Parietal neglect and visual awareness

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The last decade has seen a resurgence of interest in the neural correlates of conscious vision, with most discussion focused on the 'blindsight' that can follow damage to primary visual cortex, in the occipital lobe. We suggest that new insights into the neural basis of visual awareness may be gleaned from a different neuropsychological phenomenon, namely visual 'neglect' after injury to regions in the parietal lobe. Neglect provides several revealing contrasts with occipital blindsight. Here we summarise four key findings. First, unlike the deficits caused by damage to primary visual cortex, the loss of awareness in parietal neglect is characteristically not strictly retinotopic. Second, visual segmentation processes are preserved in neglect, and can influence what will reach the patient's awareness. Third, extensive unconscious processing takes place for those stimuli on the neglected side which escape awareness, including some degree of object identification. Finally, parietal damage affects initial stages of motor planning as well as perception. These findings are consistent with recent data on single-cell activity in the monkey brain. They also suggest why areas in the inferior parietal lobe may play a prominent role in visual awareness.

Visual neglect illustrates that visual awareness can be lost even when primary visual cortex, and its initial afferent inputs, are intact. Neglect can be observed in some form after various unilateral lesions, but is most commonly found[1,2] after lesions to the inferior parietal lobe, particularly in the right hemisphere (Fig. 1). The exact pattern of deficits seen in individual neglect patients can vary somewhat, in accordance with the exact details of their lesion. Here we concentrate on the many commonalities that are found, after the typical inferior-parietal lesion that is illustrated. A further reason to focus on the parietal lobe is that findings from patients with damage here are beginning to converge with physiological data from the parietal lobe in other primates, as we describe.

Patients with neglect after right-parietal injury have deficient awareness for stimuli towards the contralesional (left) side of space, especially when competing stimuli appear further to the right. In daily life, such patients may eat food from only the right side of their plate; ignore people who approach from their left; miss words on the left of the page when reading (or letters from the left of individual words); omit details on the left when copying pictures, and so on. A characteristic feature of neglect is that drawing the patient's attention to the neglected left information usually brings it back into awareness. In some cases, neglect can affect other sensory modalities in addition to vision (e.g. the patient may ignore sounds as well as sights on their left). We focus on just the visual deficits here, because at present these are the best understood. Although neglect was described at the turn of the century[3], its possible implications have been somewhat overlooked by recent discussions concerning the neural basis of visual awareness.

Most such discussions have focused instead on the phenomenon of blindsight, which can arise after damage to a much earlier area in the cortical visual system, namely primary visual cortex in the occipital lobe (also known as striate cortex, due to its striped appearance). Primary visual cortex provides a retinotopic map of visual space, with adjacent points on the retina being represented by adjacent points in the brain. Damage here can lead to a retinotopic 'scotoma'; that is, to a blind region in the retinally corresponding part of the visual field. However, it is now well known that some residual visual processing may still take place within this seemingly blind region. For instance, classic studies[4,5] showed that patients with lesions to primary visual cortex may saccade or point towards lights in their scotoma, while denying awareness of these stimuli. The retinotopic loss of visual awareness after striate damage, despite the residual processing, led some authors to speculate that primary visual cortex may play a special role in generating visual experience (see ref. 5).

Occipital blindsight has certainly provided important insights into the neurobiology of awareness. However, the critical point for our purposes is the contrast between occipital blindsight and parietal neglect, which may allow fresh insights. Perhaps the most puzzling aspect of neglect is why the patient so often remains unaware of visual information on the left, given that primary visual cortex is typically intact, and should therefore register the neglected stimuli (provided that the 'optic radiations' are spared, i.e. that the afferent projections along the geniculate pathway to striate cortex remain intact, which is true for many though not all neglect patients).

The deficit in neglect certainly cannot be reduced to a retinotopic scotoma like those found after damage to primary visual cortex. Neglect can occur even when there is no blind region in the visual field[6]. Conversely, blindness for one half of the visual field, after damage to primary visual cortex, does not by itself produce all the manifestations of neglect. Some parietal neglect patients have absolutely no apparent deficit for an isolated stimulus on the affected side. Their deficit in awareness only emerges when stimuli are presented on both sides simultaneously, in which case the previously detectable contralesional stimulus is now 'extinguished' from awareness by the competing ipsilesional stimulus. Such extinction is commonly thought to reflect an attentional rather than sensory disorder[7,8,9]. On this account, sensory input on either side can reach the patients' awareness, provided that no other events compete for attention. However,

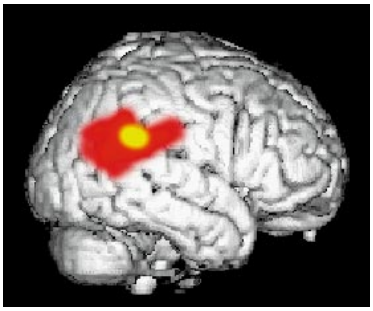

Fig. 1. Lateral view of the right hemisphere of the human brain, illustrating the parietal lesion commonly associated with left neglect[1,2]. This involves the supramarginal gyrus in the inferior parietal lobe, at the temporoparietal junction (yellow shading). The exact extent of the lesion will vary in individual cases (as indicated by the red shading). Persistent neglect is more common after right- than left-hemisphere lesions, for reasons of hemispheric specialization in humans which remain poorly understood, but which may relate to right-hemisphere dominance for spatial cognition, for attention to global properties of visual scenes, and for arousal. Whether neglect of comparable severity can be produced by experimental lesions in monkeys has been controversial. They have rarely been tested on exactly the same tasks as used in humans, and usually had more circumscribed parietal lesions. Moreover, one must be cautious as regards exact homologies between human and monkey brains. Nevertheless, a recent study[45] suggests that parietal leucotomy can produce neglect in monkeys, when neglect is defined as a unilateral spatial deficit that is more severe than occipital hemianopia. However, there is as yet no monkey evidence for hemispheric specialization in neglect like that found in humans.

when two simultaneous events appear on opposite sides, they now compete for attention, and so only the ipsilesional (right) event enters the patient's awareness, as it alone fully captures attention[9]. We will return later to the idea that parietal damage may have particularly severe consequences for attentive vision, and that this may underlie the loss of awareness in neglect.

Parietal neglect is not purely retinotopic

Some tests for neglect only reveal a deficit for contralesional events if a competing ipsilesional event is presented concurrently (as in the 'extinction' test described above). However, more sensitive tests (and/or more severely impaired patients) can sometimes reveal a degree of impairment even for an isolated contralesional stimulus[10]. Even when found for isolated stimuli, the parietal deficit still differs from the retinotopic scotomas caused by lesions to primary visual cortex, because it is characteristically not fixed in purely retinal coordinates. In one illustrative study[11], a neglect patient appeared to be blind in the left visual field when fixating straight ahead, or to her left. However, events in her left visual field became detectable when she fixated to her right, suggesting that the position of the visual target relative to her head or body was critical, not merely its retinal position. Similarly, Karnath and colleagues have found[12] that neglect for stimuli at a particular retinal position in the left visual field can be reduced by turning just the patient's trunk towards those stimuli, even though this does not change the retinotopic location of the visual inputs.

Such results show that extra-retinal information (inputs signalling the position of the eye in its orbit, or of the head on the trunk) can contribute to the spatial coordinates in which neglect arises. This contrasts with the strictly retinal coordinates of the scotomas produced by damage to primary visual cortex. The results for parietal patients are consistent with recent physiological data on the activity of single neurons in monkey posterior parietal cortex, indicating their involvement in the construction of spatial representations that integrate retinal and extra-retinal signals[13,14]. Such integration is required for many spatial visual tasks (e.g. reaching to a visual object), since on their own, retinal signals can only indicate the position of an object on the retina, and not its position relative to the head, the body, or the hand. Moreover, the retinal positions of static visual objects change during every eye movement, and so retinal signals alone cannot provide stable representations of visual position.

The evidence from neglect patients suggests that cell populations similar to those found in monkey parietal cortex, which integrate retinal and extra-retinal inputs, may play a role in generating visual awareness in the human brain. Since the spatial representations provided by such cells go beyond retinal coordinates, when lesioned they result in a loss of awareness that is, correspondingly, not strictly retinotopic (see ref. 15 for a

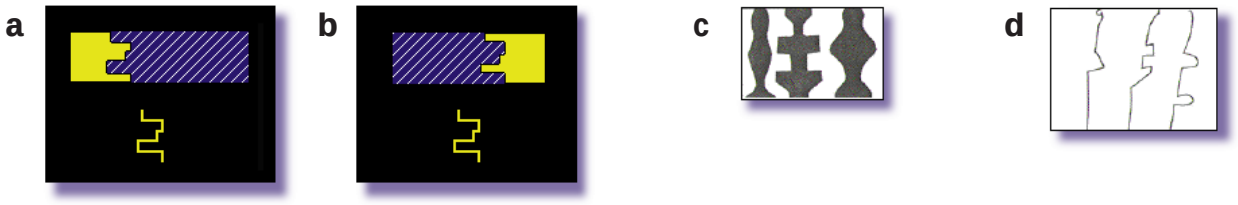

Fig. 2. (a) and **(b)** each show an example display from Driver *et al.*[18]. The task was to fixate the centre of the rectangle, and compare the single jagged edge inside it to the isolated edge which appeared centrally below, shortly after the rectangle disappeared. Normal viewers see the rectangle as comprising a yellow figural shape against a striped, shapeless blue background, due to figure-ground segmentation processes. Left neglect led to poorer performance for displays like (b) (where the initial jagged edge fell on the right of the patient, but on the left of the segmented yellow figure) than for (a) (where the jagged edge now fell on the 'good' right side of the segmented figure, even though it was further to the patient's left). **(c)** illustrates that normal viewers prefer symmetrical shapes (black here) as figures, with the intervening asymmetrical shapes serving as their background (white here). This phenomenon can be preserved in neglect patients[18], with the symmetry effect implying that both sides of each shape must still be represented by their visual system at a figure-ground segmentation stage. However, neglect patients are typically unable to judge symmetry explicitly[18], even though it determines their figural preferences, because they neglect the left side of the resulting figures. **(d)** shows a copy of (c) drawn by a neglect patient studied by Marshall and Halligan[20]. The patient attempted to draw only the right side of each symmetrical shape. This again implies that both sides of each shape were represented at initial figure-ground segmentation, but that the left of each symmetrical figure was subsequently neglected.

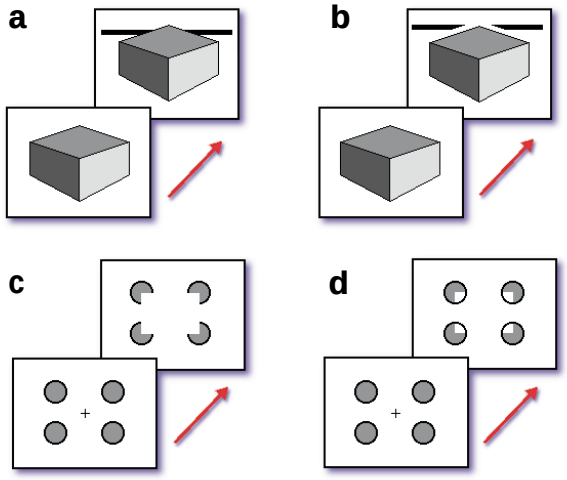

Fig. 3. Example display sequences from Mattingley *et al.*[25], with arrows indicating time. For **(a)** and **(b)**, each trial began with a central cube, followed by black bars which could briefly appear just on the left, just on the right, on both sides (as shown), or not at all. The patient's task was to respond "left", "right", "both" or "none", to indicate detection of any black bar. Note that in (a), the two black bars appear to form a single, partly-occluded rod lying behind the cube, due to image-segmentation processes. Severe left-sided extinction (responding "right" when bars actually appeared on both sides) was found for displays like (b), but was significantly reduced for displays like (a), where the two bars were segmented together as one. In **(c)** and **(d)**, each trial began with four circles arranged around a central fixation cross. In the following frame, segments could briefly be removed from just the left circles, just the right circles, the circles on both sides (as shown), or not at all, and the patient reported which occurred. Note that in (c) but not (d), the removal of segments leads to the percept of a single subjective figure between the circles. Left-sided extinction was eliminated in this situation, but was severe when the subjective figure was absent, as in (d).

neural-network simulation of this). Note that normal visual awareness, like neglect, also goes beyond purely retinal coordinates. The visual world does not appear to shift location whenever our eyes move, even though the corresponding image will shift its position on the retina[16]. Single-cell recordings from posterior parietal neurons in monkeys again suggest a possible basis for this; some units are found to update their spatial representation of the visual world immediately prior to an eye movement[17], as if predicting the retinal consequences of saccades. Thus, the discovery that parietal neglect is not strictly retinotopic[12] has intriguing parallels both with normal visual experience, and also with cellular activity in the parietal lobe of non-human primates.

Figure-ground segmentation can be preserved in neglect

A further discovery from recent neuropsychological research is that visual object-segmentation factors can powerfully influence what will be neglected by parietal patients. Neglect can apply to the left side of individual segmented objects, rather than merely to positions that are further to the left in the visual field (see Fig 2). This form of 'object-based' neglect has now been found in numerous cases, by separate research groups using various tasks[18-21], and so it appears to be a fairly general finding. It also fits with findings of object-segmentation effects on visual attention in normal human subjects[19,21].

The preserved figure-ground segmentation in neglect makes considerable sense from an anatomical perspective. Various lines of evidence (single-cell recording in monkeys, lesion effects in patients, and functional neuroimaging in normal people[22-24]) all point to areas of the occipital lobe as the likely site for figure-ground segmentation processes in early vision. These brain areas will remain intact in many parietal neglect patients, so their attentional deficit should indeed arise in the context of considerable visual preprocessing within the occipital lobe.

If image-segmentation proceeds relatively normally at early stages of visual analysis in the occipital lobe, with neglect only arising during later stages of vision, then under appropriate conditions the preserved segmentation might be capable of 'rescuing' visual stimuli that would otherwise escape the patient's awareness. As described previously, when two concurrent visual events are briefly presented, the more contralesional of the two is usually 'extinguished' from the patient's awareness, with the ipsilesional event capturing attention entirely. We and others[9,25,26] have recently found that such extinction can be eliminated if the two events get segmented as a single object by early visual analysis, such that they become allies rather than competitors in the bid to attract attention, and so reach awareness together. This was found[25] even when the link between the two events required completion behind an occluder (Fig. 3a and b), or the formation of a subjective figure (Fig. 3c and d).

These results are again consistent with single-cell data, and with functional imaging evidence, indicating that completion behind occluders can arise at fairly early stages of visual processing, in extra-striate cortical areas; and likewise for the formation of subjective figures[22,23]. Since these occipital brain areas should still be intact in many parietal patients (those with spared afferent connections from the lateral geniculate nucleus into primary visual cortex), we anticipated that the visual-segmentation processes should still operate in such cases, to modulate what reaches awareness, exactly as we found[25]. Note that these parietal results once again contrast with the strictly retinotopic scotomas that follow damage to primary visual cortex. In parietal neglect, whether or not a stimulus will reach awareness does not depend solely on its position in the visual field, but also on how it gets segmented, as determined by preserved occipital processes.

Unconscious processing of neglected information

Thus far, we have focused on factors which determine whether a visual stimulus will still reach awareness after parietal damage. We turn now to consider the fate of those stimuli which escape awareness, due to neglect. Recent studies have revealed considerable unconscious processing of neglected stimuli, usually with 'implicit' measures (i.e. with methods that reveal some influence of neglected information, yet without requiring the patient to report explicitly what the effective information is, which is typically precluded by their neglect). Superficially, implicit processing in neglect might seem analogous to the residual processing found in occipital blindsight. For instance, in both blindsight patients and neglect patients, reaction times to a visual stimulus in the intact field can be modulated by the presence of an undetected stimulus in the affected region of space[27]. However, further studies have shown that unconscious processing of neglected stimuli in parietal patients is more extensive than that found in occipital blindsight patients, especially as regards object identity.

In addition to the implicit effects produced by the mere presence of neglected visual stimuli, parietal patients often show considerable implicit knowledge about the nature of such stimuli.

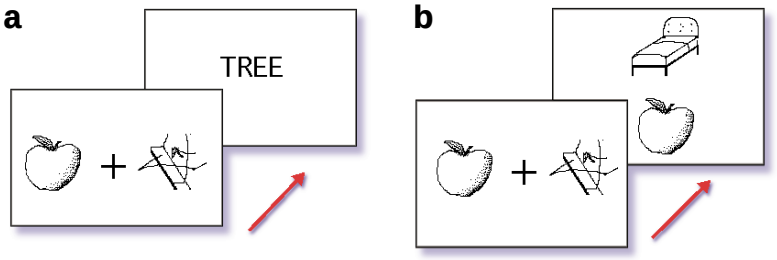

Fig. 4. Example display sequences from McGlinchey-Berroth *et al.*[30], with arrows indicating time. Each trial began with a meaningful visual object on one side of central fixation (the left in the illustration), together with a meaningless scrambled pattern on the other side, presented together for 200 msec. For the situation depicted in **(a)**, this was followed by a central letter-string, and the task was to judge rapidly if this string was a word or non-word. On some trials, the meaningful object was semantically related to the subsequent word (as for the association between the picured apple and the subsequent word 'TREE'). Such a relation speeded the word decision significantly for the neglect patients (thus revealing 'semantic priming'), regardless of whether the preceding related object had appeared on the left or right side. However, a control task **(b)** found that the neglect patients were unable to report the identity of the left object (i.e. they could not choose whether the upper or lower of two immediately-following objects matched the left object), although they could explicitly report right objects in this way. Taken together, these results suggest implicit semantic processing (i.e. activation of object identity and associations in the brain) for neglected left objects following right parietal damage.

For instance, recent studies indicate that attributes of neglected stimuli such as colour and shape still get encoded by the neglect patient's visual system, despite the loss of awareness[8,28,29]. Even more strikingly, the identity and meaning of a neglected visual stimulus can be activated unconsciously in the parietal patient's brain, as revealed by recent demonstrations[30,31] of preserved 'semantic priming' from neglected objects (see Fig. 4 for an explanation). In contrast, no such semantic priming was found[30] in the impaired visual field of a patient with unilateral occipital damage. Thus, residual unconscious processing seems more extensive in parietal neglect patients than in occipital patients; in particular, object identity and semantics may only be activated in the neglect patients. Intriguingly, close parallels have now been noted[31] between the residual processing found for neglected objects, and that observed for unattended objects in normals (e.g. both can involve some implicit object identification).

The extensive residual processing in neglect can be understood in terms of the known organization of the primate visual system. There is a general consensus that two streams of cortical visual processing can be broadly distinguished[32,33]. A 'dorsal' stream, which projects from primary visual cortex via extra-striate regions into the upper regions of the parietal lobe (i.e. superior to the typical lesion that produces neglect), is thought to be involved in the spatial control of action. A more 'ventral' stream, which projects from striate cortex via extra-striate regions into the temporal lobe (i.e. inferior to the typical lesion that produces neglect), is concerned with object recognition. Damage to primary visual cortex (as in blindsight cases) will disrupt the ventral object-recognition stream from its outset (as well as disrupting more dorsal projections from striate cortex). By contrast, the parietal lesion in neglect should leave much of the ventral object-recognition stream intact. Hence, more residual object recognition would be expected to arise along this ventral pathway in neglect, consistent with the extensively preserved implicit processing that is found for neglected objects.

Indeed, the challenge for current neuropsychological theories is not in accounting for this preserved processing in neglect; rather, it lies in explaining the dramatic loss of awareness itself, which arises for neglected stimuli despite the considerable processing that they evidently receive within the ventral object-recognition stream, as revealed by the priming effects. This disruption to awareness after parietal injury seems puzzling for accounts which associate visual awareness specifically with the functioning of posterior areas along the ventral stream (see ref. 33), or with primary visual cortex itself (see ref. 5).

Crick and Koch[34] have recently challenged the prevailing tendency to assume that visual awareness must be produced by posterior visual areas. They specifically proposed that people are not directly aware of activity in primary visual cortex. They also suggested that visual awareness may be intimately related to the planning of voluntary movements, arguing that this is its major adaptive function. They conjectured that neurons might therefore need a direct projection to motor planning systems, in order to be capable of generating awareness. We shall not provide a full assessment of their conjecture here, but instead will simply highlight its potential relevance to parietal function. Although most discussions of their conjecture have focused[34-36] on the importance of the frontal lobe in voluntary action, the initial stages of cortical motor planning in fact arise in the parietal lobe. That is, the parietal lobe is not only involved in perceptual awareness, but also in some aspects of motor control, as we discuss next.

SENSORIMOTOR FUNCTION OF THE INFERIOR PARIETAL LOBE

Areas in the superior parietal lobe (lying above the typical neglect lesion) are well known to be involved in the sensory control of movement[33]. Lesions here can produce gross misreaching to targets under visual guidance[2,33], rather than the loss of visual awareness that is seen in neglect after inferior parietal damage. Accordingly, the superior parietal lobe is commonly thought to subserve motor functions, with no involvement in perceptual awareness[33].

It is less widely appreciated that the inferior parietal lobe (i.e. lying below the superior parietal lobule, at the site of the most common neglect lesion) may also have a motor role. Many authors have noted that clinical neglect can involve pathological motor biases (specifically, a reluctance to respond leftwards, even with the ipsilesional right hand), in addition to the loss of awareness[37,38]. Such motoric aspects of neglect were previously thought to follow frontal lesions, rather than inferior parietal damage, but the evidence taken to support the apparent frontal association has recently been questioned[38]. Moreover, a recent study[39] showed that even neglect patients whose damage is restricted to the right inferior parietal lobe (sparing superior parietal and frontal cortex) can exhibit specific motor impairments, in addition to their pathological loss of visual awareness. In particular, such patients are exceptionally slow to initiate leftward movements of their right hand towards visual targets in left hemispace (Fig. 5), over and above their left-sided visual impairment. This specific motor-initiation deficit was absent in neglect patients with more anterior right-hemisphere lesions (sparing the inferior parietal lobe), who were delayed in responding to left visual

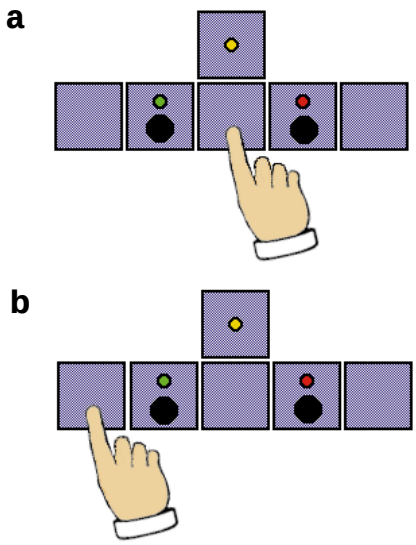

Fig. 5. Schematic illustration of Mattingley *et al.*'s study[39] of motor deficits in neglect. The task was to fixate a central light (shown as yellow in each figure), and then reach towards whichever peripheral light turned green (this is illustrated for the left light in each figure, but the right was equally likely to go green), pressing the button immediately beneath the green light. **(a)**, the hand initially rests at a central start position, so that left targets require a leftward reach, and right targets a rightward reach. Responses to the left target were initiated very slowly by patients with right inferior-parietal damage. **(b)**, the hand starts from an extreme left position, so even a left target now requires a rightward movement. Responses to the left light were much faster in this situation for the inferior-parietal patients (although they still showed some delay in visual awareness for left visual events). There was no such effect of changing reach direction in neglect patients with more anterior right-hemisphere lesions. Control experiments confirmed that the inferior-parietal results were indeed due to the direction of reach that was required, rather than merely to sensations from the hand when it rested on the left.

targets regardless of the direction of movement required. By showing a specific motor deficit in inferior-parietal neglect patients, which was absent in frontal neglect patients, this result challenges the previous orthodoxy that motor aspects of neglect are caused only by frontal damage.

The motor deficit in the inferior parietal patients[39] has parallels with recent single-cell recordings in monkey posterior parietal cortex. Cells there were previously thought to have primarily sensory or attentional roles[17]. However, a recent study[40] observed neural responses that were not only tuned to the visual location that was relevant for ongoing behaviour, but also for the kind of movement being planned towards it (a saccade versus a reach).

The recent patient data[39] thus suggest that the inferior parietal lobe is involved in the initial stages of generating motor intentions, in addition to its important role in visual awareness. This seems particularly intriguing in the context of Crick and Koch's proposal[34] that visual awareness is closely linked to initial motor planning. Future discussions of their conjecture might usefully consider the role of the inferior parietal lobe, in addition to the premotor and prefrontal cortices that have been discussed to date[34-36].

THE INFERIOR PARIETAL LOBE AND VISUAL AWARENESS

The loss of awareness in neglect implies that the inferior parietal lobe and its connections contribute to visual experience. Recent findings on neglect, together with single-cell recordings in monkey parietal cortex, suggest several reasons for this. The first relates to extra-retinal contributions in the perception of visual space. As noted earlier, the stability of our visual world across saccades suggests that normal visual experience is not strictly retinotopic. The parietal lobe contains the first cortical visual neurons known to combine retinal with extra-retinal signals in their coding of visual space[13,14,17]. This in turn fits with recent findings that the loss of awareness in neglect after parietal damage is not purely retinotopic[12], unlike scotomas after damage to primary visual cortex. These results suggest that one contribution of the parietal lobe to visual awareness lies in combining retinal visual signals with other sources of information, to provide the more stable representation of visual space that we experience.

The second insight from neglect research is that the loss of awareness following parietal damage arises even though considerable processing still takes place for neglected stimuli. This implicit processing includes figure-ground segmentation and visual completion (presumably in the occipital lobe[22-25]), and even the activation of object identity and semantics[30,31] (presumably along the ventral pathway into inferotemporal cortex[31-33]). This extensive processing is evidently insufficient to produce visual awareness when a lesion prevents appropriate activation of the inferior parietal lobe (and its forward and back projections), particularly when competing stimuli are present, as in extinction tests. Future studies of neglect should use functional imaging measures to directly study the neural activity produced by neglected visual stimuli, within the visual pathways that remain intact after the lesion. This should reveal any role that connections from the inferior parietal lobe may play in modulating activity in the ventral stream, or in other visual areas. We have proposed[19,31] that the inferior parietal lobe may serve to link properties extracted in the ventral stream (e.g. abstract object identity) to those properties extracted more dorsally (e.g. the position of the object, and the current layout of its surfaces, relative to the observer). Normal visual awareness may depend on such linkage[31,41]. The inferior parietal lobe, in terms of its connectivity, certainly seems well placed to act as an interface between the ventral and dorsal streams in this way.

A further recent finding from neglect is that the inferior parietal lobe appears to be involved in the initial stages of motor planning, in addition to its role in perceptual awareness[39]. This seems consistent with single-cell findings[13,17] indicating that parietal neurons are neither exclusively sensory, nor exclusively motor, but rather lie at the interface between these functions. Such findings may in turn relate to Crick and Koch's conjecture[34] that visual awareness is closely related to initial stages of motor planning.

Finally, we turn to perhaps the major reason for the parietal lobe's close involvement in visual awareness, namely its role in selective attention[7,31,41,42]. Neglect is increasingly regarded as a deficit in attention, and the findings reviewed here do indeed suggest that early 'preattentive' vision[43] remains largely intact after parietal injury, as shown by the preserved image-segmentation and implicit processing that we have described. Moreover, specific abnormalities of spatial attention have been documented in parietal patients[7]. On the other hand, spatial attention effects have now been observed for single neurons in many cortical visual areas of the monkey, and this has led some authors[44] to argue that no single brain area is predominant in the control of attention. However, spatial attention effects do seem to be par-

ticularly pronounced for cells in the parietal lobe, consistent with its special role in neglect. For instance, a recent study[42] showed that posterior parietal neurons have an exceptionally sparse representation of the visual world, in effect responding only to the currently attended object among a cluttered scene, even across eye-movements that drastically change the retinal input.

Normal visual awareness depends critically on selective attention; the same retinal stimulus can enter or escape our awareness as a function of purely attentional factors[31,43]. This fundamental point has been overlooked by most recent discussions on the neuropsychology of visual awareness, but is strongly emphasized by the recent work on neglect. We hope the findings on neglect that we have described will bring the issue to the attention of those interested in the neural correlates of conscious vision; and conclude that the contribution of the inferior parietal lobe to awareness can no longer be neglected.

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