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Review

The contribution of spatial remapping impairments to unilateral visual neglect

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Abstract

Left visual neglect following right hemisphere damage is a heterogeneous phenomenon, in which several underlying impairments have been identified. Despite recent advances in understanding the neural and cognitive bases of these impairments, current theories of neglect, particularly those that emphasise attentional deficits, do not explain a number of phenomena, including:

- 'Ipsilesional' neglect after left orienting
- Positive or 'productive' manifestations
- Spatial transposition errors
- Mislocalisations
- Revisiting behaviour during visual search
- Lack of awareness for objects toward the contralesional side of space

We propose that these manifestations of neglect can be accounted for by an additional underlying disorder of spatial remapping due to parietal dysfunction. In primary visual areas, retinotopic maps are renewed and thus overwritten at each new ocular fixation. Remapping processes operating in higher-level oculocentric visual maps of the parietal cortex ensure visual integration of these successive retinal images over time and space, by creating a constantly updated representation of stimulus locations in terms of distance and direction from the fovea. They consist in the storage, refreshment and re-localization of the different components of the visual scene that are successively attended during its exploration, and provide spatial constancy of visual perception and a spatial buffer for working memory [Cereb Cortex 5 (1995) 470; Visual Cogn 7 (2000) 17]. We begin this article by reviewing theoretical and experimental arguments that have highlighted the importance of parietal remapping processes in maintaining an accurate representation of space across saccadic shifts. We then focus on findings from the double-step saccade task, [Ann Neurol 38 (1995) 739] as a basis for our model of the role of remapping impairments in many of the symptoms of neglect. From these results, remapping impairments would be demonstrated when a saccade has to be guided across the midline after having fixated an object in either the left or right visual field for patients with either left- or right-side parietal lesions. In addition, patients with right-side lesions will have remapping impairments within the left visual field following a saccade to a left-side target (see Fig. 5). In a large part of the article, we seek to build our hypothesis based on this basic model and more speculative assumptions supported with extensive evidence from the literature.

Keywords: Spatial remapping; Visual representation; Neurones; Spatial hemineglect; Parietal cortex

1. Introduction

Unilateral spatial neglect is commonly observed after right hemisphere lesions in humans [4], particularly those that affect the temporo–parietal junction (inferior parietal lobule (IPL; [5]) or superior temporal region [6]). It is usually

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described as a failure to report, orient toward or respond to left (contralesional) stimuli. The clinical manifestations of visual neglect are heterogeneous [7,8], although a core component involves a spatial bias of selective attention that favors ipsilesional over more contralesional locations [9]. Despite recent advances in understanding the symptoms of neglect, there is still no coherent explanation for the *lack of consciousness* for left space, or patients' failure to compensate for their deficits in perception and exploration. Why do patients fail to detect all the targets in a visual cancellation test, apparently unaware of their contralesional omissions, when they are able to correctly tick the four corners of the testing sheet beforehand [10]? Why do they copy only half a daisy or a house, apparently unaware that they have not completed their drawing, when they retain knowledge of all their components [11]? Why do they report only the ipsilesional side of well-known scenes from mental (visual) imagery [12,13]?

Attentional theories of visual neglect have distinguished two main pathological components: a reduced spatial scale of attentional selection (i.e. a local bias; [14,15]) and an ipsilesional bias in the allocation of spatial attention (i.e. an orienting bias; [16,17]). In the first part of this article we consider evidence for these attentional impairments. We then review evidence which suggests that an additional key component of neglect involves an impairment of spatial remapping during shifts of attention, both overt (accompanied by eye movements) and covert (with visual fixation maintained).

2. Identified components of visual neglect

Lesions of the right hemisphere have been shown to induce a *local bias* of attention (reviews in Refs. [15,18]). Many neglect patients tend to focus their attention on the local features of a scene or object, and thus may have difficulty perceiving its global configuration [10,19].

Most neglect patients also exhibit an *orienting bias* toward the side of space ipsilateral to the lesion [16,17], leading them to neglect the presence of stimuli on the contralesional side. Many neglect patients begin to explore visual scenes toward the ipsilesional side of space, and often restrict their exploration to this side. Note that the ipsilesional orienting bias has been suggested to arise from the distribution of receptive fields of neurones in the parietal cortices that represent the right or the left visual fields [20]. In monkeys, the number of neurones representing the visual environment in each cortical hemisphere consists of an asymmetric curve, with most neurones responding to visual stimuli located around 15° within the contralateral hemifield, and a decreasing number of neurones responding to visual stimuli from this optimal location to the periphery of the ipsilateral field (Fig. 1a adapted from unpublished work of Ben-Hamed and Duhamel; see Ref. [20]). After unilateral parietal damage, this distribution of receptive fields of

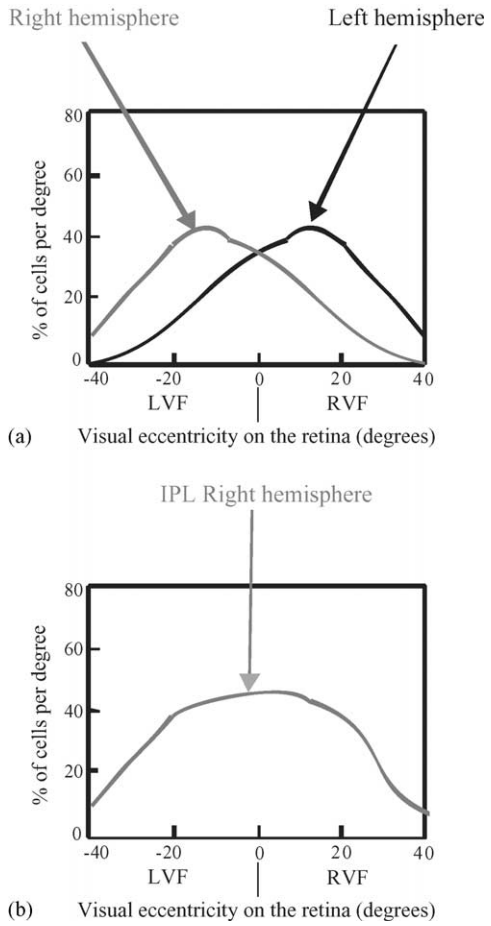

Fig. 1. Prediction of the spatial distribution of the parietal cortex neurons' receptive fields for the right and the left hemisphere separately. (a): The curve for the left hemisphere is derived from the actual graph plotting obtained by Ben-Hamed and Duhamel (see Ref. [20]) for a large sample of neurons recorded in monkey area VIP of the left hemisphere. Note that, as expected, neurons mainly respond to the contralateral visual field. However, the percentage of cells responding to the ipsilesional hemifield is still not negligeable. From $-40°$ to $+20°$, a gradual increase of neural representation within the left hemisphere is observed. The same gradient may be expected within the left and right human superior parietal lobules (SPL). (b): However, many arguments exists for an asymmetry in the human parietal cortex for spatial processing in favour to the right hemisphere. Imaging data suggest that the asymmetry would rely on a bilateral representation of space specifically in the right inferior parietal lobule (IPL).

neurones in the spared hemisphere leads to a gradient of spatial representation, with most neurones responding to the ipsilesional visual field and fewer representing progressively more contralesional locations. Behavioral correlates of this gradient can be found in the gradually slower reaction times to visual targets presented at different horizontal eccentricities from ipsilesional to contralesional side observed in left spatial neglect [21].

In order to fit with observations demonstrating that visual neglect is more severe and chronic after right hemispheric lesions [4] and that the right hemisphere may be able to direct attention to both sides of space [16,22–24], it is postulated that in humans the representation of visual space from the distribution of neurones in the right parietal

cortex is a more symmetrical, bell-shaped curve (Fig. 1b). From recent brain imaging studies [25–27], we suggest that this representation of both the left and right visual fields within the right hemisphere in humans may hold for the IPL, but not for the superior parietal lobule (SPL).

Attentional theories postulate that adding a local bias specific to right brain damage, an orienting bias, and also a reduction of overall attentional capacity [28] provides a satisfactory framework for explaining left visual neglect. Phenomena of attentional competition between stimuli have nicely completed this framework [29]. This framework can explain a failure to orient and respond to contralesional stimuli that is more severe for right brain-damaged patients. However, we will argue in the present paper that it does not alone provide a compelling explanation for neglect behaviors that we will describe in detail in the present review, such as right *ipsilesional* neglect after leftward orienting [30], *positive/productive manifestations* [31]), *transpositioning errors*, alloesthesia and *mislocalisations* [32,33], *revisiting behavior* [34] and, more crucially, for the characteristic *lack of awareness* for left space [35]. So-called 'representational theories' of neglect [36,37] offer some attempt at an explanation for the lack of conscious awareness of neglected stimuli, but even these accounts fail to indicate how a representational deficit might be realised at the neural level. We believe this can be successfully achieved by considering the role of *remapping mechanisms in the dynamic representation of visual space*, and the particular contribution of parietal neurones in their instantiation.

3. Remapping processes: definitions and impairments after parietal damage

3.1. Remapping and normal attention: model for the 'change blindness' phenomenon

The lack of awareness for obvious visual stimuli characteristic of spatial neglect can also be elicited in normal healthy observers under appropriate conditions. Even healthy individuals often fail to detect changes in images of real-world scenes when these are made during a saccade, flicker, blink, or movie cut, a phenomenon known as *change blindness* [2]. An analogy can be made between the lack of conscious perception in neglect patients and this change blindness in healthy subjects: in both cases, the primary visual areas receive afferent visual information from the retina, but a subset of the visual inputs is not consciously detected [9].

The impression created by vision is that of a coherent, richly detailed world where everything is present simultaneously. Although our environment is certainly this way, the phenomenon of change blindness provides strong evidence against the idea that the brain contains such a 'picture-like' representation of the visual world that is stable, coherent and everywhere detailed. Visual information is sampled at high resolution over only a few degrees of visual angle at the fovea. A complete representation of a scene therefore requires the contents of individual eye fixations to be integrated over space and time [2]. Viewed from this perspective, vision is an active process involving constant covert and overt exploration of the external world supported by dynamic updating processes. Remapping mechanisms are responsible for the integration of these different points of view over time and space.

In their computational model of change blindness, Niebur and Koch [38] postulated that visual perception consists of two stages of processing. In the first, early, stage extraction of elementary features is performed in parallel across all locations in the visual scene and coded with different activity on a *salience map* depending on bottom-up processes (e.g. contrast, colour, motion, etc); note that this salience map can be modified by top-down processes (e.g. during visual search for a specified target object). In a second, later, stage there is detailed processing via the serial application of covert or overt attention at different spatial locations within the visual scene (the focus, or 'spotlight' of attention; see Ref. [39]) implementing a *winner-take-all* array of space representation, i.e. only the most salient elements are selected for this upper representational stage, with respect to a threshold. Only information represented at this later stage of processing is available for conscious report. According to this view, a change that occurs in the visual scene at a non-attended location will not be processed at the level of conscious perception, leading to change blindness.

3.2. The 'salience map' and its role in prioritising spatial representations

It is well known that there are limits in the amount of sensory information that can be processed simultaneously [40]. In vision, as in other sense modalities, there is a need for mechanisms to prioritise which stimuli will be selected for further processing, i.e., selectively attended. Koch and Ullman [41] have postulated the existence of a topographic feature map, which codes the 'salience' of all information within the visual field. By definition, the amount of activity at a given location within the salience map represents the relative 'conspicuity' or relevance of the corresponding location in the visual field. The autonomous, sequential selection of salient regions to be explored, overtly or covertly, is postulated to follow the firing rate of populations of neurones in the underlying network, starting with the most salient location and sampling the visual input in decreasing order of salience [42]. The salience map would thus be the first representational level from which the pattern of attentional or ocular exploration of the visual world is derived.

Is there a neural correlate of these two feature maps proposed to account for normal perception? The concept of a salience map has been used by electrophysiologists to describe the type of visual representation constructed by the parietal cortex. Single-unit recordings from the lateral

intraparietal area (LIP) suggest that the primate parietal cortex contains a relatively sparse representation of space, with only those stimuli that are most salient or behaviorally relevant being strongly represented. For instance, Gottlieb, Kusunoki and Goldberg [43] recorded the activity of single neurones in macaque LIP. The receptive field of each neurone was first assessed in a passive task in which visual stimuli were flashed during eye fixation. Neuronal responses were then assessed when stimuli were brought into the receptive field of the cell by a saccade (Fig. 2). A circular array of eight stimuli remained visible from the beginning of the experiment, so that when the monkey made a saccade towards the center of the array, the receptive field of the cell matched the location of one of the array elements. In this condition, the same stimulus that activated the cell strongly in the passive condition entered the receptive field but elicited a significantly attentuated neuronal response. In a variant of this task, only seven symbols of the stable array were present initially on the screen; an eighth symbol then appeared within the cell's receptive field immediately prior to the monkey making a saccade toward the center of the array. This time, the neurone responded intensely, indicating that its activity was critically dependent on the abrupt onset of the stimulus, which rendered it salient. In another variant, when the monkey maintained peripheral fixation, a cue appeared that matched one stimulus of the array. Then the monkey made a first saccade to the center of the array (bringing one array stimulus into the receptive field) and a second saccade to the cued array element. When the cued element was brought into the receptive field by the first saccade, the neurone discharge started around the first saccade and continued until after the second saccade. In contrast, when an uncued element of the array was brought into the receptive field, the neurone did not respond to the irrelevant item, even though it entered the receptive field by means of the first saccade.

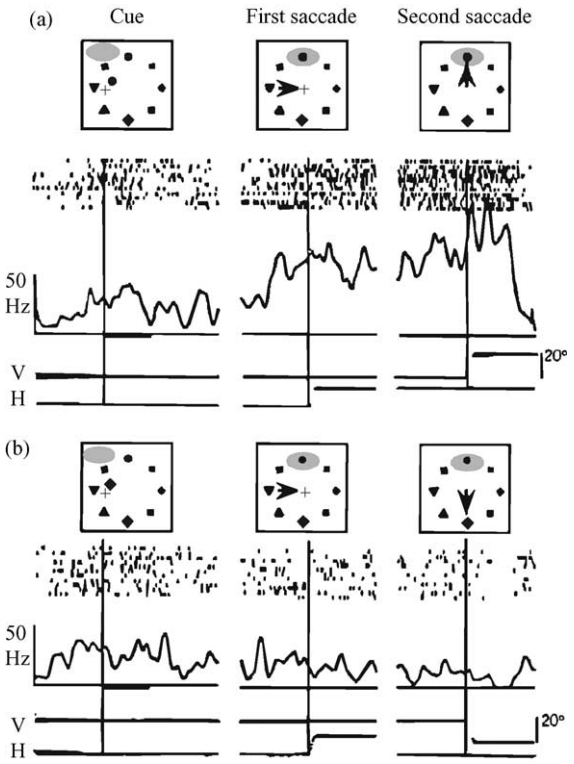

Fig. 2. Response of the same parietal neuron during a double-step task instructed by a cue in a condition where the stimulus entering its receptive field is relevant for the task (condition a) and in a condition where it is not relevant for the task (condition b). The cue (first panel) indicates the target of the array to look at, after looking at the array centre. The triangle of the first panel indicates the initial position of the eyes. The receptive field of the neuron, represented by a shaded ellipse, moves with the saccade. In both cases a and b, the receptive field of this neuron matches the location of the permanent stimulus presented at the top of a circular array of eight stimuli after the first saccade (second panel). When the second saccade has to be guided to this top position (condition a), the firing of the neuron precedes, and is maintained after the second saccade. However, when the second saccade has to be guided to another position (condition b), the neuron remains silent although the stimulus is similarly present in its receptive field. Adapted from Ref. [43].

Taken together, these results suggest that LIP neurones have little or no response to visual stimuli in their receptive field unless the stimuli are salient or behaviorally significant for the task. This area of the parietal lobe provides one potential neural substrate for the first level of spatial representation proposed by Niebur and Koch [38], according to whom visual perception corresponds not to a basic retinotopic representation of the visual input, but to a complex, prioritized interpretation of the environment.

3.3. Effect of graded representations within the salience map

We suggest here that the salience map is pathologically biased after parietal damage, such that visual inputs arising on the contralesional side have relatively little salience, and thus only minimal attentional weight. Recall that following unilateral parietal damage, the pool of spared neurones is proposed to exhibit a spatial gradient with a maximum response to stimuli in the ipsilesional visual field [20]. Since the parietal cortex is postulated to provide the neural substrate for the salience map, this map is also likely to have a left-right gradient. Consequently, the amount of activity associated with the presentation of the same visual stimulus will change depending on its position in azimuth, so that when located toward the ipsilesional side the stimulus will be represented by a larger number of neurones, which in turn will yield a stronger representation in the salience map. For example, following right hemisphere damage the representation of the same stimuli distributed across visual space will tend to decrease in strength from the right to the left side (Fig. 3c). This biased salience map explains why patients with left visual neglect systematically orient toward the right extremity of a stimulus array during standard clinical tests (e.g. line bisection and cancellation), before progressively exploring some distance toward the left side ([44–47]; Fig. 3b).

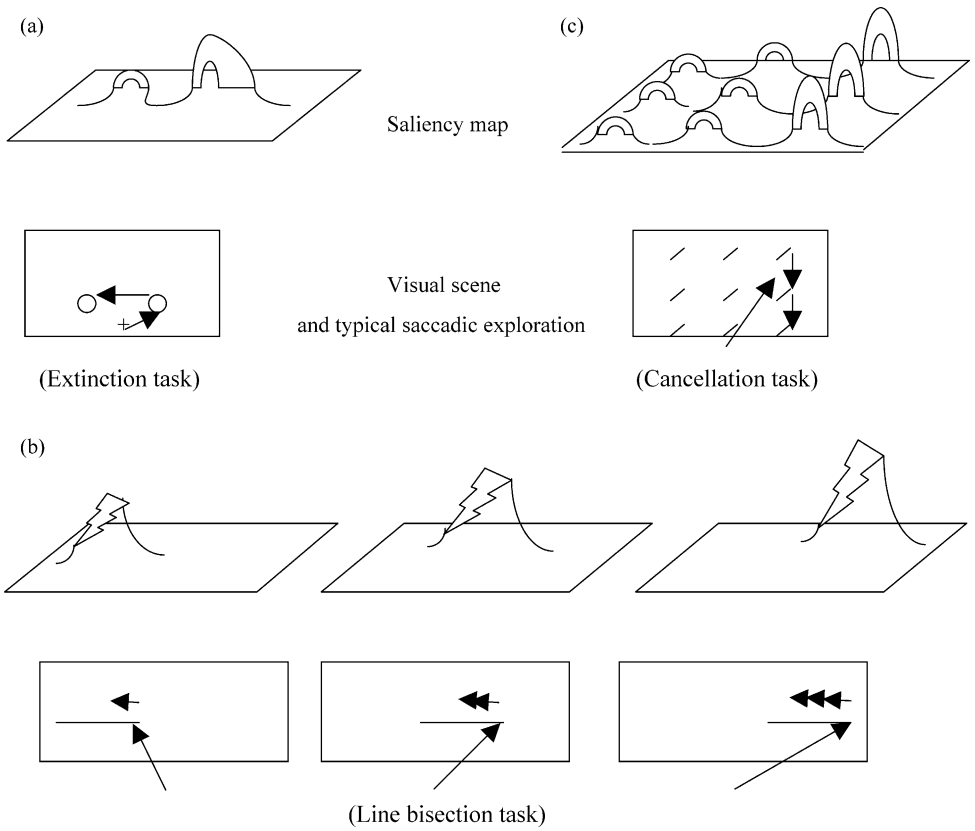

Fig. 3. Predicted representation on the salience map in left neglect of the stimuli presented during (a) an extinction task, (b) a line cancellation task and (c) a line bisection task with the line presented at different horizontal positions. The stronger the representation of the stimulus, the higher the activity at the corresponding location on the salience map schematised above. Note that the same stimulus presented at different locations in the azimuth will not be represented at the same level in neglect patients, as a result of a biased (graded) salience map. The classical pattern of ocular exploration in these patients is superimposed on the stimuli (eye movements represented by arrows) and is supposed to follow the representation on the salience map from the highest location to the lowest.

The nature of the ipsilesional orienting bias associated with parietal lesions has recently been explored by Ro et al. [48] using an extinction-like paradigm in which two identical stimuli were flashed in the right and the left visual fields, with different stimulus onset asynchronies (SOAs). In the perceptual task, patients were instructed to press a button to indicate whether the left or right stimulus was flashed first. In the saccade task, the patients made an eye movement toward the first stimulus to attract their gaze. Both left and right parietal patients showed dissociated performance between the two tasks, for a given SOA. They made accurate perceptual judgments of the first flashed target, as revealed by their button-press responses, whereas their saccades were biased toward the stimulus flashed in the ipsilesional visual field (Fig. 3a) for SOAs between +50 and −50 ms. Ro et al. [48] suggested that the orienting bias of their patients consisted in a pathological ipsilesional orientation of the first saccade rather than deficient conscious perception of the contralesional stimuli. Nevertheless, the ipsilesional orienting bias associated with parietal damage should not be considered as a primary motor deficit but rather as a bias in saccade generation due to the gradual parietal representation of visual space at the level of the salience map (first stage of space representation). According to the model of Niebur and Koch [42], this bias in saccade generation constrains the implementation of the second (later) stage corresponding to the conscious space representation (the 'winner-take-all' array), implying that both levels will show a pathological gradient of space representation. However, the result of Ro et al. [48] suggests that the salience map and the winner-take-all array may have separate neural substrates in humans. Furthermore, it is possible to distinguish between the ipsilesional bias and the perceptual remapping impairments as additional and dissociated components of neglect. One possibility we develop here is that the salience map may be constituted by the two symmetrical representations of contralateral space within the human right and left SPL, and that the upper conscious level of visual space representation may be located in the human right IPL. Indeed, we speculate that the bias in saccade generation is possibly common to neglect and extinction and creates a lack of consciousness for left events only in the non-ecological situation of simultaneous and very brief target presentation. Extinction-like biases have been obtained in normal subjects by applying transcranial magnetic stimulation (TMS) to both the right or left SPL [119], which can be proposed as neural substrates for the first level (salience maps) of the model

of Niebur and Koch [38]. A deficit at the second level creating a constant lack of consciousness for left stimuli might be more specific to patients with neglect. For example, the ipsilesional bias in the perceptual line bisection test (the 'landmark' test-[49]) is acknowledged as a characteristic bias of neglect [50] and is observed in normal subjects specifically during TMS applied to the right IPL [120]. The results of Rees [51] and Beck et al. [52] showing specific activation of the right IPL as a correlate of conscious visual experience are additional arguments for our speculation that the right IPL is an important anatomical correlate of conscious visual space representation.

3.4. What is spatial remapping?

In the previous section, we outlined a model for the nature of visual representations that arise when the eyes are fixating on a visual scene (salience map). As mentioned earlier, however, perception is not a passive, static phenomenon. During monitoring of a visual scene, we move our eyes constantly, performing large saccades during inspection of the global form of scenes or objects, and smaller saccades when sampling information at a smaller spatial scale [53]. The visual scene may also be processed at different spatial scales to allow attention to be narrowed or enlarged depending on the level of detail required for the task at hand [10]. Despite these constant shifts of the eyes and attention, resulting in discontinuous samples of visual information, our subjective impression is of a stable and seamless visual world. This is not the case for patients with dorsal simultanagnosia, who report intermittent perception: these patients have 'piecemeal perception of the visual environment wherein objects may look fragmented or even appear to vanish from direct view' ([121] p. 448). Dorsal simultanagnosia is observed after bilateral lesions of the occipito-temporo-parietal junction, similar to the locus of unilateral damage that leads to spatial neglect. From this anatomical analogy, it could be considered as a form of bilateral neglect. The idea we develop here is that the intermittent perception in dorsal simultanagnosia results from the overwriting of visual information at the level of the salience map due to impairments of remapping mechanisms. During fixation visual information is processed within the intact ventral visual stream (because the eyes are at this location) but disappears from awareness as a consequence of covert or overt shifts of attention.

We define remapping mechanisms as the processes that operate on later stages of visual processing (between the salience map and the winner-take all array) in order to maintain stable and spatially relevant representations of visual stimuli across shifts of spatial selective attention, and to update their spatial locations across ocular shifts. Thus, for example, remapping mechanisms allow us to maintain a trace of the global structure of a scene to help guide focal sampling at the level of local detail. Similarly, after attention has selected an object in a visual scene, remapping mechanisms allow the representation of this object to be maintained when attention is directed to another part of the visual scene. Under natural viewing conditions attention shifts are accompanied by eye movements. An important point to note is that, in the primary visual cortex, the retinal image is constructed anew at each eye fixation, overwriting all information previously encoded. Without remapping mechanisms to maintain and re-locate neural activity corresponding to these inputs, this general overwriting phenomenon would extend further than the level of primary retinotopic maps. During active exploration, the first eye movement will typically be oriented toward the most strongly represented side of the visual scene within the salience map, generating a new retinal image. After this first saccade, implementation of dynamic remapping mechanisms of visual space is crucial for preventing the sampled scene from being fully overwritten, and for providing the ability to orient the second and subsequent saccades. Indeed, contrary to the primary visual cortex, where no trace from previous ocular fixations is conserved, remapping mechanisms allow the previous representational map to be integrated into the new one at each ocular fixation. Normal integration processes over time and space should prevent the most relevant information sampled in the previous retinal image from being lost or mislocated.

Without such remapping mechanisms, our perception of the world would be manifested as a sequence of discrete 'snapshots', at different spatial scales, and all located directly in front of the viewer; this may be precisely what happens in patients with dorsal simultanagnosia. Even in normal perception, however, inputs that are not strongly represented due to inattention have a high probability of being overwritten, as occurs in the phenomenon of change blindness. The fact that change blindness has been observed not only across saccades but also in conditions of maintained ocular fixation across blinks, flickers or movie cuts [54] seems to confirm that visual representations are remapped after both overt (accompanied by eye movements) and covert (with visual fixation maintained) shifts of attention. According to this change blindness phenomenon in normals, when patients with dorsal simultanagnosia foveate a steadily illuminated LED, during fixation time the LED rapidly disappears from conscious perception [55], corresponding probably to wandering visual attention. This suggests the creation of new retinal images even during ocular fixation, that have to be integrated with previous ones at the level of the parietal cortex. Since it is acknowledged that overt and covert shifts of attention share some common mechanisms (review in Ref. [56]), similar remapping mechanisms might be involved whether the orienting shifts are overt or covert. However, because the higher-level dynamic visual representations in the parietal cortex have been defined as eye-centered ([57], for experimental evidence [58,122]), a more sensible hypothesis would be that in case of covert attention shifts remapping processes

would consist in a refreshment but not a re-location of the different activities of these visual maps.

To summarize, an impairment of the selection processes of the information to be remapped would lead to the disappearance of relevant information from awareness across ocular or attentional shifts, even when this information has been previously attended and consciously represented. Moreover, previously relevant information protected from overwriting must be remapped in spatial coherence with new visual inputs associated with each ocular or attentional shift. An impairment of such refreshment and re-location mechanisms would lead to loss of awareness and/or mislocalisations for objects in the visual world. Because of the local bias associated with right brain damage [10,14,15], an impairment of remapping mechanisms in left neglect is likely to be particularly debilitating because visual information is accumulated in abnormally small samples at each exploration shift.

3.5. Maintaining and updating visual representations: neural correlates

In order to build a stable and coherent representation of the visual world, relevant objects brought sequentially within the focus of attention need to be represented and their relative spatial relations need to be conserved [59]. We postulate that remapping mechanisms carry out these two important functions.

Electrophysiological data have shown that, in oculomotor centres also known to be crucial for the orientation of attention, such as the superior colliculus (SC), the response of neurones can outlast the duration of the stimulus within their receptive field, thus providing a trace to keep the stimulus location in memory. Some of these neurones also discharge if the site of an extinguished visual stimulus is brought into the cell's receptive field by an eye movement. Such *memory-based remapping mechanisms* have been described for cells in the posterior parietal cortex (PPC-[1,60]) and also in the SC ([61]) and the frontal eye field (FEF; [62,63]), which receive connections from the PPC.

Anticipatory remapping mechanisms across saccades have also been described for cells in the PPC (area LIP; [64,65]), whose receptive fields can be shifted rapidly and transiently from the center of fixation toward a target position, just before an eye movement. Direct behavioral correlates of these remapping mechanisms have been shown in humans in a paradigm including a target-directed saccade associated with a letter discrimination task: during the saccadic reaction time, visual attention is selectively and obligatorily oriented to the target position [66,67]. In support of our argument that remapping mechanisms can occur for both overt and covert orienting, Deubel, Schneider and Paprotta [68] have reported that the shift of attention that precedes a saccade [66,67] also occurs when participants maintain fixation and point toward a peripheral target.

3.6. Paradigm measures of spatial remapping: deficits following parietal lesions

Inhibition of return (IOR), a phenomenon characterized by slowed reaction times to targets appearing at recently cued locations, occurs when there is sufficient delay between cue and target events; at shorter SOAs there is facilitation (the conventional cueing effect). Sapir et al. [69] used the phenomenon of IOR to examine the ability of patients with parietal damage to re-map cued locations across saccades. In their task patients were required to make a saccade during the interval between the cue and the target presentation. In normals, after the saccade, IOR occurs for targets appearing at the same absolute spatial location where the cue was presented and not for targets appearing at the location reproducing the retinal location of the cue [70]. In contrast, Sapir et al [69] observed that IOR was present at the retinal location of the cue and not at the absolute spatial location of the cue in patients with posterior parietal damage. In these patients, therefore, IOR arose at the location where it would have occurred without an intermediate saccade, suggesting an impairment in updating the locations of stimuli across saccades.

The double-step paradigm (Fig. 4), was also used to characterize an impairment in the remapping processes necessary to orient the second saccade in the visual field in neglect patients with posterior parietal damage [3,30]. This double-step paradigm has been used to study

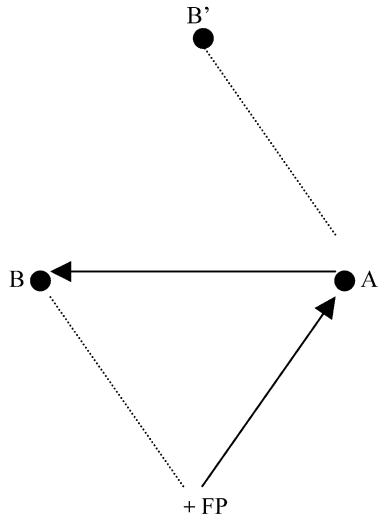

Fig. 4. Example of a double-step stimulus with the two targets A, and B, being flashed successively while the gaze is directed to a central fixation point (FP). When both saccades are performed after all of the targets have disappeared, the motor vector of the second saccade (A → B) is different from the retinal vector of the second target (FP → B or A → B′). However, in this condition, the saccade toward position B is achieved correctly both in humans and in animals. There is thus a need to postulate remapping mechanisms allowing the oculo-motor system to anticipate the new retinal position B by integrating the displacement on the retina produced by the first saccade toward position A. (Redrawn from Ref. [71]).

remapping mechanisms in monkeys [6,60–62]. Two sequentially flashed visual targets (A and B) have to be fixated by two consecutive saccades departing from a central fixation point (FP) toward A, and then from A to B. If the two targets are extinguished during execution of the first saccade, then the second saccade requires the use of extra-retinal information about eye displacement associated with the first saccade (from FP to A) for updating the spatial (retinotopic) representation of the extinguished target B. The generation of a spatially accurate second saccade is thus achieved by remapping mechanisms that combine oculomotor information with retinal information. In the typical double-step saccade paradigm, neurons in the SC [61,62], the FEF [62] and LIP [60,62] exhibit patterns of firing that are associated with the execution of a saccade toward the actual position of the second target, rather than with a saccade predicted by the retinal location of the target (Fig. 4). These results indicate the potential role of these neural structures in remapping space across saccades.

Duhamel et al. [30] had a neglect patient with right frontoparietal damage make two successive saccades to fixate two sequentially flashed targets each of which disappeared before the first saccade. When the patient was asked to make double-step saccades with targets flashed first into the right field and then into the left field, she performed well. When she was asked to do the same task with a target flashed first into the left field and then into the right field she made the first saccade correctly but never acquired the second target, even though this required her to make a saccade in the ipsilesional direction to a stimulus, which according to our rationale should be coded with higher strength in the salience map. The authors concluded that such a deficit therefore cannot be one of retinotopic or spatial coding, nor can it be one of generating a certain direction of saccade. They suggest that this deficit corresponds to a failure of corollary discharge, so that the amplitude and direction of the contralesional saccade is not registered and not compensated correctly at the representational level. We want to highlight here in particular that such a deficit cannot be explained only in the context of the classical attentional hypothesis by an orienting bias due to a pathological gradient of representation of visual space. This result suggests a remapping deficit in right hemisphere patients in which there is an inability to prevent the right target from being overwritten, after a left saccadic shift.

The group study of Heide et al. [3] (see also Ref. [71]) confirmed and extended the demonstration of the crucial role of the PPC in spatial remapping. They used the double-step paradigm to examine patients with unilateral lesions of various structures, including the prefrontal cortex (PFC) anterior to the FEF, the right FEF, the left supplementary motor area (SMA, including the supplementary eye field) and the left and right PPC. Four different types of double-step stimuli were presented. Each pair of targets was located either in the same hemifield (left or right) at horizontal eccentricities of 10°and 5° (conditions R–R and L–L for the two targets presented within the right or the left visual field, respectively), or in different hemifields at 6°/6° (conditions R–L or L–R, depending on whether the right or the left target was presented first). For all groups of patients, a control condition consisted of the same double-step task, but with targets A and B presented long enough to allow the second saccade to be visually guided toward target B, thus obviating the need for spatial remapping. Although patients with right PFC lesions produced large errors in the double-step task, these errors were also significant in the non-remapping control task. Only the patients with damage of the PPC exhibited elevated error rates specific to the double-step paradigm in which remapping was required (retino-spatial dissonance), in at least one of the four types of double-step tested (Fig. 5). Importantly, the study of Heide et al. [3] permits comparison of the deficits in remapping for left and right PPC lesions and, for each group, the performance in conditions where remapping is required between-hemifields versus within the right or the left hemifield only. Based on the mean, absolute-error of final eye position (Fig. 5), the results can be summarised as follows: (1) Both right and left PPC lesions caused errors in double-step saccades that involved crossing the midline (L–R and R–L *between-hemifields* conditions); and (2) Patients with right PPC lesions (all of whom had left neglect initially by clinical assessment) showed significant errors under conditions in which double-step saccades had to be performed entirely within the left visual field (the L–L *within-hemifield* condition). These results reveal an asymmetrical pattern of errors between right and left PPC lesions, the impairment being particularly severe for neglect patients with right PPC lesions.

In accordance with the findings of Duhamel et al [30] and Heide et al [3] concluded that *both* patients with right or left PPC lesions showed an elevated percentage of erroneous (aborted or dysmetric) second saccades, especially when the first saccade was directed toward the contralesional field. However, the asymmetry of occurrence of severe and chronic visual neglect for right versus left hemisphere pathology [16,22] suggests a possible interpretation of the results of Heide et al. [3] that highlights the asymmetrical pattern of errors that follows right and left PPC lesions. The pattern of results indeed reveals a specific impairment of patients with right parietal lesions (with neglect) that prevents them from executing a correct second saccade after left orienting. We believe this result is important because it accounts for the behavior of neglect patients in lateralised cueing tasks, as we outline below.

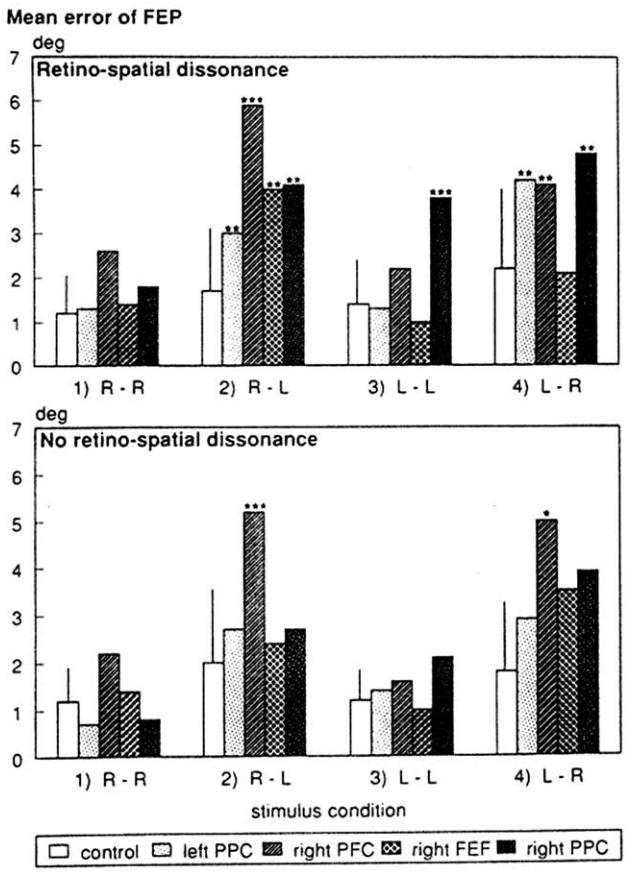

Fig. 5. Results of the double-step task in patients with left or right parietal lesions (PPC: Posterior Parietal Cortex) compared to patients right frontal (FEF: Frontal Eye Field or PFC: Pre-Frontal Cortex) lesion and Controls. This figure is reproduced from Ref. [3] with permission. It represents the mean absolute error of final eye positions (FEP) after double-step trials, plotted separately for the four different stimulus conditions of the study (R–R: centripetal double-step within the right hemifield; L–L: centripetal double-step within the left hemifield; R-L: double-step between hemifields starting with target on the right; L–R: double-step between hemifields starting with target on the left). Double-steps with retino-spatial dissonance (upper panel) require remapping processes whereas double-step with no retino-spatial dissonance do not need remapping processes. Significant errors relative to control performance are indicated by stars. Note that patients with parietal lesions exhibit errors specific to double-step with retino-spatial dissonance (contrary to patients with frontal lesion): patients with right PPC lesions are impaired for both between-hemifield double-steps and also for the L–L stimulus, whereas patients with right PPC lesions are only impaired for between hemifields stimuli. Standard deviations of the control group are indicated by vertical lines. Results of left prefrontal cortex patients are not shown in this diagram, because they were not significantly different from the control group.

4. How remapping impairments can explain what attentional accounts alone cannot

4.1. The contribution of remapping mechanisms to lateralised cueing tasks

The graded representation of the visual environment causes a bias that leads patients with parietal damage to start their exploration from the ipsilesional side. However, this is just one component of the syndrome of spatial neglect. Most tests of neglect, both clinical and experimental, involve two or more orienting shifts. Remapping deficits explain why patients with left neglect after right hemisphere damage may also exhibit a paradoxical *right* (ipsilesional) neglect when cued toward the left side. For example, they may bisect horizontal lines to the *left* of centre after being cued to attend to the left end [72–74], or they may fail to report visual stimuli on the *right* side in cancellation or extinction tasks [28,75]. The effect of cueing on line bisection or cancellation concerns the eye movement performed after a first saccade is directed toward the left extremity of the line or the testing sheet, respectively. Depending on the severity of neglect, it can be predicted that, after the first saccade toward the cued extremity of the line, only a relatively steep gradient would tend to attract the patient's gaze back toward the right ipsilesional side. Accordingly, the effect of cueing has been shown to follow two different patterns: either a slightly improved pattern of left neglect in the most severe patients, or a reversed (ipsilesional) neglect pattern in milder cases [73,74].

It has been proposed that the occurrence of transient ipsilesional neglect reflects in part the overall reduction in attentional capacity that is common in such patients [28]. But such an account does not readily explain the reversal of spatial errors on line bisection, where there is no time constraint and only a single object (the line) is presented at once. Moreover, patients with a local bias and reduced attentional capacity without neglect [76] do not show such pathological patterns of performance in line bisection (personal observation; see also Ref. [77]).

Ipsilesional neglect may also be explained by impaired 'disengagement' of attention from visual cues, as revealed by the Posner spatial-cueing task [78]. In this task participants keep their eyes on a central FP and respond as fast as possible to targets presented in the right or the left visual field. Targets can be preceded by cues presented either on the same side (congruent trials) or on the opposite side (incongruent trials). An increased cost in responding to the target presented on the opposite side to the cue (incongruent trials) is attributed to a covert orienting shift toward the cue [78]. In parietal patients, targets on the contralesional side are detected more slowly than those on the ipsilesional side, consistent with a pathological attentional gradient. A deficit in 'disengaging' attention from a right cue following right parietal damage has been highlighted as a specific additional characteristic of neglect [79–81,123]. Note, however, in Fig. 6 that the disengage cost (about 100 ms) observed between the other condition of spatial incongruency (when the cue appears on the left side and the target on the right side) and the congruent condition with target presented in the contralesional field is largely superior to the difference obtained between contralesional and ipsilesional orienting in congruent conditions (about 20 ms only—[79]). Indeed, this means that for these patients the need to generate sequential orienting toward the left then

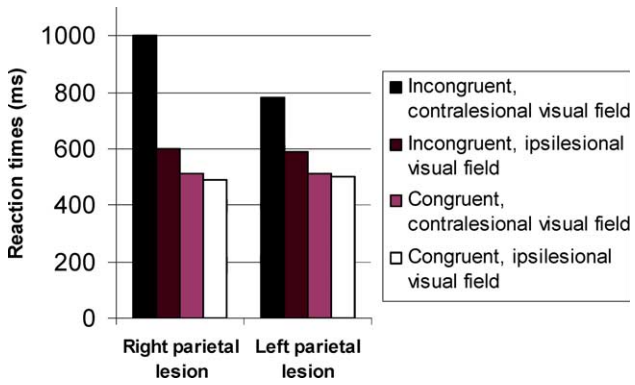

Fig. 6. Histogram redrawn from the results obtained by Posner et al. [79] in first group of six patients with right parietal lesions and a second group of seven patients with left parietal lesions during the typical Posner paradigm (see text) with 500 ms cue-target interval. As a result of the lesion, there is a significant advantage for congruent trials overall, this implies that the cue is effective in attracting attention to either the ipsilesional or the contralesional visual field. The two conditions of spatial incongruency yields longer reaction times to the target, especially when the target is presented in the contralesional visual field (and especially for right parietal lesions) but also when the target is presented in the ipsilesional visual field.

the right side appears far more problematic than the need to orient a saccade in a direction opposite to their pathological rightward attentional gradient (analogous to the deficit in saccade generation observed for the patient of Ref. [30]). In congruent trials, attention is covertly oriented toward the side where the target is presented, eliminating the requirement for such double-step orienting processes. This superiority of the disengage costs with respect to the bias resulting from the pathological attentional gradient in patients has been explained by distinguishing exogeneous and endogeneous attention [81]. We rather suggest that remapping mechanisms, crucial for double-step orienting, can account for a general disengage cost in neglect patients (additional to the attentional pathological gradient) and further for ipsilesional neglect patterns after left cueing.

Note that the effect of cueing on the performance of neglect patients reported above was very similar whether the patient was instructed to overtly orient toward the cue (e.g. in line bisection) or not (e.g. in Posner tasks): both right and left cue were effective in reducing attention for the opposite side, which cannot be explained in the context of the attentional gradient hypothesis exclusively.

4.2. Hemispheric asymmetries in the representation of space

Experimental evidence that the human right hemisphere can direct attention in both left and right visual fields, whereas the left hemisphere can direct attention only in the right visual field, is postulated to account for the asymmetry of occurrence of severe and chronic visual neglect between the two hemispheres [16,22]. This hypothesis is consistent with observations of the behavior of split-brain patients [16,23,24]. Mangun et al. [24] showed that the right hemisphere was influenced by cues presented either in the right or the left visual field, whereas the left hemisphere is constantly oriented toward the right visual field. Other evidence comes from brain imaging studies where tasks involving spatial attention in the right or left visual field have revealed contralateral activation of the SPL, but also specific activation of the right IPL [25–27].

In this context, we suggest a new interpretation of the results of Heide et al. [3]. In their double-step saccade study, patients with left parietal lesions showed normal remapping performance within-hemifields, whereas patients with right parietal lesions exhibited a profound impairment in the condition where targets A and B were both presented *within* the left visual field. In the conditions of within-hemifield remapping (conditions R–R or L–L), the two visual targets (A and B) are both represented in the same (contralateral) SPL, but in addition the two right targets of the R–R condition may also be represented in the right IPL. After lesions of the left PPC, the spared hemisphere would then be able to ensure remapping within both hemifields (i.e. support accurate double-step saccades within both L–L and R–R conditions after left parietal damage), whereas after lesions of the right PPC the spared left PPC would only be able to remap locations within the right hemifield. To fit with our reading of the data of Heide et al. [3], however, one has to postulate that the bilateral representation of space within the right IPL is not able to remap between hemifields.

4.3. Remapping representations across cerebral hemispheres

The results of the cueing experiments reviewed above, as well as the Heide et al. [3] study, underlines the poor performance of neglect patients under conditions in which they have to shift attention covertly or overtly *between* hemifields. It is therefore important to consider the role of callosal transfer in spatial remapping mechanisms. The remapping of relevant information could be implemented by the transfer of neuronal activity within the salience map, whose representation we have speculated consists of the two symmetrical representations of contralateral space contained within the right and left SPL. Anderson and Van Essen [57] proposed that ocular shifts are implemented by shifts of neuronal activity *across cortex*. This has been confirmed by imaging studies highlighting shifts of activation across the parietal cortex [58,82]. Crucially, each cortical hemisphere contains the neural circuitry for maintaining and updating its own salience map of the contralateral visual field. For the within-hemifield condition of the double-step task, both targets (A and B) are initially represented in the same hemisphere and within the same salience map. However, in the between-hemifields condition, each of the two visual targets is initially represented in a different hemisphere. Spatial remapping therefore requires a transfer of information between hemispheres.

Several studies have demonstrated the importance of callosal connections for the remapping and integration of spatial information. Ingle [83] had callosotomy patients point toward memorised targets after passive turns of the body. Of three patients with transection of the anterior part of the callosum, one showed a normal pattern, whereas the two others showed impairments in between—hemifield remapping of spatial location. Ingle [83] suggested that the callosotomy of the two latter patients was more extended and probably disconnected homologous parietal areas. Split-brain studies have also underlined the role of the corpus callosum in the integration of visual information across hemifields [23,84,85]. Other studies have shown that spatial neglect may occur after callosotomy. A right-handed split-brain patient (with an additional left temporo-occipital lesion), demonstrated consistent left neglect either when using his right hand on various tasks including line bisection, copy of half-pictures, or when naming chimeric pictures and reading. A specific posterior callosotomy disconnecting the two parietal cortices (parietal leucotomy: [86]) alone has been shown to produce neglect in monkeys, as predicted by the studies of Ingle [83] in humans. It has also been observed that neglect after unilateral cortical ablations in non-human animals becomes significantly worse [87], or reappears after recovery [88], as a consequence of transection of the corpus callosum. Together, these results demonstrate the importance of transfer of neural activity via the corpus callosum between the two parietal lobes for the integration and remapping of visual space across hemifields.

4.4. A new model of spatial remapping impairments in neglect

We argue that remapping deficits are not specific to a particular location of space, nor simply to an eye-movement direction. From the results of Ref. [3] and the mechanisms postulated above, we propose the following detailed model of remapping deficits:

- With damage of the right PPC, including the IPL (patients with left spatial neglect), after a leftward saccade the whole salience map is overwritten, whereas after a rightward saccade only the representation of the previous left visual field in the salience map is overwritten.
- With damage of the left PPC, after any saccade directed toward the left or right visual field, only the representation of the visual field located on the side opposite the direction of the saccade from the previous fixation position is overwritten.

Fig. 7 illustrates this pattern of deficit after right and left unilateral parietal damage. The objects represented in the visual scene in Fig. 7 can also represent different elements of the same object, when it is the current focus of attention.

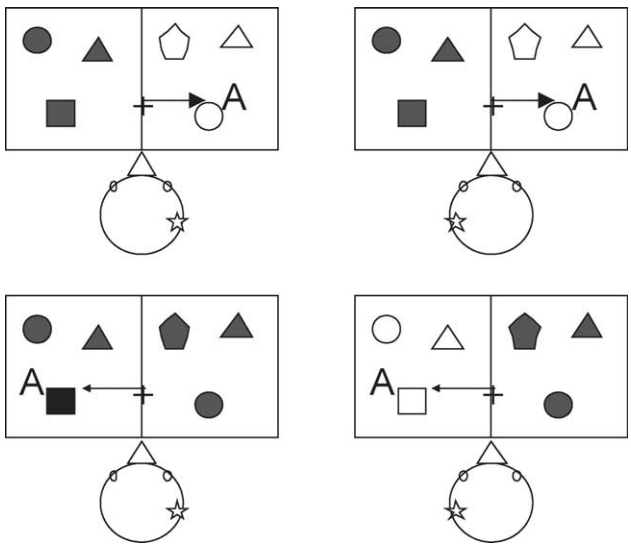

Fig. 7. Visual space overwritten depending on saccade direction (arrow from fixation cross to object A) and lesion side (star). Basic model of the remapping impairments in case of parietal lesion based upon the results of [3] (Fig. 5). The filled shapes represent objects that will not be recombined correctly with point A in the visual maps of the parietal cortex, or that will be completely overwritten in these maps. The presence of all objects is possibly detected when the eyes are on fixation. As soon as the first saccade is oriented to point A, the objects in black will be misrepresented or will disappear from the visual representation of the parietal cortex, and thus from visual awareness.

For schematisation, the direction of gaze is assumed to be initially straight-ahead. But note that because the deficit is retinotopic at the basic level, the region of visual space that is impaired always changes at each eye movement and depends on the direction of the next eye movement to be produced and remapped (see Fig. 8).

Based on this model, the same overwriting of left space is observed in right and left brain-damaged patients after a rightward saccade or shift of attention. Further rightward saccades are therefore likely to occur, thus increasing the size of the region on the left that will be overwritten (see Fig. 8a). Note, however, that when the exploration reaches the right extremity of the visual scene, leftward return saccades are likely to occur, and from the model these have different effects in patients with right versus left hemispheric lesions (respectively, RH and LH). Leftward orienting makes objects of the left visual field reappear and be re-processed in LH patients, who would therefore complete their exploration to the left. But the model predicts symmetrical, progressive overwriting during leftward exploration of the visual scene in LH patients. This suggests that they would be able to attend to the whole visual scene by scanning from left to right or from right to left, but during such scanning a newly explored object cannot be compared and linked in the representation of the salience map with previously explored objects (see Fig. 8a and b). By contrast, in RH patients, leftward orienting produces a general loss of awareness for the location of the objects of the visual scene,

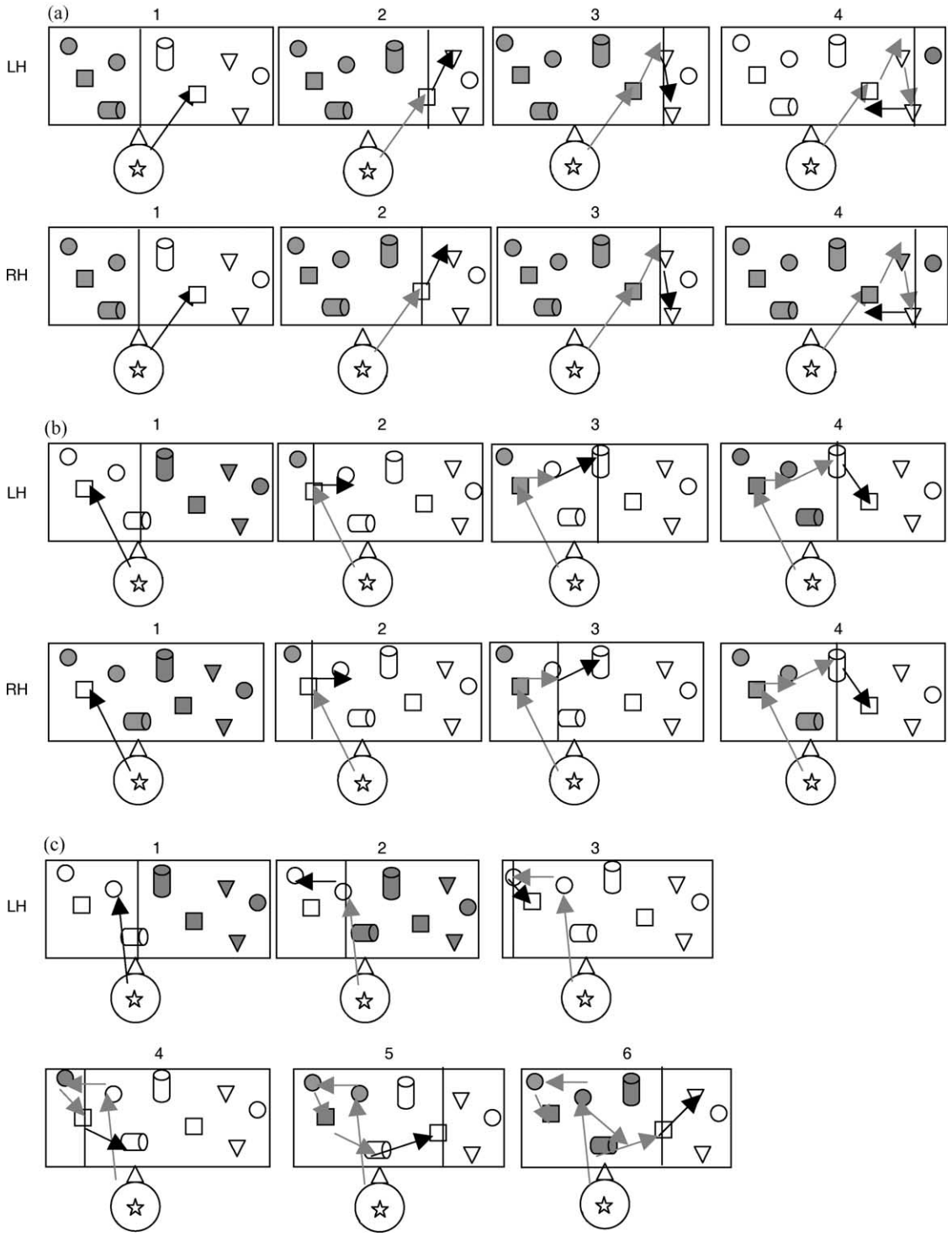

Fig. 8. See legends in the text. New saccades are illustrated by black arrows, previous ones remain on the figure with a grey colour in order to show the ocular track. For simplification, the gaze is supposed to be central for patients with left or right parietal lesions (respectively LH and RH) at the beginning of the ocular exploration of the visual scene. (a) I*llustration of the predicted part of space that would be overwritten on the salience map at each step of a multi-saccadic ocular exploration of a visual scene starting with a rightward saccade*. Note that at the end of the progressive exploration of the right space, our model predicts that right brain-damaged patients will stop, whereas left brain-damaged patients may explore the left space. (b) *Illustration of the predicted part of space that would be overwritten on the salience map at each step of a multi-saccadic ocular exploration of a visual scene starting with a leftward saccade.* Note that for right brain-damaged patients, after an initial leftward saccade, the whole salience map is overwritten, which predicts that the patient may stop the scanning straight away. For left brain-damaged patients, the case of a following rightward saccade is illustrated. However, left brain-damaged patients may explore further to the left space, before exploring toward the right space. This is illustrated in c. Note that, in this latter case, when patients a with left parietal lesion begin to explore toward the right space, our model predicts that the left space is progressively overwritten.

and also for the identity and number of the objects explored before if these have not been encoded in a more enduring store. This loss of visual coherence (block-out) after leftward orienting is likely to result in patients discontinuing their exploration of the visual scene with no feeling of incompleteness.

The loss of awareness following leftward orienting in right parietal patients might also explain the dramatic effects of left cueing in patients with left neglect. Fig. 8b is an illustration of the patterns of impairment after leftward saccades in RH and LH patients, assuming, for the schematisation, that both types of patient exhibit the same scan path of the visual scene. This is highly improbable, considering that they exhibit different patterns of spatial impairment. LH patients may explore further the left space, before exploring the right space (see Fig. 8c). RH patients may either do the task around position A and stop at this step without exploring the right space (reversed pattern of neglect after left cueing), or be attracted back to the right space toward a more ipsilesional position by their steep attentional gradient. In this latter case step 1 would be directly followed by step 4 in Fig. 8b, without exploration of the intermediate items located centrally in the visual scene (still resulting in an improved pattern of left neglect after left cueing).

4.5. Spatial mislocalisations in neglect are due to dysfunctional remapping mechanisms

We suggested that remapping involves two separate processes. The first involves selecting information from the previous representation that must be retained in the next one. The physiological basis of this selection is likely to be a probabilistic threshold applied to the amount of activity on the salience map above which the element is preserved in the salience map and therefore protected from overwriting. An impairment in this process would lead to a truncated representation of the visual scene, and hence to a complete loss of awareness for information not maintained within the salience map across saccades. The second process involves re-positioning selected information in the map in a way that takes into account changes in the retinal image due to changes in eye position. This might be achieved by transferring neuronal activation across the cortex. If this aspect of the remapping process is impaired, the different elements sampled in the visual scene may be relocated and combined in a manner that distorts the underlying representation. In this case, neglect patients should show errors of mislocalisation and distortion under appropriate conditions.

In fact there have been numerous demonstrations of mislocalisation errors in spatial neglect. Findings suggestive of space compression [89] and size distortion [90] have been reported in left neglect patients. As mentioned by Milner [90], it is notable that size distortion occurs under free viewing conditions, ‘in which an extended object is presumably constructed from a sequence of snapshots separated by saccadic eye movements’ (p. 87). Note that size distortion appears when the two items to compare are horizontally adjacent and presented in the right and left visual fields, and are thus compared by successive horizontal saccades. It does not appear when the two items are presented one above the other. The same pattern is observed in the covert situation of extinction [91]. These ideas are in accordance with our hypothesis of remapping mechanisms across horizontal shifts being the basis of the representational deficits of neglect. A compressed visual representation could result from a constant rightward bias during the remapping process of relocating relevant information of the previous ocular fixation on the salience map. This constant bias might for example be produced by a gain modification due to the lesion. It is apparent when patients make errors in locating left targets during brief target presentations. Di Pellegrino and De Renzi [33] used an experimental design consisting of three boxes on either side of fixation, each corresponding to a location at which a peripheral target could appear. The three boxes were located at increasing eccentricities in each visual field (see Fig. 9). A right hemisphere patient with extinction was asked to report the occurrence of brief targets on the right and/or on the left of the FP, as in the classical extinction paradigm, but also to indicate in which of the boxes the stimuli appeared. The right stimuli were always reported and correctly located. On bilateral presentation, the patient reliably detected the right stimulus, but always extinguished targets on the left. For single left trials, the stimulus was always perceived but was consistently mislocalised to the box on the immediate right. Such metric problems concerning stimuli in the left visual field are likely to arise from a constant bias in the remapping of visual information, after an overt or covert orientation toward the left (contralesional) visual field. Referring to the model presented in Fig. 7, in this patient with right temporo-parietal damage [33], remapping of the ipsilesional (right) space would not be expected to be affected by ipsidirectional (rightward) orienting, and so isolated right targets are localised correctly. But remapping of the contralesional (left) space should be affected by the same ipsidirectional (rightward) orienting, so that elements of the left space disappear from the salience map. In bilateral trials, the gradient of representation in the salience map causes the patient to orient first toward the right stimulus, (thus overwriting the left stimulus in the salience map), and to abort the second orienting response to the left space altogether. This would explain why the stimulus on the left is not reported in cases of bilateral presentations (i.e., the classical extinction effect). As predicted by our model, a contradirectional (leftward) orienting shift would lead to the general orienting problem specific to right brain-damaged patients. This first leftward orienting would occur for left single target presentations. When the left target disappears, attention is likely to leave the box where the target was presented,

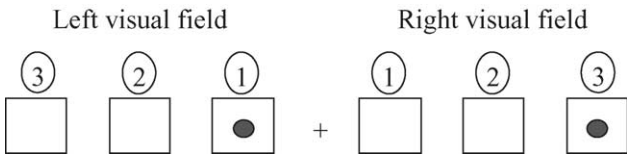

Fig. 9. The three–box extinction display (Adapted from Ref. [33]). On each trial, either a single target was displayed in one of the left or right boxes, or two targets were presented simultaneously on both sides of fixation, at equal or different eccentricity (e.g. as illustrated). The patient first had to report the number of targets (one or two) and then to indicate verbally their location by using conventional numbers attributed to the boxes as illustrated (1, 2 or 3).

the attentional gradient of the salience map eliciting a rightward return shift. The whole salience map might then be remapped with a constant rightward bias in such a patient. Consequently, targets appearing in left space, toward which the patient first oriented, would be mislocated to the next box on the right.

Remapping mechanisms are crucial for drawing and copying tasks, where information needs to be transferred from one place to another with multiple fixations and re-fixations on the details of the drawing. This explains why drawing and copying have proved to be reliable tests of neglect, where constructional apraxia and allesthesia patterns are often observed. The drawing copies made by constructional apraxic patients (Fig. 10: Complex Figure of Rey) are extremely disorganised, and may result from defective remapping of the spatial relations between the different elements sampled. Copying involves numerous ocular movements between the different elements of the drawing, and between the model and the ongoing copy, all of which require spatial remapping. Halligan et al. [32] described an interesting pattern of mislocalisation ('allochiria/allesthesia') in the copies of a butterfly made by a RH patient with left neglect (Fig. 11). The patient transposed the left-sided details of the figure to the right, consistent with the impairment of spatial remapping postulated here. Halligan et al. [32] suggested that "transpositioning may be viewed as a process whereby partially analyzed left-sided information is either placed in different spatial loci with the intact portion of the visual buffer or incorrectly transcribed from the buffer" (p. 131). They added: "questions remain as to the neuronal locus of transposition effects". We suggest that such transpositions occur at the level of the salience map due to impaired remapping mechanisms. Halligan et al.

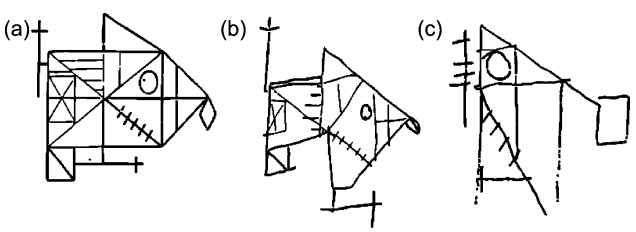

Fig. 10. Examples of drawing copies of Rey's figure made by two patients with constructional apraxia following a posterior parietal lesion (from Ref. [71]).

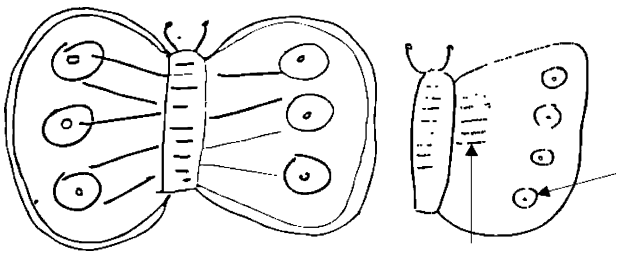

Fig. 11. Example of a pattern of mislocalisation (allochiria/allesthesia) in the copies of a butterfly made by a right hemisphere patient with left neglect described by Halligan et al. [32]. In his copy, the patient omits the left wing but add one line and one circle (elements probably pertaining to the left wing) to the right wing.

[32] also postulated that "transpositions and rotations of left objects on the drawing were used to compensate for some lateralised spatial impairments involved in visual neglect" (p. 130). As mentioned before, this lateral direction of the deficits of neglect is probably due to the disruption of interhemispheric transfer. Remapping can be impaired such that re-location of left elements is biased toward the right (ipsilesional) side, i.e. confined within the intact left hemisphere. Similarly, on the task of drawing a clock from memory, some patients with visual neglect insert only those numbers appropriate to the right side of the clockface, whereas others transpose left-sided numbers onto the right side [12]. These two patterns of impairment (omission versus transposition) may correspond to situations in which elements in the left visual field are overwritten after a rightward shift of attention, or in which elements are relocated incorrectly on the salience map after being attended.

4.6. General restatement of the model and predictions

The salience map codes the strength or 'salience' of each visual stimulus, and determines in which order they will be selected for further processing. It determines which visual information will be represented at the level of conscious perception. Imaging studies have implicated the parietal cortex as a crucial structure for visual awareness [51,52], by showing correlations between neural activity changes in parietal cortex with changes in perceptual awareness. The salience map of the parietal cortex is retinotopic/oculocentric [43], which explains why neglect can occur at the scale of one visual object as well as at the scale of an entire visual scene (object-based and space-based neglect, respectively; see [92]). Similarly, dorsal simultanagnosia can be observed at various spatial scales, which could correspond to different sizes of the focus of attention. In patients with dorsal simultanagnosia, disappearance of stationary objects from conscious perception can occur even in central vision, i.e. when the stimulus is fixated [55]. In this case, we propose that the object is processed by the primary visual areas but disappears from conscious perception after a delay, allowing the patient to covertly shift attention

and thus requiring remapping of the visual scene. We note that this level of deficit may still allow for implicit perception of stimuli that elude awareness (review in Ref. [9]), since objects may continue to be represented within the 'what' (ventral) stream (review in Ref. [93]), but do not reach consciousness unless they are represented and remapped within the parietal cortex.

In order to account for the behavioral deficits of neglect, we have to postulate not only a pathological gradient of representation of the visual world at the level of the salience map, but also a remapping deficit of the information coded on the salience map. Two main reasons have been given for the necessary involvement of a remapping deficit. They are summarized below and illustrated with supplementary examples.

First, the hypothesis of a lack or a weakness of representation of the left visual field on the salience map would predict that the left visual space never reaches awareness. It would not account for the fact that left visual information is sometimes consciously perceived and can be represented even more strongly than the right space at the level of the 'winner-take-all' array [38], e.g. in cueing paradigms. It would not account also for the fact that some visual information frequently disappears from awareness after being consciously perceived. The disappearance of the conscious percept of a target in central vision described in simultanagnosia [55] is one pertinent example. In the copying test of Gainotti et al. [92], in which multiple line drawings are presented for copy, neglect patients who draw only the right side of an item on the extreme right may initially perceive and identify more than one item among the five objects presented on the sheet. But when their attention is narrowed in order to copy the details of the item on the extreme right, relevant information to the left would not be remapped [10,94]. Patients would therefore fail to copy the left side of this item, and the remaining items, and stop with the feeling of having finished the copying task. (Note that further exploration may be guided by cognitive exploration strategies based on higher-level memory of the number of items initially evaluated in the visual scene; cf below). Existence of such attentional 'zoom lens' correlated with loss of awareness in neglect patients is strongly suggested by the findings of Ishiai et al. [11]; see also Refs. [10,94,124]. In that study, neglect patients were initially presented with complete pictures of flowers (petals surrounding a central circle) or truncated pictures of flowers (with the right- or left-sided petals missing). When asked to judge whether these pictures were complete or incomplete, neglect patients could discriminate well between the complete and incomplete drawings, presumably because their attention was allocated to the global form of the flowers depicted. The results suggest that inputs from the left side of the pictures reached visual awareness, even though they were probably only weakly represented in the salience map. Strikingly, when the patients were asked to copy the same (complete) pictures, they showed the classical omission or distortion of the left part of the model, presumably because copying requires a narrow attentional focus and many saccades and attentional shifts between the different elements of the drawing in order to link them together. We postulate that these various shifts are not compensated by remapping mechanisms, leading to distorsion or loss of visual awareness for the elements encoded within the left visual field (in the right hemisphere). Such contralesional neglect is far less likely to occur for patients with damage of the LH, who we suggest are only impaired for shifts between hemifields. Crucially, when the patients of Ishiai et al. inspected their finished copies, none of them noticed their omissions, instead judging their copies as complete. When they were asked to inspect their copies after a delay, however, they noticed the left-sided omissions. This study highlights that neglect is not perfectly defined by a static (permanent) weakness of representation of the left space; in a dynamical view of space representation, visual information from the left space can be represented and then disappear due to a failure of remapping.

The second aspect of neglect behavior that cannot readily be explained by the gradient hypothesis is the mislocalisation of visual stimuli. Husain et al. [34] showed that during visual search, a right-parietal neglect patient re-explored and re-considered previously cancelled targets as if they were new targets. This 'amnesic' aspect of exploration during visual search was called 're-visiting behavior' by Husain et al. [34]. The patient's re-visiting behavior was revealed in a series of experiments using variants of the classical cancellation task, in which 'invisible' marks were made using carbon backing paper or a computer display and mouse [34,95,96]. These displays prevented the patient from seeing the marks he had previously made during target cancellation. With these modifications the patient tended to re-select and re-mark previously visited object locations (predominantly on the ipsilesional side) after having explored further toward the neglected side (this latter element being important to rule out mere motor perseveration). This re-visiting pattern was observed even though the patient was explicitly asked to mark each target only once. The authors then created a variant cancellation task where all the elements presented on the screen/sheet were different. They used drawings of different objects and animals. The task was again to mark all the objects presented on the screen/sheet only once. In this task providing the patient an additional memory cue to each visited location (verbal encoding or memory of an already visited perceptual image), the results showed a drastic decrease of re-marking/re-clicking behavior. This last experiment ruled out the presence of a general working memory deficit in this patient. The authors concluded that neglect patients exhibit a spatial working memory (SWM) deficit which makes them consider previously explored target locations as new ones. A more direct test of this SWM hypothesis [97] confirmed this conclusion. Furthermore, Pisella et al. [97] demonstrated the specificity of the SWM impairment for neglect due to parietal damage. This leads to

focus on the remapping processes, that can be considered as the elementary stages of processing for SWM, specifically implemented within the parietal cortex (for further distinction between remapping and working memory, see below). Although an attentional-gradient hypothesis would predict repeated attraction toward right-sided stimuli, it cannot explain why a patient would consider previously located targets as new stimuli. After marking the stimuli present at the extreme right, the patient is likely to re-orient toward the left. This leftward orienting would thus perturb the representation of all stimuli encoded within the salience map during previous ocular fixations. Lack of awareness or mislocalisation of the previously explored locations is thus likely to occur. It can therefore be postulated that the visited location of the ipsilesional stimuli would be not only re-visited but also re-marked as a result of the remapping deficit of leftward orienting in neglect. In this sense, it could be predicted that the occurrence of re-marking errors will be correlated with the previous occurrence of a leftward saccade.

We propose that the revisiting behavior described by Husain et al. [34] in cancellation tasks is reminiscent of remapping more than a SWM deficit per se. This behavior reveals a deficit of building a coherent representation of visual space *across exploratory saccades* [96], at the level of the salience map. Future studies are needed to establish the putative role of the remapping processes in SWM and whether remapping and SWM correspond to dissociated levels. Remapping mechanisms operate at a fraction of a second [1], whereas SWM is classically studied at the timing scale of seconds or minutes ([99,100]; see also Refs. [101,102]). Note that cancellation performances can be improved by cognitive exploration strategies or when the objects are presented on the sheet with a columnar organization, which facilitates vertical exploration and minimises the number of horizontal eye-movements [103]. By allowing the neglect patient to take cognitive landmarks in space, structured stimulus arrays and structured patterns of exploration presumably provide the possibility for compensation of the remapping deficit via higher-level guidance and memory of the exploratory track. This suggests that the level of the salience map could be distinguished from more cognitive levels of SWM. The salience map would correspond to a visuo-spatial buffer where the remapping mechanisms control the exploration pattern and the construction of the visual image. The level of SWM could correspond to more symbolic/verbal encoding of the spatial information for stable, longer-term storage and potential exploration strategies. More investigations are needed to determine whether the former and/or the latter level of spatial representation and storage is impaired in neglect patients with parietal and/or frontal damage.

Transposition errors have also been described in imagery tasks [12,32,104] during the process of drawing a stored scene. The typical task requires the patient to report a novel or familiar scene. Strikingly, patients show a spared ability to construct novel mental images, thus implying an ability to establish accurate spatial locations between objects. The patient of Halligan et al. [32] was able to construct an accurate visual image from verbal description of a novel scene consisting of a room where 16 objects were positioned. Her verbal descriptions of the layout of the room demonstrated intact verbal comprehension and working memory, but also SWM efficient enough to construct an accurate spatial representation of the room. By contrast, her drawing of the room showed substantial mislocalisations. It seems therefore that basic processes involved in drawing or copying tasks are specifically impaired in visuo-spatial neglect, instead of more general/high-level processes. We think that these piecemeal tasks crucially involve saccades or attentional shifts requiring accurate remapping mechanisms. Note however that recent studies have shown contradictory results suggesting that neglect patients have difficulty in generating mental visuo-spatial representations from verbal descriptions [105]. By comparison with Halligan et al. [32], the verbal material used by the authors corresponded to more specific between-object spatial relationships such as “the carrot is in front of the orange, the orange is to the left of the banana…”. With this type of verbal information, the patient may need to progressively construct the image by using active attentional shifts and remapping processes.

To conclude, our model of a remapping deficit at the level of the salience map, in addition to a local bias and an orienting bias, explains a range of neglect symptoms. To validate our model, direct evidence for such remapping impairment in patients with documented clinical symptoms of neglect need to be added to the studies in Refs. [3,30,69]. Crucially, the model also generates several testable predictions, which we outline below.

The hemispheric asymmetry for attention assumed by the model predicts a different pattern of extinction for right and left parietal patients. Recall the results in Ref. [3]; patients with LH parietal damage were able to achieve normal double-step orienting within the left (ipsilesional) hemifield as well as within the right (contralateral) hemifield, suggesting that the RH is able to remap stimuli from both sides of space. By contrast, patients with RH parietal damage were impaired in performing double-step orienting within the contralesional (left) visual field. Both left and right brain-damaged patients were impaired in between—hemifield remapping conditions. If we assume that remapping deficits contribute to extinction behavior, the pattern of extinction in LH and RH patients should match this pattern of results. Following the finding of Ro et al. [48] of a systematic ipsilesional bias of the first saccade, we would predict that patients with right parietal lesions should show extinction for the left target (opposite to the systematic rightward orienting bias), both when the two targets are presented in the left visual field, and when they are presented on either side of fixation. In contrast, patients with left parietal lesions should show extinction for the right

target exclusively when the two targets are presented on either side of the central FP; within-hemifield detection of single or double targets should be normal in these patients due to their spared right parietal lobe.

A similar prediction arises from the model concerning the integration of the left and right parts of objects. Let us first consider the pattern of unimpaired double-step processes in patients with left or right parietal damage (Fig. 5). The schemas suggest that left brain-damaged patients can correctly explore and construct a visual representation of the right (contralesional) space, as well as the left (ipsilesional) space. But, for both LH and RH patients, our model postulates that if the first saccade is directed to the left, then the right visual field (RVF) is overwritten from the salience map, and if it is directed to the right, then the left visual field (LVF) is overwritten. This pattern of impairment of between—hemifield remapping is assumed to be the same for patients with right or left damage, based on the findings in Ref. [3]. All patients with unilateral parietal damage may therefore have difficulty in comparing the left and right parts of single objects on the same salience map, and thus to integrate the two parts of one object explored sequentially into one complete visual image. It can therefore be predicted that both types of patient should show a systematic bias on the greyscales task, in which patients must compare the brightness of two vertically aligned rectangles that are darker on opposite ends [106,107], or in the judgement of chimeric faces, which implies the comparison of subtle details (like the shape of the mouth or of the hairs) between the right and the left part of the face [73,74]. In these two tasks, right brain-damaged patients tend to base their judgement on the right part of the object [73,74,106,107], and left brain-damaged patients on its left part. This still needs to be tested systematically, but our own observations suggest that both types of patients do not detect, or are slow to detect, that the faces presented to them are chimeric, whereas this fact is immediately striking for healthy subjects. Similarly, in the greyscales task, the symmetrical distribution of the grey levels within and between the two rectangles is very rarely reported by either type of patients. We have recently shown that both RH and LH patients show an ipilesional bias on the greyscale task [125].

Because our model applies to both overt and covert shifts of attention, it is likely to explain findings for mental images and their exploration. Further electrophysiological studies should focus on possible remapping processes across covert shifts of attention. Note also that, even if the idea of remapping developed here concerns only visual neglect, the acknowledged dominance of vision on other modalities suggests that if deficit of remapping concerns visual imagery and visual attention it could lead to more general sensory neglect. The cross-modal aspect of attention can be explained by the existence of amodal processes, or, by the preponderant use of visual attention in many tasks and many sensory modalities. The latter would suggest that remapping mechanisms are not restricted to the visual modality, which is an interesting issue for future research. Note that in support of this hypothesis recent results have highlighted that the oculo-centric coding of locations is largely used in non-visual sensory modalities [108–111].

Finally, even though our model postulates a retinotopic deficit at the basic level, we recognise that neglect can be expressed in different reference frames. It is also conceivable that proprioceptive information from head and body may influence the representation of the salience map, or the remapping mechanisms, or both. Indeed, an effect of real or simulated head or body turns on the severity of neglect performance has been shown in the literature [112,113]. An effect of body turns on visual signal detection has also been shown in normals [126]. Possibly by its plastic modification of coding and integration of proprioceptive and visual information, prism adaptation has been shown to improve neglect significantly and durably [13,114–117]. It would be interesting to examine the potential effect of prismatic adaptation on the double-step saccade task, or on the equivalent IOR task used by Sapir et al. [69]. One may postulate that prism adaptation influences the distribution of the salience map between the two cortical hemispheres and/or the gain of the remapping mechanisms. However, this important issue needs further investigation, especially with regard to the processes and the neural substrates of prism adaptation [98,118].

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