can predict that it too should be reduced if the two competing events could be grouped together. Several recent findings from right-parietal patients with left extinction confirm this prediction (e.g. Gilchrist, Humphreys, & Riddoch, 1996; Mattingley, Davis, & Driver, 1997; Ward, Goodrich, & Driver, 1994), suggesting that 'preattentive' grouping mechanisms may still operate despite the pathological spatial bias of the patient to influence whether a particular stimulus will reach the patient's awareness.

Kanizsa subjective figures (Kanizsa, 1976) provide one interesting stimulus for addressing this issue. They comprise spatially discontinuous elements that can nevertheless yield the subjective percept of a single object, due to modal surface completion (e.g. see the bright white rectangle in Fig. 4B). Moreover, their neural basis is relatively well understood. Neurophysiological work in monkeys indicates that neurons in early cortical visual areas of the occipital lobe (e.g. V2) respond to the illusory contours in Kanizsa stimuli as for real contrast-defined contours (Von Der Heydt, Peterhans, & Baumgartner, 1984). Furthermore, psychological evidence in normal observers suggests that grouping of elements into Kanizsa figures can arise 'preattentively' (i.e. without any need to attend to each set of inducers in turn; see Davis & Driver, 1998).

Mattingley, Davis, and Driver (1997) used Kanizsa rectangles in an extinction study with a patient suffering from an extensive lesion centred on the right inferior parietal lobe, which spared early visual areas (similar to Fig. 2B). The patient had to report[1] whether she saw quarter-segments being removed from four black circles on the right, left, or both sides of central fixation (see illustrative display sequences for bilateral trials in Fig. 4). She extinguished most left-sided events in bilateral trials when narrow arcs on the circles prevented the formation of a connecting subjective surface (Fig. 4A). However, her extinction was virtually abolished when such arcs were removed, as the bilateral events then yielded a single subjective object (a bright white rectangle, apparently superimposed on the black circles) due to modal surface completion (Fig. 4B). This suggests that extinction is reduced when the concurrent target events can be linked into a single subjective object, becoming allies rather than competitors in the bid to attract attention. It also implies that the visual segmentation processes generating subjective figures were still intact in the patient, despite her extensive lesion and associated spatial bias. The latter conclusion was also reached in a study of three neglect patients, which again used Kanizsa stimuli, but now in the task of bisecting 'real' or subjective figures (Vuilleumier & Landis, 1998). The subjective figures produced performance like that for real (physically complete) figures, and unlike control stimuli that did not support grouping into a subjective figure.

[1] As with other investigations of awareness in normal people, we have to rely on such reports as indices of phenomenal awareness (though see Block in this volume). As emphasized later, the conclusions from the patient studies agree well with those from studies of normal attention. For the latter studies, a sceptical but neurologically-healthy reader can confirm the phenomenology of most attentional demonstrations for themselves!

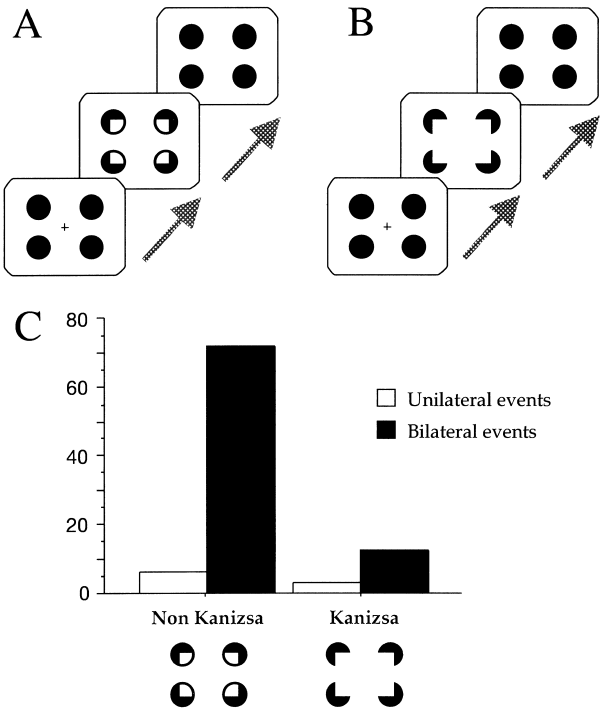


Fig. 4. Reduction of extinction by illusory surface completion in Kanizsa figures. Example sequence of events in one trial when bilateral events did not form (A) or did form (B) a single object bounded by illusory contours (bright white rectangle in B). Arrows depict time between successive frames. In each trial, four black circles were first presented around a central fixation cross; then quarter-segments could be briefly removed from all four black circles (bilateral trials) as shown here, or just from two circles on one side (right or left), or not at all. These displays were used to test a patient with right-hemisphere damage and left extinction. Her task was to detect and report the side(s) of the brief offsets. The percentage of contralesional (left) events missed in each condition is shown below (C). The patient exhibited little extinction when removals from both sides produced a subjective Kanizsa rectangle (as in B), although there was a marked extinction when small arcs remained on the circles so as to prevent the formation of an illusory surface (as in A). Unilateral left events were correctly detected on most trials in both situations. Adapted from Mattingley, Davis, and Driver (1997).

Visual extinction is now known to be modulated by other forms of grouping, including connectedness, collinearity of edges, and similarity of contrast polarity, with less extinction in all cases when the two events can be grouped together (Driver et al., 1997; Gilchrist et al., 1996; Ward et al., 1994). Pavlovskaya, Sagi, Soroker, and Ring (1997) showed a similar phenomenon using formal psychophysical threshold measures and gabor-patch stimuli (which have well-characterized physical properties and are thought to drive highly selective subsets of visual neurons in occipital cortex). Extinction was reduced when two patches in different hemifields were co-oriented and coaxial. Moreover, this depended on the distance between the gratings in a manner consistent with known lateral interac-

tions between neurons at relatively early stages of cortical visual processing, possibly within primary visual cortex.

In summary, these recent studies show that visual extinction can be modulated by grouping processes, consistent with the prediction derived from the analogy with normal attentional limitations. This implies that 'preattentive' grouping processes still take place on contralesional inputs despite the lesion and associated spatial bias of the patients, and that visual extinction reflects 'attentional' competition between segmented objects, not just between particular points on the retina. The preserved segmentation processes can have a dramatic influence on whether or not the patient will become aware of a stimulus at a particular point on the retina. The extensive literature on so-called 'object-based' neglect, which we do not have space to review in full here, makes very similar points (e.g. see Buxbaum & Farah, 1997; Driver, 1999, for reviews).

8. Task effects on extinction

In standard visual extinction studies, the patient is asked to report whether anything is seen on the left, the right, on both sides, or not at all. A seemingly minor change to this task can have a dramatic effect on what the patient reports seeing. Vuilleumier and Rafal (1999) presented stimuli in one, two, or four possible locations across hemifields (Fig. 5). When asked to report *where* the shapes appeared (i.e. on the left, right or both sides), as usually required, three right-parietal patients consistently extinguished left-sided stimuli in bilateral displays. However, when shown the same stimuli but now asked to *enumerate* them (i.e. one, two, or four), these patients had no difficulty reporting 'two' or 'four' shapes in bilateral displays; extinction was eliminated. This remarkable change in outcome seems consistent with evidence in normals that enumerating a few ($\leq$4) visual elements may exploit special 'subitizing' mechanisms (Dehaene & Cohen, 1994; Mandler & Shebo, 1982), which allow individual elements to be processed together 'preattentively' as a single numerable group (Trick & Pylshyn, 1993), rather than each being attended in a serial manner, as for the counting of larger sets.

In this situation, the reduction in extinction was produced by a change in task-set, not in the stimuli. Nevertheless, the general principle may be similar to that for the grouping experiments described above; extinction is reduced when the concurrent events can be attended as a single perceptual unit, becoming allies rather than competitors in the bid to attract attention.

9. The fate of extinguished stimuli

The previous section focused on factors determining whether a stimulus will be extinguished from awareness in the patients. The results implied some preserved processing for contralesional events, with this (preattentive) processing determining which perceptual units will go on to act as competitors (for attention), and thus which will reach awareness. We turn now to consider the fate of those contralesional

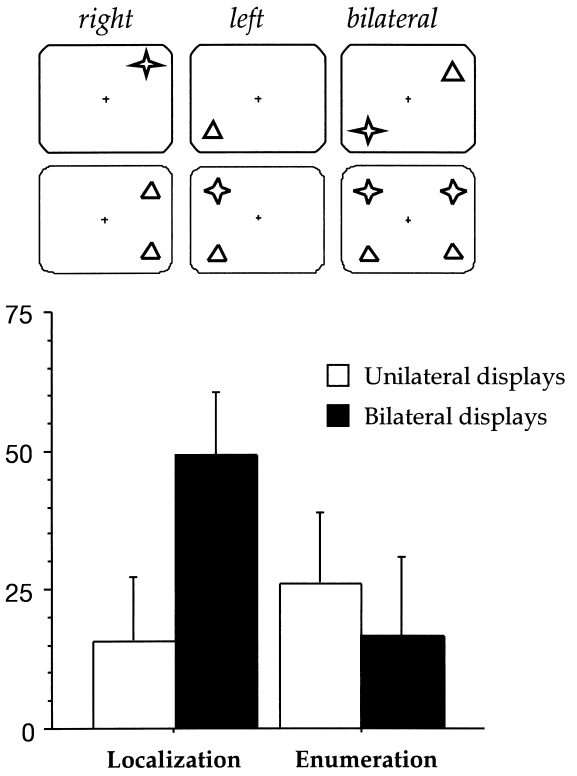


Fig. 5. Subitizing. (Top) Example of visual displays used for comparing extinction in enumeration and localization. One, two, or four shapes were presented on each trial in a random order, with an equal probability for unilateral and bilateral displays. (Bottom) Percentage of stimuli extinguished on the contralesional (left) side in each task (mean ± SD for four patients). Extinction was severe when the patients had to locate the stimuli but not significant when they had to count them. Adapted from Vuilleumier and Rafal (1999).

stimuli which are extinguished, losing the competition and thus escaping awareness. Many recent studies have found residual unconscious processing for extinguished stimuli in right-parietal patients (see Driver, 1996, for review). Most such studies adapted methods that were initially developed to study unattended processing in normals (see Merikle et al. in this volume); the two fields of study have become complementary.

Unconscious residual processing of extinguished visual stimuli can be revealed by reaction times in the patients. Marzi et al. (1996) had patients with left extinction press a button whenever a visual stimulus appeared, immediately followed by verbal report of what they saw. Like normals, the patients were faster to respond to bilateral trials than unilateral right trials, even though the patients were not aware of any visual difference between the two types of trials, reporting that they saw only the right stimulus. In another study where the task required target localization (on the

right, left, or both sides), responses were significantly slower on bilateral trials when a left target was extinguished than on unilateral right trials, even though the patients reported the same subjective percept (Vuilleumier & Rafal, 1999).

Other studies have found that reaction times can show unconscious influences not only from the presence of an extinguished stimulus, but also from more complex properties such as its colour, shape, or even identity and semantics. Using a flanker-interference procedure adapted from the normal literature, Audet, Bub, and Lecours (1991) found that the identity of an extinguished letter could affect the speed of response to a concurrent target letter presented centrally. Similar flanker effects have been obtained on the basis of shape and colour (Cohen, Ivry, Rafal, & Kohn, 1995; Di Pellegrino & De Renzi, 1995). In a related vein, Baylis, Rafal, and Driver (1993) observed that similarity in the colour or shape of two bilateral stimuli could modulate the rate of contralesional extinction, in keeping with 'repetition blindness' effects found in normals (Kanwisher, Driver, & Machado, 1995). As with the normal effect, this occurred only for similarity on the currently attended dimension (i.e. shape when reporting shapes, colour when reporting colours).

Further observations suggest unconscious processing of object category or semantics level in patients with extinction. An early study by Volpe, Ledoux, and Gazzaniga (1979) used pairs of pictured objects. Parietal patients who apparently extinguished the contralesional picture were nevertheless able to make above-chance guesses as to whether objects in the pair were the same or different. This study has since been criticized and extended (Berti et al., 1992; Farah, Monheit, Brunn, & Wallace, 1991). Berti and Rizzolatti (1992) found category effects from extinguished stimuli in a classification task (fruit/animal), with faster categorizations for ipsilesional objects when an object from the same category was presented on the contralesional side. McGlinchey-Berroth, Milberg, Verfaellie, Alexander, and Kilduff (1993) reported semantic priming in a lexical decision task. An extinguished picture on the contralesional side speeded response to subsequent semantically-related words at central fixation (see also Ladavas, Paladini, & Cubelli, 1993). Further studies have reported Stroop effects, similar to those seen in normals (Logan, 1980; MacLeod, 1991), from neglected incongruent colour-words in tasks requiring an ink-colour to be named (Berti, Frassinetti, & Umilta, 1994; Sharon, Henik, & Nachum, 1999).

One can quibble with the measure of awareness in some of these studies (e.g. see Driver, 1996), but when taken together, they provide impressive evidence for unconscious processing of remarkably complex properties for extinguished and neglected stimuli, even up to a level where the common semantics of words and pictured objects can apparently have some influence. Moreover, this conclusion accords naturally with similar conclusions for unattended processing in normals for some situations (see Driver, 1996; see also Merikle et al. in this volume).

It should be emphasized that the evidence for considerable unconscious processing in the patients by no means implies that such processing is as full as for consciously perceived events, nor that it must be equivalent in all respects to unattended processing in normals. Fuentes and Humphreys (1996) and Kim and Ivry (1998) found that flanking stimuli which produce *negative* priming effects on reac-

tion times to subsequent central targets in normals (Tipper & Driver, 1988) actually produce *positive* priming effects in the patients, perhaps because activated representations for extinguished stimuli in the patients do not need to be suppressed in order to be ignored. A further caveat is that the extent of residual processing for extinguished or neglected stimuli can vary from one patient to another, depending on the exact extent of their lesion, as we elaborate below.

The examples of residual unconscious processing so far all concern the visual modality. Evidence is starting to emerge that similar effects may exist for extinguished tactile stimuli (Aglioti, Smania, Moro, & Peru, 1998; Berti et al., 1999; Maravita, 1997), and we predict that they may be found in audition also.

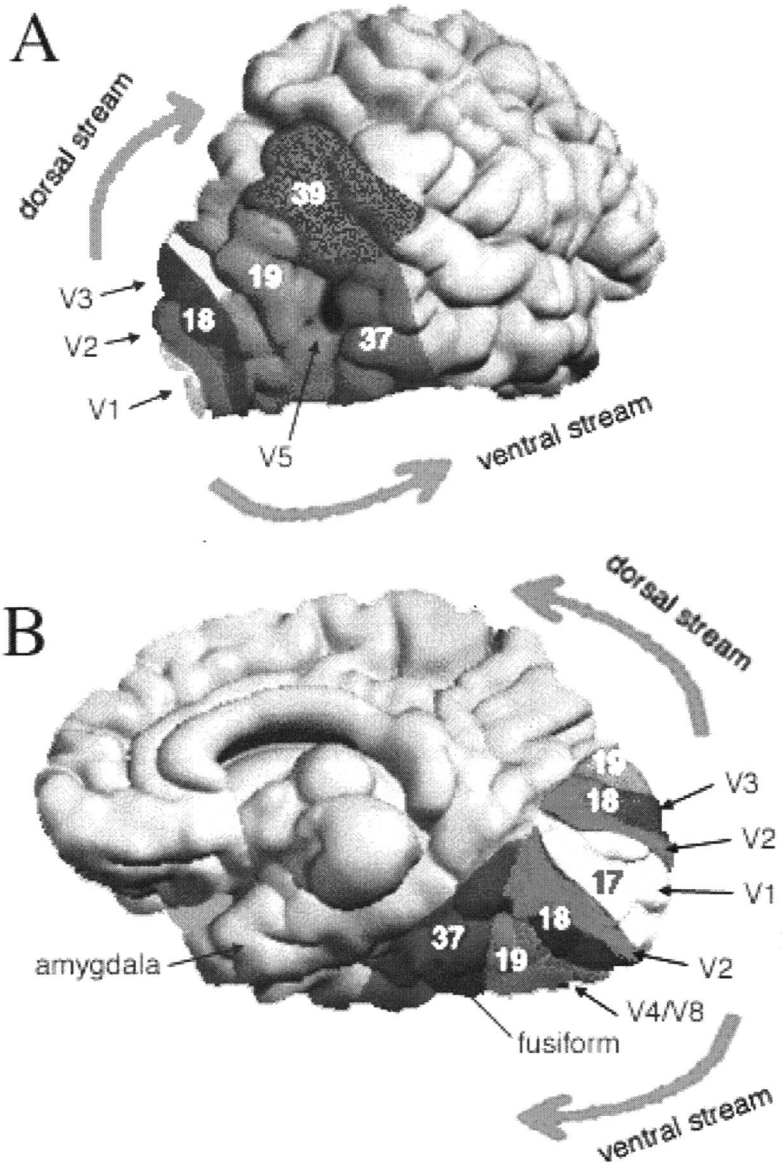
10. Anatomy of conscious and unconscious perception in relation to neglect and extinction

The accumulating evidence for considerable unconscious processing in neglect patients has now reached the point where it raises specific questions about the underlying anatomical substrates. Here again our discussion will concentrate mostly on vision, as this is best understood, though similar principles may apply to other modalities. The visual system of primates includes many distinct cortical areas, organized hierarchically in both parallel and serial pathways (Felleman & Van Essen, 1991). Long established in animal studies, such areas can now be mapped non-invasively for humans via functional imaging (see Fig. 6). One major pathway from the retina projects via subcortical relays in the thalamus into the primary visual cortex (V1), destruction of which is associated with field cuts and blindsight, and then from V1 into several extrastriate areas within the occipital lobe (Fig. 6). Neurophysiology indicates that striate and early extrastriate areas subserve basic visual operations, such as analysis by spatial frequency and orientation, extraction of contours, figure-ground segmentation, plus some forms of grouping (e.g. Peterhans & Von Der Heydt, 1991). Extraction of colour and of motion parameters has also been associated with particular extrastriate areas (Tootell, Hadjikhani, Mendola, Marrett, & Dale, 1998; Zeki, 1993).

Subsequent stages of visual processing are broadly considered in terms of two major parallel pathways: a *ventral* stream of successive areas along occipito-temporal cortex encoding object attributes such as shape, colour or identity for the purpose of recognition by contact with long-term memories in the temporal lobe; and a *dorsal* stream into the parietal cortex encoding spatial parameters (Ungeleider & Mishkin, 1982) for the on-line control of spatial action (Milner & Goodale, 1995). A role of *dorsal* pathways in unconscious residual vision (plus subcortical thalamo-collicular circuits and their cortical projections bypassing primary visual cortex) has been emphasized by past studies of patients with blindsight, given their ability to point or saccade towards unseen stimuli presented within a field cut (e.g. Cowey & Stoerig, 1991; Rossetti, 1998; Weiskrantz, 1986). Dorsal pathways have also been implicated in the preserved visuomotor function in a patient with a severe loss of conscious form vision after extensive ventral damage (Milner et al., 1991).

This has led to a common view that the dorsal stream may operate unconsciously, with only the ventral stream producing conscious visual awareness.

However, a compelling hypothesis to explain the behavioural results we have described from patients with neglect and extinction is that considerable unconscious processing may take place not only within early visual areas of the occipital lobe, but also along the ventral pathway into the temporal lobe. Neglect patients typically suffer from parietal lesions which can leave posterior occipital and inferior temporal



cortices relatively intact (e.g. Fig. 2C). These brain areas may still receive sufficient input from contralesional stimuli to support the preserved grouping processes and unconscious activation of representations for identity and semantics, which we have described.

This anatomical perspective predicts that the extent of unconscious residual processing in neglect should depend on how far the parietal lesion extends towards the occipital or temporal cortex in a given patient. Consistent with this, a recent study of 12 neglect patients found that effects of grouping into Kanizsa subjective figures occurred only in some cases (Vuilleumier, Valenza, & Landis, submitted for publication), depending closely on the lesion. Patients with a preserved influence of subjective figures had focal brain damage centred in parietal or subcortical regions, sparing the occipital lobe (Fig. 7A, top row). The subjective-figure influence was absent in patients whose damage extended posteriorly into lateral occipital cortex (Fig. 7B, bottom row). This accords with the fact that areas in monkey lateral occipital cortex (e.g. V2) contain neurons responding to Kanizsa subjective contours (Von Der Heydt et al., 1984). Moreover, similar occipital extrastriate regions can be activated by illusory Kanizsa figures during functional neuroimaging in normal humans (Ffytche & Zeki, 1996).

Other behavioural effects in neglect patients, such as those implying extraction of identity, category or semantics for extinguished visual objects (e.g. Audet et al., 1991; McGlinchey-Berroth et al., 1993), presumably implicate ventral temporal areas (Driver, 1996). Such brain areas have long been associated with visual shape recognition and object identification (Grüsser & Landis, 1991; Milner & Goodale, 1995), as lesions there produce various deficits in visual recognition, termed 'agnosia'. This association has been supported by functional imaging evidence for activation in ventral brain areas by seen objects, words or faces,

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Fig. 6. Pathways of cortical visual processing. (A) Three-quarter view of the posterior lateral side of the human brain. (B) View of the medial side of the human brain. Distinct areas of the occipital lobe are indicated with shades of grey. In most cases, each shaded area corresponds to a Brodmann area (numbered in white), as traditionally defined by cytoarchitecture. In a few cases (specifically for those marked V2, V3 and V5) the shaded areas shown were defined by mapping of functionally distinct areas with fMRI in humans (see Tootell et al., 1998). The geniculate pathway from the retina projects to primary striate visual cortex (V1, Brodmann's area 17), and then from V1 to several extrastriate areas in the occipital lobe (e.g. V2, V3, and V3A, Brodmann's area 18). These early occipital areas appear concerned with the analysis of spatial frequency and orientation, and some forms of grouping and surface-extraction. Later extrastriate areas may be associated with more specific visual properties (e.g. colour in V4 and V8, or motion in V5). Two streams of forward projection from the occipital lobe can be broadly distinguished: a 'dorsal' stream into the parietal lobe, and a 'ventral' stream into the temporal lobe (see thick arrows). Areas in ventral temporal cortex subserve discrimination of object shape, identity and semantics, in relation to long-term memory. For instance, the fusiform gyrus (area 37) may be specialized in the recognition of faces, while distinct adjacent areas are more important for objects or letter strings. The amygdala is implicated in the recognition and learning of emotional cues. By contrast, areas in dorsal parietal pathways encode spatial location of stimuli and parameters for directing action towards them, including hand, or eye movements. Lesions associated with neglect involve the right inferior parietal cortex (Brodmann's areas 39–40, see speckled area in A) and may therefore leave many occipito-temporal areas intact (at least in some patients; see Fig. 7 for comparison). Adapted from Tootell et al. (1998).

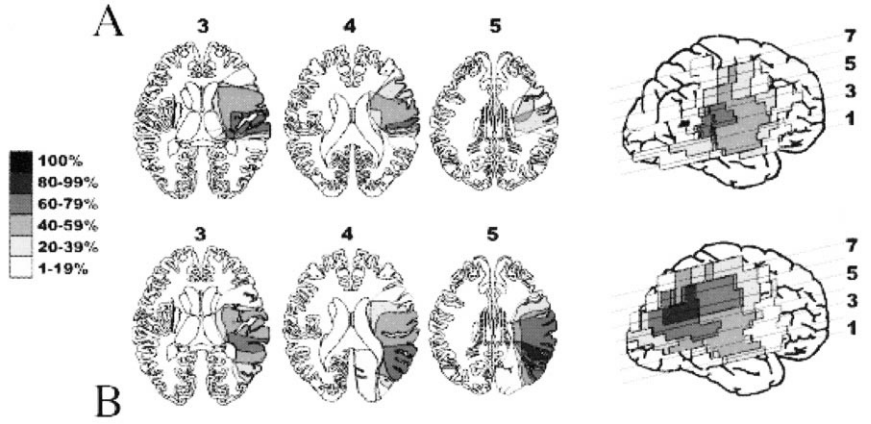


Fig. 7. Average overlap of brain lesions in patients with unilateral left neglect who either showed preserved grouping effects with illusory Kanizsa figures (A: Group + , $n = 6$) or did not show preserved grouping effects (B: Group − , $n = 6$). Lesions were reconstructed from CT or MRI scans in each patient. Three axial slices and the lateral aspect of the right hemisphere are shown. The grey shade scale indicates the percentage of overlapping lesions. Grouping into illusory figures was revealed in a bisection task where all patients in Group + produced midpoint judgements similar for Kanizsa stimuli and figures with real contours, but different from other discontinuous displays. The two groups of patients did not differ in the severity of neglect in standard line bisection and cancellation tasks. However, there was a clear difference in the extent of cortical damage. Patients in Group + had damage overlapping in the inferior posterior parietal lobe (Brodmann's area 40) or in the thalamus, with posterior lateral occipital areas always spared. By contrast, patients in Group − had larger lesions extending from the inferior parietal cortex to the lateral occipital lobe (Brodmann's areas 18–19). The latter might include human areas homologous to monkey V2, where neurons have been found to code similarly for real contrast-defined contours and illusory contours of the Kanizsa type (compare with Fig. 6). Adapted from Vuilleumier et al. (2000).

with different areas activated most strongly by each stimulus class (Farah & Aguirre, 1999). For instance, some areas along the fusiform gyrus appear to respond particularly strongly to seen faces (Kanwisher, McDermott, & Chun, 1996).

A recent study (Vuilleumier, 2000) found suggestive behavioural support for preserved activation of such structures by contralesional stimuli in extinction patients, observing that schematic faces show less extinction than other types of stimuli (e.g. scrambled faces, letter strings, or ring shapes, all of which should produces less activation of the 'fusiform face area' than intact faces). With a constant stimulus on the ipsilesional side during bilateral trials, less extinction was suffered by a contralesional intact face than by the other classes of stimuli (Fig. 8A,B), although faces could still be extinguished to some extent. Conversely, a face on the right side produced more extinction of a constant left stimulus than other types of right stimuli. Both aspects of the results suggest a competitive advantage for face stimuli, which may relate both to their biological significance and to the existence of many specialized neurons responding to them in the fusiform cortex.

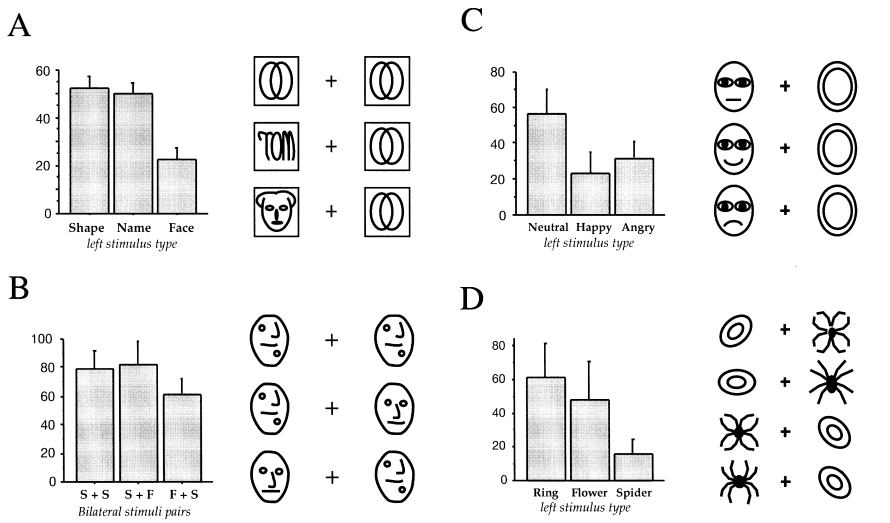


Fig. 8. Stimulus-dependent effects on the severity of extinction. For each experiment, stimulus examples are shown in the right-hand panel and the percentage of contralesional (left) stimuli extinguished on bilateral trials is shown in the left-hand panel (mean ± SD in three patients). Unilateral stimuli (data not shown) were most often correctly detected and identified on either side. (A) In one experiment, a schematic face, a familiar name, or a symmetrical shape could be briefly presented on either side. On bilateral trials where there was a constant competing shape ipsilesionally (e.g. circles, as shown), a face in the contralesional hemifield was much less extinguished than other types of visual stimuli. Adapted from Vuilleumier (2000). (B) In a similar experiment, either schematic faces or scrambled faces were briefly presented. On bilateral trials, the intact faces were again less extinguished, now compared to the scrambled ones. (C) In a further experiment, a schematic face or a shape was presented in either hemifield, with the face having one of three possible emotional expressions: neutral, happy, or angry. On bilateral trials, when there was always the same competing shape on the ipsilesional side, contralesional faces with a happy or angry expression were less extinguished than neutral faces. There was no significant difference between happy and angry faces. Adapted from Vuilleumier and Schwartz (in press). (D) In this experiment, the stimuli included schematic spiders, flowers, or meaningless ring shapes that were presented bilaterally or unilaterally on either side. On bilateral trials, contralesional spiders were extinguished much less often than flowers or rings, while both flowers and meaningless rings were extinguished to the same degree. Data for left-sided rings are collapsed across two possible bilateral pairs with either flowers or spiders on the right, since extinction in these two conditions did not differ. Adapted from Vuilleumier and Schwartz (2000).

These extinction results also accord with claims that some perceptual organization of facial features may occur ‘preattentively’ in normal vision (Suzuki & Cavanagh, 1995).

The modulation of extinction by face stimuli provides yet another example of preserved processing (presumably activation of category-specific ventral regions, such as the fusiform) affecting what reaches awareness, similar to Merikle et al.’s (this volume) description of how preattentive processes modulate what reaches awareness in normals. It makes adaptive sense that the limited contents of awareness should be determined to some extent by the biological significance of the competing

inputs. Further extinction findings suggest that emotional significance can exert a similar influence. Vuilleumier and Schwartz (in press) found not only less extinction for contralesional faces than other shapes, but also for schematic faces with a happy or angry facial expression versus those with a neutral expression (Fig. 8C). This may relate to similar observations on the capture of normal attention by emotional faces (Mack & Rock, 1998). Such effects may also have a neural basis in the activation of anterior temporal structures, including the amygdala.

Amygdala activation has been reported when viewing emotional facial expressions in both humans (Breiter et al., 1996; Morris, Öhman, & Dolan, 1998b) and monkeys (Brothers, Ring, & Kling, 1990). This may feedback to enhance fusiform responses to face stimuli (Morris et al., 1998a; Sugase, Yamane, Ueno, & Kawano, 1999). Such amygdala activations have been reported in normals even for subliminal stimuli (Whalen et al., 1998), in particular when previously associated with fear (e.g. Morris, et al., 1998b). As for emotional faces, parietal patients also show less extinction for contralesional pictures of spiders, known to elicit consistent fear responses even in non-phobic individuals (e.g. Öhman, 1986), than for pictures of flowers made from similar visual features (Fig. 8D) (Vuilleumier & Schwartz, 2000). Emotionally salient contralesional stimuli may thus tend to capture attention and awareness. Here again, the results seem to imply considerable processing of contralesional stimuli (here, concerning their biological or affective significance) despite the patient's lesion and associated spatial bias.

11. The neural fate of extinguished stimuli: neurophysiological measures

In the previous sections, we speculatively related the modulation of extinction by factors such as grouping or emotional salience, and the preserved unconscious processing for extinguished stimuli, to activation of known neural pathways. More direct evidence for such activation would be provided by measuring neural activity directly in the patients, with functional imaging or event-related potentials. Such methods have been widely used to study normal attention (e.g. Corbetta, Meizin, Dobmeyer, Shulman, & Petersen, 1990; Mangun, 1995), and have been applied to neurological deficits of awareness in occipital blindsight patients (Sahraie et al., 1997; Shefrin, Goodin, & Aminoff, 1988). But apart from a few pioneering studies (Spinelli, Burr, & Morrone, 1994; Vallar, Sandroni, Rusconi, & Barbieri, 1991; Viggiano, Spinelli, & Mecacci, 1995), they have only been applied to neglect and extinction very recently.

Rees et al. (2000) used event-related fMRI to study a patient (GK) with left extinction and neglect after a focal inferior parietal lesion (shown in Fig. 3C). Pictures of faces and houses were briefly presented in either the right, left, or both hemifields, with stimulus parameters chosen such that even faces were extinguished. Comparing bilateral trials on which the left stimulus was extinguished with unilateral right trials (for which the patient's conscious report was identical) revealed that extinguished contralesional stimuli significantly activated primary visual cortex and early extrastriate visual areas in the damaged right hemisphere (Fig. 9) in a similar

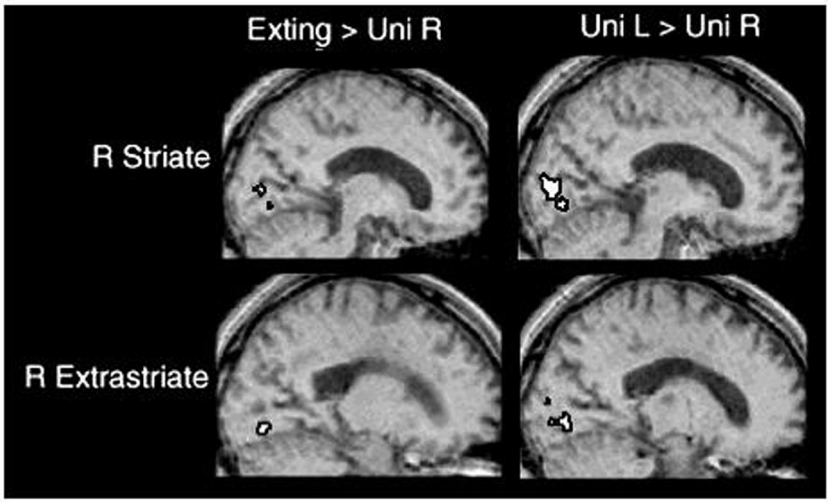
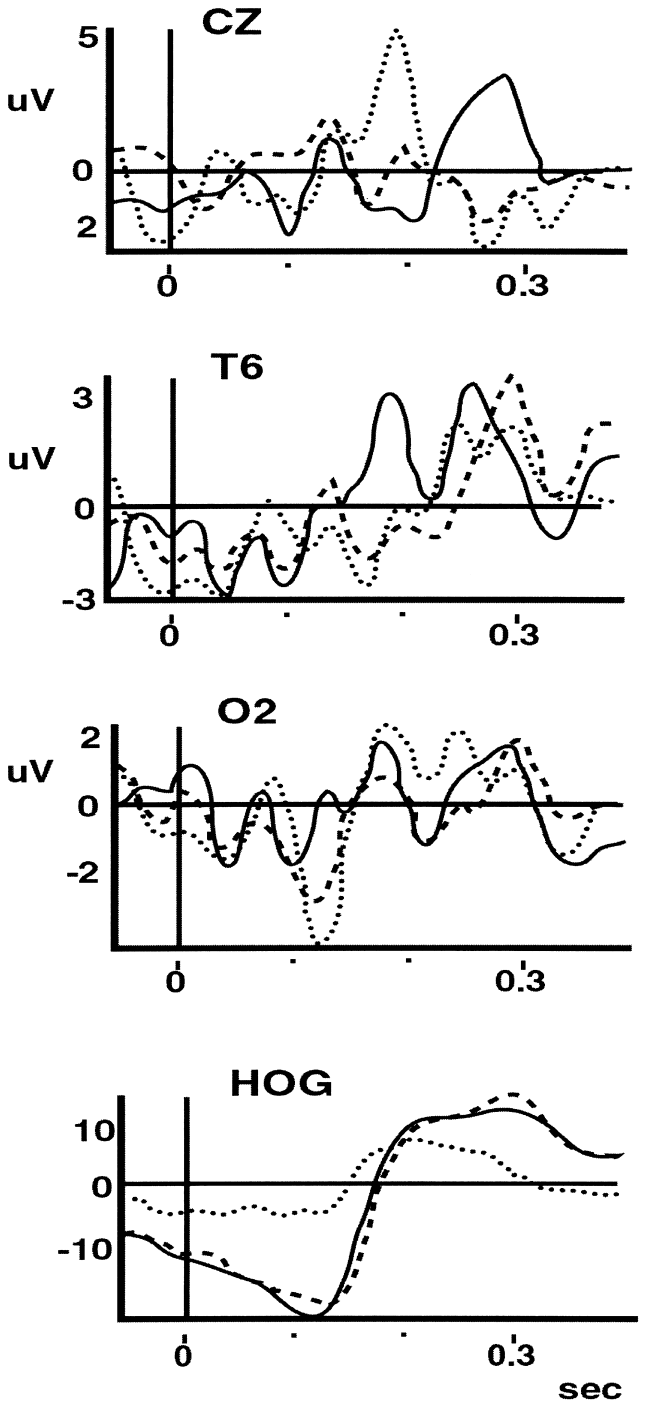


Fig. 9. Functional magnetic resonance imaging of seen and extinguished stimuli in the contralesional visual hemifield of a patient with right-parietal damage. Compared to the activation produced by unilateral right events, bilateral events in which the patient extinguished the left-side stimulus (left column) produced more activation in both striate and extrastriate visual areas of the damaged right hemisphere (Exting > UniR), although the patient's conscious report was identical in these two conditions. The location of such activation by unseen contralesional events in extinguished trials (left column) was similar to that associated with seen contralesional events in unilateral trials (UniL > UniR). Adapted from Rees et al. (2000).

manner to that found for consciously seen unilateral left stimuli. This confirms for the first time our prediction that early visual areas in the occipital lobe, including those lesioned in blindsight patients, can be activated in an unconscious manner in parietal patients.

Furthermore, comparing extinguished faces to extinguished houses (Rees et al., 2000) revealed weak but reliable activation in a right-hemisphere fusiform region, whose anatomical co-ordinates corresponded to face-selective regions in normal subjects (Kanwisher et al., 1996), and to the response to faces versus houses when consciously seen (and fixated) in the patient. A similar event-related fMRI study was independently carried out by Vuilleumier et al. (2000) on another right-parietal patient (CW), who showed extinction for only a proportion of bilateral trials. Their results agree with Rees et al. (2000), showing some preserved occipito-temporal response for extinguished stimuli. They further suggest that those contralesional stimuli which are consciously perceived on bilateral trials may produce stronger activity within occipital and ventral regions, and activate additional structures also, including parietal and frontal areas in the intact (left) hemisphere. Moreover, analysis of trial-specific 'coupling' between areas showed increased coupling between right striate cortex and left parietal and frontal areas on those bilateral trials where the left stimulus was detected.


CZ
5
uV
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-2
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HOG
10
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-10
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sec

Event-related potential (ERP) scalp recordings have also recently been employed in the same two parietal patients (Eimer, pers. commun.; Sagiv, Vuilleumier, & Swick, 2000). The same stimuli were used as in the fMRI experiments to exploit the fact that face stimuli can evoke specific ERP components around 170–200 ms after stimulus onset (Bentin, Allison, Puce, Perez, & McCarthy, 1996; Eimer, 1998), in addition to earlier activity that is not category-specific (around 100 ms). In patient CW (Sagiv et al., 2000), stimulus duration was titrated to produce extinction on approximately 50% of bilateral trials. In comparison with perceived contralesional faces, extinguished faces evoked similar early visual responses at electrodes over the occipital regions, though slightly decreased in amplitude, plus a similar face-specific component (the 'N170') around 170 ms after stimulus onset at posterior temporal electrodes (see Fig. 10). This suggests that some category-specific face activity was triggered even for an extinguished face, in agreement with the fMRI data.

There were some differences in the ERPs for extinguished versus perceived contralesional stimuli in patient CW. In particular, only perceived faces evoked a positive potential over midline and anterior sites at a later latency (around 200 ms). Another recent ERP study of extinction (Marzi, Girelli, Miniussi, Smania, & Maravita, in press) found considerable differences between the ERPs for extinguished versus perceived contralesional stimuli, even for quite early occipito-parietal components (P100-N100). The stimuli used in this study were small peripheral LEDs, which seem unlikely to drive the ventral activity observed in the recent fMRI studies of extinction (Rees et al., 2000; Vuilleumier et al., 2000). Finally, Hämäläinen, Pirilä, Lahtinen, Lindroos, and Salmelin (1999) reported reduced or absent early potentials (N100) at occipital sites in a series of 20 patients with neglect who failed to detect unilateral stimuli, although distinct ERP responses to occasional deviant stimuli (mismatch negativity components) revealed that some residual processing still took place for the neglected events (but see also Deouell, Bentin, & Soroker, 2000).

Fig. 10. Evoked potentials in a patient with right focal parietal damage and left visual extinction. Stimuli consisted of schematic faces or symmetrical shapes that could be briefly presented in the left, right, or both hemifields. The critical bilateral trials were those with a face on the left side and a shape on the right side. Stimuli duration was titrated so that in roughly half of bilateral trials, left faces were extinguished, while in the other half they were perceived. Average potentials are shown for occipital (O2) and posterior temporal (T6) electrodes, as well as a midline electrode (CZ) over a period of 300 ms after stimulus onset. Horizontal eye saccades are also shown below (HOG). In bilateral trials, left-side faces that were perceived (dotted lines) evoked early visual components around 100 ms at the occipital electrode (O2) and face-specific negative components around 170 ms (N170) at the temporal electrode (T6) in the contralateral damaged hemisphere. Both occipital visual and temporal facial components were still present when left-side faces were extinguished (dashed line) but not when there was only a right-side shape (continuous line), although the patient's conscious perception and eye movements were similar in these two latter conditions. Another face-specific component was observed around 200 ms at the midline electrode only when left-side faces were consciously perceived in bilateral trials (dotted line), and in unilateral trials with left-side faces as well (data not shown). Although oculographic differences (see HOG) may arise for bilateral trials with perceived left faces, note the similarity in eye movements for extinguished faces in bilateral displays versus unilateral right trials. Adapted from Sagiv et al. (2000).

It is heartening when fMRI data and ERP data converge, as for the residual processing for extinguished stimuli revealed in the studies of Rees et al. (2000), Sagiv et al. (2000) and Vuilleumier et al. (2000). However, these two methods of indexing neural activity measure very different things, and so need not always agree. In particular, ERP measures of neural activity, via voltage fluctuations at the scalp, depend heavily on the synchronous firing of many neurons at a fine temporal scale. By contrast, fMRI is much less dependent on the fine time-scale of neural events. One consequence is that a neural activation (e.g. of striate cortex, in response to a contralateral extinguished stimulus) which looks relatively normal with fMRI could in principle look quite abnormal with ERP if the synchrony of firing was somehow disrupted by the parietal lesion. Preliminary ERP data in patient GK (Eimer, pers. commun.) suggest just this.

Given recent suggestions that conscious perception may depend in a large part on the exact timing of neural events (Engel, Konig, Kreiter, & Singer, 1991; von der Malsburg, 1994), it should be interesting to compare fMRI and ERP measures further in future studies of extinction. It should also be informative to look with these methods at extinction in non-visual modalities (Beversdorf et al., 1999; Remy et al., 1999), and at cross-modal extinction (Di Pellegrino, Ladavas, & Farne, 1997; Ladavas, Di Pellegrino, Farne, & Zeloni, 1998; Mattingley, Driver, Beschin, & Robertson, 1997). For the moment, the recent evidence provides initial, tentative steps in delineating the neural correlates for conscious and unconscious perception in extinction patients. Overall, the results converge with the prediction derived from behavioural experiments that extinguished visual events may still activate visual areas of occipital and ventral temporal cortex, without awareness. This may differ from blindsight after occipital damage (Cowey & Stoerig, 1991; Weiskrantz, 1990), where unconscious residual vision has typically been attributed to pathways which bypass occipital cortex, projecting subcortically and into the dorsal stream. Moreover, as mentioned earlier, while the challenge in the study of blindsight has always been to explain the unconscious residual processing with primary visual cortex destroyed, a major challenge in the study of neglect and extinction is to explain the loss of awareness itself, given that many visual afferents are spared. As we have seen, projections into primary visual cortex, and thence to the ventral stream, can still be intact in neglect patients, and the recent fMRI studies show that these areas can still be activated for extinguished stimuli. How then can the patient remain unaware of a contralesional stimulus, even when it can still activate the pathways that are most often considered to support conscious experience?

It is tempting to suppose that some interaction between the (damaged) parietal areas and occipito-temporal areas is involved, with the occipito-temporal activations being insufficient to generate awareness in the absence of appropriate parietal activation or feedback. It remains to be determined exactly how comparable the residual occipito-temporal processing is for extinguished stimuli versus consciously seen stimuli, and exactly how far along the ventral pathway extinguished stimuli can proceed (e.g. can they influence the formation of new learning in temporal or amygdala structures?). In recent unpublished work, we have found that extinguished

stimuli can produce implicit learning effects lasting tens of minutes, but no explicit memory.[2]

In the normal visual system, it is already known that spatial attention can modulate the activity of ventral temporal cortical areas, as well as early occipital areas right back to primary visual cortex (e.g. Corbetta et al., 1990; Martinez et al., 1999). Conceivably, such modulation in normals might reflect some interaction of these visual areas with parietal and/or frontal areas that is critical for awareness, and which goes pathologically awry following the unilateral lesions which produce neglect and extinction. The dramatic effect of parietal damage on awareness in neglect patients certainly suggests that interaction of this kind must exist between parietal areas and other parts of the visual system. New methods of analysis are currently being developed in functional imaging (e.g. Friston, 1994; Lumer & Rees, 1999) and event-related potential research (Rodriguez et al., 1999), which should allow researchers to investigate how activity in different brain areas dynamically interacts. For instance, in this way one might determine whether occipital visual areas or the fusiform correlate differentially with other brain areas (e.g. in the parietal or frontal lobe) when a particular stimulus is consciously experienced versus extinguished in the patients (see Lumer & Rees, 1999, for a related finding in normals). Vuilleumier et al. (2000) provide initial evidence for this in a right-parietal extinction patient.

The recent findings that ventral occipito-temporal structures can be activated unconsciously in neglect patients do not fit the simplistic dichotomy sometimes implied in the literature on the neuropsychology of visual awareness between conscious ventral processing versus unconscious dorsal processing. However, Milner and Goodale (1995) had already argued that the *inferior* parietal areas which are damaged in most neglect patients may be hard to pigeonhole as exclusively dorsal or ventral (Milner, 1997; Milner & Goodale, 1995). Patients with *superior* parietal lesions typically show the classic 'dorsal' visuomotor impairments in reaching and grasping, but no neglect (Perenin, 1997). Furthermore, one inferior parietal patient with neglect has been reported to show normal scaling of hand grip when reaching for objects in the contralesional space, despite gross perceptual underestimation of size for the same objects, implying some preserved function in dorsal visuomotor pathways in the superior parietal lobe, even in the presence of inferior parietal neglect (Pritchard, Milner, & Harvey, 1997).

Driver (1996) and Heilman, Watson, and Valenstein (1997) have speculated that the inferior parietal lobe areas impaired in most neglect patients may serve as an interface between dorsal and ventral streams, allowing dorsal spatial properties to be linked with ventral non-spatial properties, such as object identity. Watson et al. (1994) made a related suggestion based on monkey studies, but emphasizing areas in the superior temporal sulcus, which can also be affected in many neglect patients. It may be that we cannot become consciously aware of an identified object

[2] A recent study (Bisiach, Ricci, Silani, Cossa, & Crespi, 1999) made the striking claim that neglected visual objects may resurface at a later time in explicit memory (see also Block in this volume for philosophical discussion of such a possibility). However, the existing evidence for this can be criticized on methodological and statistical grounds.

when there is no awareness of its location, as following the lesion in neglect patients. A failure to develop appropriate representations for contralesional locations may also preclude intentional actions towards the affected region of space, which in turn may also have implications for awareness. We return to discuss whether spatial location and the parietal lobe may play a special role in awareness after a brief consideration of some of the potentially relevant properties of single neurons in monkey parietal cortex.

12. Cellular properties of the parietal lobe in relation to neglect and extinction

Single-cell recording in awake behaving monkeys has provided dramatic insights into the computational properties of neurons in the parietal lobe and related structures (e.g. see Andersen, Snyder, Bradley, & Xing, 1997, for review). Much of this evidence comes from recordings in posterior parietal cortex, in and around the intraparietal sulcus, though explorations continue into further parietal areas, and similar cellular properties have even been reported in a few of the other brain areas associated with neglect (e.g. premotor and prefrontal regions). We cannot provide a comprehensive review of the complex neurophysiological literature here, but will give some overview of salient features which may relate to human neglect and extinction.

As noted earlier, the spatial deficit in neglect and extinction patients is typically *graded* in nature, with performance gradually declining for stimulus locations that are further towards the contralesional side (Kinsbourne, 1993; Ladavas, 1990; Smania et al., 1998). Moreover, there is often an off-centre peak in performance for the patients. For instance, many visual tasks are performed best at the centre of the visual field in normals, yet neglect patients often do better within the right visual field than centrally (e.g. for detection RTs and accuracy; Smania et al., 1998). This is quite unlike the impairments seen after lesions to primary sensory cortex, which typically produce deficits with sharp borders, without any off-centre peak within intact parts of the field. The graded impairments that characterize neglect might correspond to a pathological gradient in the number of neurons remaining for particular regions of space within the parietal lobe after the lesion (Pouget & Driver, in press; Pouget & Sejnovski, 1997; see also Rizzolatti & Berti, 1990).

Physiological studies in monkey show that, unlike earlier visual areas, parietal regions include some neurons with ipsilateral receptive fields, so that while the representation within one hemisphere emphasizes contralateral space overall, some ipsilateral representation is present also. More specifically (see Fig. 11), the number of left-hemisphere neurons with visual receptive fields at a particular location decreases monotonically as one considers increasingly peripheral locations in the left visual field, and vice versa in the right hemisphere (Andersen, Asanuma, Essick, & Siegel, 1990; Ben-Hamed and Duhamel, pers. commun.). The number of neurons for different lateral positions within parietal areas for one monkey hemisphere thus resembles (see Fig. 11) the graded performance of neglect patients with

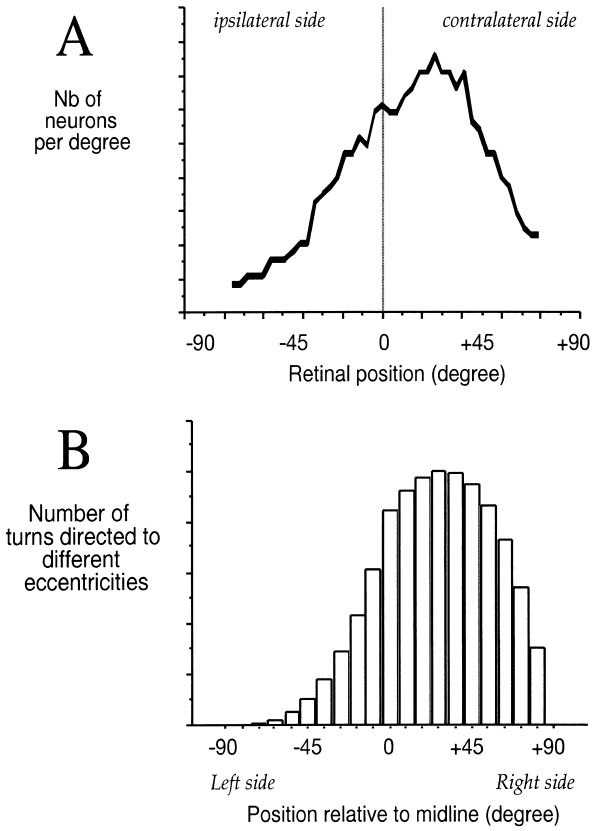

Fig. 11. Schematic illustration of how neuronal representation for a parietal area within one hemisphere may relate to the graded deficits seen in neglect patients. (A) Neurophysiological recordings in the monkey (here from area VIP in the left hemisphere; Ben-Hamed & Duhamel, pers. commun.) show a graded profile in the number of neurons which have receptive fields at particular locations on the retina, with an off-centre peak in the contralateral field (i.e. right field for the left hemisphere), and then a gradual decline for more eccentric locations in the ipsilateral field (left). This is very different from the distribution of receptive fields in the early visual areas of occipital cortex, where neurons have strictly contralateral retinotopic responses and maximally represent the central region near the fovea. Adapted from Andersen et al. (1990) and Ben-Hamed and Duhamel (pers. commun.). (B) After lesions affecting parietal neurons in the right hemisphere, patients with left neglect typically exhibit a spatially graded deficit in their performance, which may relate to the number of remaining neurons (mainly within the intact hemisphere) representing particular points in space. For instance, the number of spontaneous eye and head movements to particular locations in space is pathologically biased towards the ipsilesional side with an off-centre peak, as shown schematically here. Adapted from Karnath et al. (1998). The resemblance to the neural gradient shown above, which might correspond to that within the intact hemisphere, is striking, although it remains to be specified exactly how cellular activity would be read out into overt behaviour.

only one hemisphere intact (Behrmann, Ghiselli-Crippa, Sweeney, Dimatteo, & Kass, 2000; Karnath, Niemeier, & Dichgans, 1998; Ladavas, 1990; Smania et al., 1998). Both show a gradual decline against lateral position, rather than a step-

function centred on anatomical midlines, together with an off-centre peak (Fig. 11A,B). Of course, without a computational model that relates single neurons to performance (see Pouget & Sejnovski, 1997), this is just an analogy, plus there may be some differences in the exact neural distribution between humans and monkeys. For instance, while the gradients within the two hemispheres are mirror images of each other in monkeys, these may be asymmetric in humans (Anderson, 1996; Heilman & Van Den Abell, 1980), such that the gradient in the human right hemisphere is shallower (perhaps because this hemisphere has become specialized for representing both sides of space, as the converse of the left hemisphere becoming specialized for language). This might go some way towards explaining why neglect is more severe after right-hemisphere lesions in people, leaving the patient with just the steep gradient of the intact left hemisphere.

The properties of parietal neurons may relate to other aspects of neglect also, beyond its graded nature. As noted earlier, further spatial properties of neglect also distinguish it from primary sensory losses. In particular, whether or not a particular stimulus reaches awareness can depend on current posture, rather than just location on the receptor surface. Thus, a previously neglected left visual field stimulus may become detectable with eyes and/or head deviated to the right (Kooistra & Heilman, 1989; Vuilleumier et al., 1999), or with the trunk deviated towards the left (Karnath et al., 1991) even though the retinal input remains unchanged. The response of neurons in the monkey parietal lobe is also influenced by posture. For instance, although most neurons in parietal areas LIP and 7a have retinotopic receptive fields, the amplitude (or 'gain') of their retinal response is modulated by postural factors such as eye position (Andersen, Essick, & Siegel, 1985). Hence such cells effectively responds to a specific combination of sensory input and posture, thus potentially representing stimulus location in more complex spatial co-ordinates than purely retinal (e.g. the head-centred position of a stimulus could in principle be extracted from such cells). Such '*gain modulation*' by non-retinal factors (see Fig. 12) can explain why neglect typically arises in a mixture of egocentric co-ordinates after losses of such cells, with effects of posture in addition to retinal location (Pouget & Sejnovski, 1997). Recent physiological evidence shows that the gain-modulation principle extends to many different types of information (e.g. proprioceptive, vestibular) that are each combined with retinal information in particular areas of parietal cortex (e.g. Snyder, Grieve, Brotchie, & Andersen, 1998). These types of information are also known to influence neglect (e.g. Vallar, 1998). Indeed, neurons that integrate retinal information with vestibular information may conceivably be responsible for the dramatic effects that vestibular interventions (e.g. caloric stimulation via iced water in the left ear) can have in ameliorating the deficit of awareness in neglect (e.g. Rubens, 1985).

As discussed extensively in our sections on extinction, the pathology in the patients typically becomes most evident when several stimuli are presented simultaneously, and this again may relate to recent findings on monkey parietal neurons. Interestingly, extinction in the patients can be determined less by the absolute location of a stimulus within the left or right hemifield than by its position *relative*

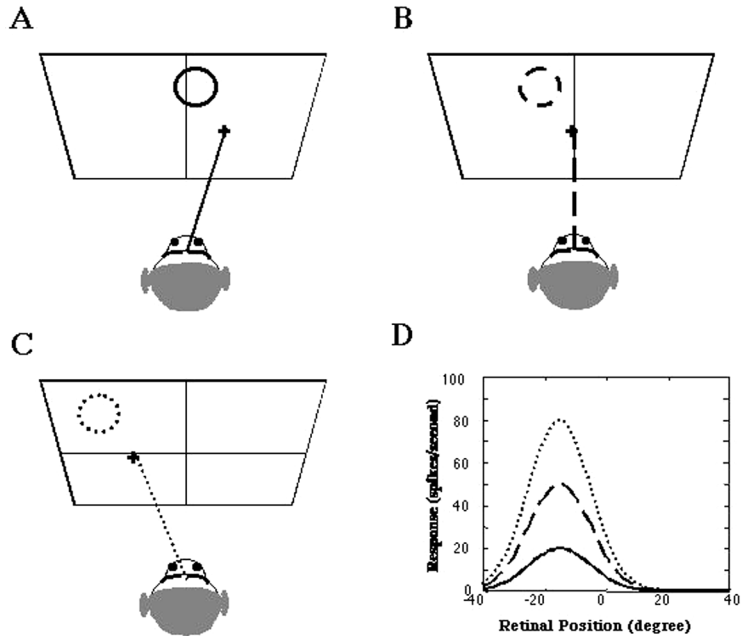

Fig. 12. Schematic showing modulation of visual response by gaze direction in parietal neurons. When a visual stimulus is presented near the receptive field (see circles depicted in A–C) of a given neuron in cortical area LIP of the monkey, the neuron's response (depicted for an idealized neuron in D) depends not only on retinal position of the stimulus, but also on the current eye-in-orbit position (see fixation lines in A–C). While the receptive field remains fixed on the retina (so that its Gaussian profile moves with the eyes), the amplitude of the response increases with gaze directed to the left (C, dotted lines in D) and decreases with gaze to the right (A, continuous lines in D), as compared to the response with eyes central (B, dashed line in D). The gain of visual responses is thus modulated by extraretinal information about posture (see D). In principle, such gain modulation could be used to transform retinal location into visual location with respect to the head or other frameworks. Adapted from Andersen et al. (1985).

to other competing stimuli (Kinsbourne, 1993; Pouget & Driver, in press). Thus, visual extinction can be observed for the leftmost of two stimuli even when both are presented within the right visual field, not just when one is in the left visual field. Whichever stimulus is further to the ipsilesional side will typically capture attention, as if the suggested gradient in the strength of remaining representations in parietal cortex (see Fig. 11A) has some winner-takes-all outcome.

This may relate to the fact that, even in the normal brain, parietal neurons provide a highly selective representation of current sensory input, as compared with earlier visual areas. For instance, neurons in parietal area LIP of the monkey effectively respond only to the most salient (or attended) stimulus in the current visual array (Gottlieb et al., 1998), or to that which the animal currently intends to respond to (Snyder, Batista, & Andersen, 1997). Although many brain areas are now known to show some modulation of visual responses by attention (e.g. Desimone & Duncan, 1995; Moran & Desimone, 1985), these effects are particularly

marked in parietal areas (and in other brain regions associated with neglect, such as premotor and prefrontal regions), where a winner-takes-all principle may operate spatially across the *entire* visual field, so that only a single stimulus becomes dominant. This may relate to the attentional bias associated with neglect, since a lesion in such a population of neurons would bias the suggested winner-takes-all process; in competitive situations the winner will inevitably be a stimulus towards the ipsilesional side of space. Thus, one might think of the gradient depicted in Fig. 11A not only as the number of neurons for particular lateral positions in space, but perhaps also as corresponding to the competitive strength, or 'attentional weight' (Desimone & Duncan, 1995; Moran & Desimone, 1985) of stimuli at these different locations.

A further aspect of neurons in the parietal lobe and related areas is that many have been shown to respond to stimuli in several modalities. For instance, visuo-auditory cells have been reported in parietal areas LIP (Linden, Grunewald, & Andersen, 1999) and MIP (Cohen & Andersen, 1998), and visuo-tactile cells have been found in VIP (Duhamel, Colby, & Goldberg, 1998). Trimodal neurons responding to visual, tactile, and auditory stimuli have also been observed (Graziano & Gross, 1998). These cells typically have spatially congruent receptive fields in the different modalities which can stimulate them. Such convergence of multimodal spatial inputs in posterior parietal cortex seems consistent with the fact that neglect can arise cross-modally (Vallar, 1998). Moreover, several recent studies (e.g. Ladavas et al., 1998; Mattingley, Driver, Beschin, & Robertson, 1997) have shown that an ipsilesional event in one sensory modality can extinguish the patients' awareness of a contralesional event in a separate modality. Ladavas and colleagues (Di Pellegrino et al., 1997; Ladavas et al., 1998) found in right-hemisphere patients that a visual event in the right hemifield extinguishes awareness of a touch on the left hand more strongly when placed closer to the (unstimulated) right hand. It is tempting to relate such cross-modal interactions to those found at a cellular level in neurons with spatially congruent receptive fields in vision and touch. Such neurons have recently been studied in parietal area VIP as well as in prefrontal cortex, and their visual receptive field is found to follow the tactile receptive field in space as the corresponding body part is moved (Duhamel et al., 1998; Graziano, Yap, & Gross, 1994).

Finally, recent single-cell studies in monkeys reveal that parietal neurons are involved not only in multimodal spatial integration of sensory input, but also in the early stages of planning intentional spatial movements. Area LIP seems to be intimately linked to saccadic eye movements (e.g. Li, Mazzoni, & Andersen, 1999), whereas other parts of the parietal lobe appear to be involved in the control of reaching rather than saccades. For instance, Snyder et al. (1997) found that parietal neurons in LIP respond strongest when their receptive field is the target for an upcoming eye movement (but not for a hand movement), whereas other, anatomically-segregated neurons (in the 'parietal reach region') show the reverse motoric preference. Parietal neurons may thus represent not only the location of a particular stimulus that has been selected for attention, but also some aspects of the decision to make a particular intentional response to it.

This recent evidence that some parietal areas serve as sensorimotor interfaces is consistent with findings that human neglect can involve both perceptual and motor components. It has long been suspected that human neglect may include motor deficits, although any such components were traditionally attributed to frontal rather than parietal damage (Bisiach et al., 1990; Mesulam, 1981; Tegner & Levander, 1991). Recent studies suggest that even neglect patients with focal parietal damage can have deficits in initiating movements towards the affected side, in addition to their perceptual impairments. For instance, they may be slow to direct their 'good' ipsilesional hand in the affected contralesional direction over and above their perceptual deficit for that side (Husain, Mattingley, Rorden, Kennard, & Driver, in press; Mattingley, Husain, Rorden, Kennard, & Driver, 1997), and may show saccadic biases also (Ro, Rorden, Driver, & Rafal, 2000).

13. Do parietal spatial circuits play a special role in perceptual awareness?

Early studies of blindsight (Weiskrantz, 1986) led to suggestions that visual awareness may depend on early visual areas of cortex. More recently, Crick and Koch (1995) sceptically asked 'Are we aware of neural activity in primary visual cortex?', to which we can now reply 'Not in the absence of coupled parietal activity' (see Rees et al., 2000; Vuilleumier et al., 2000). The evidence from neglect and extinction indicates that considerable sensory processing in the occipito-temporal visual system may take place, yet it may not be sufficient for conscious vision. This suggests that spatial circuits within the posterior parietal cortex may make some special contribution to perceptual awareness. We do not wish to imply that neurons here provide the *sole* neural correlate of perceptual awareness. We doubt that awareness depends on just a single critical brain region, but rather on the interplay between many brain areas in an extended network (Baars, 1997; Dehaene, Kerszberg, & Changeux, 1998; Lumer & Rees, 1999; see also Dehaene & Naccahe, Dennett, and Kanwisher in this volume). The sceptical reader may note that, even within this volume, different authors tend to stress the special role in awareness of those brain areas in which they themselves happen to specialize (be these in the parietal lobe, as here, or the frontal lobe, the hippocampus, the occipital lobe, the cingulate cortex, etc). Perhaps the more important point is the distinctive contributions which these different brain regions may make, and the interplay between them.

Inferior parietal cortex lies in a strategic position within a distributed neural network that interconnects different sensory and motor areas at the cortical and subcortical level (Mesulam, 1981). The recent monkey physiology indicates that posterior parietal areas serve as cross-modal and sensorimotor interfaces which highlight currently relevant locations as targets for intentional action. Parietal areas receive converging inputs from all sensory modalities, including not only vision, audition or touch, but also proprioceptive and vestibular signals about the position of the limbs, head, and eyes. All this information is required to compute stable representations of the location of external stimuli relative to the observer (e.g.

representations which are more stable than the purely retinal inputs to the occipital lobe, which shift every time an eye movement is made, as often as five times per second in daily life). Such disparate sources of spatial information are also required in order to compute how to respond spatially to external stimuli with the eye, head or hand. Neurons within individual parietal areas provide a highly selective representation of current sensory input, effectively responding only to the stimulus that has been selected as the current target for attention or for a particular spatial action. Moreover, they show 'intentional' activity in addition to sensory activity, corresponding to the earliest cortical stages of spatial motor planning, and projecting to various motor-output structures cortically and subcortically.[3]

Given all this evidence for the representation of currently relevant *location* information within the parietal lobe, perhaps the most intriguing aspect of the human neglect syndrome is that the patient does *not* lose awareness solely for the *location* of neglected stimuli, but for their other properties also, and indeed even for the very existence of the neglected information. This is despite the considerable evidence we have reviewed suggesting that the presence and nature of neglected stimuli may still be unconsciously processed in many patients. While the textbook neural pathways for, say, visual face perception and recognition (i.e. from the occipital lobe down into the fusiform gyrus and later temporal areas) may be structurally intact in many patients, under the appropriate conditions these patients can remain entirely unaware of the presence of a face on the left (even though it demonstrably still activates the appropriate occipital and temporal structures to some extent, as in the fMRI studies of extinction). It thus appears that when the appropriate representation of stimulus location is lost or degraded, as in neglect after parietal damage, then awareness of other stimulus properties (presumably coded elsewhere in the brain) is also lost, as if our internal representations of external space (at the level of multimodal sensorimotor interfaces) provided the basic medium for perceptual experience (see Bisiach & Berti, 1987; Robertson, Treisman, Friedman-Hill, & Grabowecky, 1997, for related proposals; see also

[3] Although there is some homology between monkeys and humans for the relevant brain areas, it should be acknowledged that differences also exist. Humans appear endowed with a particular development of regions in inferior parietal and frontal cortex (Eidelberg & Galaburda, 1984; Watson et al., 1994), and show more hemispheric specialization than monkeys. It is striking that in humans the right-hemisphere network of brain areas associated with neglect has some symmetry with the left-hemisphere network involved in aphasia, not only for inferior parietal areas in the supramarginal-angular gyri (Vallar, 1993; Vallar & Perani, 1986) but also for areas in the inferior frontal cortex (Husain & Kennard, 1996). Could some of those anatomical factors which led to a special role of these structures for *spatial* cognition within the human right hemisphere (i.e. strategic position for cross-modal links and for perceptual selection serving intentional sensorimotor transformation) apply also to the special role of left-hemisphere structures in human *language* (Logan & Zbrodoff, 1999; Mesulam, 1998) and numerical cognition (Dehaene, 1999)? One can even speculate about whether the latter cognitive abilities could somehow have evolved from mechanisms suited to selecting and tagging individuated objects as the targets for purposeful action (with acts like pointing serving as primitive communication and enumeration; Lakoff & Johnson, 1999). Moreover, such mechanisms may relate to a stimulus becoming 'reportable', the conventional objective criterion for awareness.

Kanwisher in this volume for related arguments based on a largely independent database, namely functional imaging in normals). We are not suggesting that other properties do not form part of our conscious percepts, but rather that there seems to be a neuropsychological asymmetry. Losing spatial awareness, as in neglect, invariably leads to losses of, say, 'colour awareness' or 'face awareness' in the affected locations for neglected stimuli there. But losing colour or face awareness (as in cerebral achromatopsia or prosopagnosia) apparently never leads to losses of spatial awareness for the affected colours or faces.

From a philosophical perceptive, this may in part reflect a special role for location in the mental representation of what is 'out there' (even this simple way of referring to perceptual experience in everyday language has a remarkably spatial emphasis). From a neurophysiological and mechanistic perspective, we suspect that it may reflect the extreme nature of the 'winner-takes-all' process in parietal cortex, which we alluded to above, as well as its particular position at the interface between sensory and motor processes. Judging by the recent single-cell evidence from monkey physiology, the function of many of the brain areas implicated in neglect is to select just the currently most relevant location, at a suitably abstract, multimodal and durable level of spatial representation, as the target for a particular type of action. The work in monkeys and other species suggests that the evolutionary origin of such extreme selectivity may lie in the need to select just *one* location as the target for the next eye, head or hand movement. A major limitation on any motor-output system, such as that for eye movements, is that no matter how big the brain, the eye can only be moved in one direction at a time. Hence, there is a very real need to have just a single 'winning' stimulus become the target for the next eye movement from among the many competitors in the cluttered scenes of daily life. This may be why the winner-takes-all principle seems to apply in such extreme form in parietal cortex and related areas, with perhaps only a single winner across the entire visual scene (e.g. Gottlieb et al., 1998).

An extreme winner-takes-all principle might explain why a lesion which biases the competition within parietal areas can have such dramatic effects on awareness for stimulus properties presumed to be coded in other brain areas (see also Hahnloser, Douglas, Mahowald, & Hepp, 1999). This point can be made by reference to Desimone and Duncan's (Desimone & Duncan, 1995; Duncan, 1996) 'integrated competition' approach to selective attention, which seeks to explain both physiological data and psychological observations. In their framework, multiple concurrent stimuli always compete to drive neurons and dominate the network (and thus ultimately to dominate awareness and behaviour). The various phenomena of 'attention' are cast as emergent properties of whichever stimuli happen to win this competition (see also Dennett in this volume). Particularly salient stimuli will have a competitive advantage and thus may tend to 'attract attention' (on purely bottom-up grounds), but the particular stimulus you are currently looking or listening out for may likewise possess some advantage (on purely top-down grounds) accrued by biasing the system in its favour, perhaps even by pre-activating the appropriate set of neurons (Chawla, Rees, & Friston, 1999; Kastner, Pinsk, DeWeerd, Desimone, & Ungerleider, 1999). Within this general framework, the

effect of a lesion (e.g. to an area representing candidate targets for attention, saccades, or reaches within one side of space) would be to bias the competition pathologically (in favour of the other side of space). Thus, extinction-like phenomena can be readily explained in much the same way as we suggested earlier.

A key feature of Desimone and Duncan's (Desimone & Duncan, 1995; Duncan, 1996) proposals is that they envisage winner-takes-all competitions to arise initially within many separate modules (e.g. in different areas of the visual system), but with some subsequent integration of the outcomes between modules, so that a strong initial winner in one module then becomes more likely to dominate competition within other models also. The system as a whole ultimately tends to settle on the same particular stimulus as the overall winner, producing those phenomena traditionally attributed to limited-capacity attention. This general perspective leads Desimone and Duncan to argue that no single brain area is special for selective attention, which is seen as the emergent property of competition arising initially within all the modules, and subsequently being integrated across all of them.

While we are sympathetic to many aspects of this powerful approach, we are less sympathetic to the implication that all brain areas are equal for selective attention. Although winner-takes-all operations may be a common principle within many different brain areas, as argued above they may take place according to more extreme rules within inferior parietal cortex (and other brain areas associated with neglect), than in, say, the occipital lobe or other primary sensory areas, where competitive interactions seem to arise on a more local scale (e.g. just between neighbouring visual stimuli (e.g. Polat & Sagi, 1994), rather than across the entire visual field) so that there may be several concurrent 'winners' within the occipital lobe. Parietal areas, each concerned with selection of one stimulus location as the target for a particular type of response, may provide a more clear-cut *single* 'winner' from cluttered sensory input.

If so, then one can begin to see how the integrated competition proposal could lead to a very extended network becoming dominated by whichever stimulus 'wins' in those particular modules that have the most extreme competition (i.e. within areas of parietal cortex, on our argument). Rather like a Presidential election, winning some Primaries may be more critical than others. This might explain why a pathological bias within brain areas that primarily represent just a currently relevant *location* could spread to produce a corresponding spatial bias in awareness for *all* forms of information, including those coded in other brain systems. This may correspond to the marked effects of spatial attention on perceptual awareness.

A further reason for a winner within the parietal competition to be spurred by its victory into dominating competition in other modules is that some degree of location coding exists in most visual modules, whereas coding of face and other object properties is restricted to one or a few. Hence, the outright winner in some parietal area coding space, for a specific purpose, may be well placed to communicate its victory to the various front-runners in other modules, as location may be the main communicative medium. The cascading effects of parietal victory on competitions within many other brain areas could in turn relate to ideas that the content of awareness corresponds to that information which becomes available to a

widely distributed network of areas (Baars, 1997; Dehaene et al., 1998; Lumer & Rees, 1999; see also Dehaene & Naccache, and Dennett in this volume).[4]

14. Moving beyond intuitions to the neural basis of awareness

There is currently much excitement about the possibility of relating awareness to neural substrates, and studies of neglect and extinction have much to offer this growing field. However, any pronouncements of 'consciousness explained' remain premature. Part of the problem is that neuroscientists wrestling with intriguing neurological or neurophysiological data often have, at best, only intuitive notions of what awareness constitutes in psychological or philosophical terms (see Block in this volume). Philosophy can of course help to sharpen intuitions and challenge preconceptions but we suspect that even the most sophisticated philosophies of mind may have to be revised drastically as the neuroscience progresses. For now, we cannot resist pointing out that many of the prevailing intuitions about (primarily visual) awareness have some clear resonance with current ideas about spatial function in the parietal lobe.

Visual awareness is highly selective with respect to the current retinal input (Rensink et al., 1997); so too is stimulus representation in the posterior parietal lobe (Gottlieb et al., 1998). Our awareness of visual space is more stable than that provided by fleeting retinal images across rapid eye movements (von Helmholtz, 1867); so too for spatial coding in the parietal cortex (Duhamel, Bremmer, Ben-Hamed, & Graf, 1997; Gottlieb et al., 1998). Many authors have suggested that visual awareness may relate to the binding of diverse properties coded in different brain areas (Baars, 1997; Treisman, 1998); speculatively, this could relate to the contribution of the inferior parietal lobe as an interface between dorsal and ventral streams (Driver, 1996; Heilman et al., 1997; Robertson et al., 1997), and between different modalities (Andersen et al., 1997). It may also relate to the knock-on effects of extreme winner-takes-all competitions within parietal areas, as we have proposed. Some authors suggest that visual awareness may depend more on entry into 'working memory' (Baars, 1997; Crick & Koch, 1995) than on less durable sensory activations. Although areas of prefrontal cortex are often invoked in such discussions, in fact parietal neurons also show clear sustained activity in delays periods of working memory tasks, where an animal must withhold a spatial response to a stimulus which has been removed until a go signal is presented (e.g. Andersen et al., 1985, 1997). Indeed, parietal areas may represent the current *contents* of working memory, with frontal areas instead controlling and manipulating what enters the proposed store (D'Esposito et al., 1998; Ungerleider, Courtney, & Haxby, 1998; Shallice, 1988). Finally, other authors suggest that perceptual awareness may reside in close contact with intentional motor planning and response selection (Allport, 1988; Crick & Koch, 1995; Rizzolatti & Berti, 1990). While such proposals are

[4] The combined ideas of more extreme winner-takes-all functions in parietal areas, plus communication between modules primarily via spatial codes, could provide the mechanism for 'wide broadcasting' which Block (this volume) considers to be lacking.

often accompanied by gestures towards the frontal lobes, in fact initial stages of cortical motor planning (or 'intention') seem to arise in the parietal lobe (Snyder et al., 1997), within the same neural populations as those which show extreme winner-takes-all functions, multimodal integration, and delay activity.

We would not suggest that perceptual awareness resides exclusively in the parietal lobe and those interconnected brain structures which are also implicated in neglect. But we would be amazed if these brain areas do not turn out to play a very major role.

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