Spatial remapping of the visual world across saccades

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Received 3 April 2007; accepted 3 May 2007

Recent research has identified neurons in the visual system that remap their receptive fields before a saccade. The activity of these neurons may signal a prediction of postsaccadic visual input, derived from an efference copy of saccadic motor output. Such a prediction is often thought to underlie our perception of a stable visual world, by compensating for the shifts in retinal image that accompany each eye movement. Here we review the evidence, and conclude that prediction does *not* in fact play a significant role in maintaining visual stability. Instead, we consider a novel perspective in which the primary function of spatial remapping is to support three key nonperceptual processes: action control, sensorimotor adaptation and spatial memory. *NeuroReport* 18:1207–1213 © 2007 Lippincott Williams & Wilkins.

Keywords: corollary discharge, dorsal visual pathway, efference copy, parietal lobe, saccades, sensorimotor control, sensory prediction, spatial remapping, visual memory, visual stability

Introduction

The perceived stability of the visual world during eye movements is one of the classic problems in the study of perception. As Descartes observed in his *Treatise of Man*, if one taps on the side of one's eye with a fingertip, the resulting shift in the image falling on the retina induces a strong perception of external motion. In contrast, the similar shifts in retinal image produced several times every second by saccadic eye movements do not result in an equivalent perception of movement. This puzzle subsequently drew the attention of Purkinje, and later Helmholtz, who made the related observation that people suffering from eye paralysis report jumps of the visual scene when they attempt to move their eyes, even though in this case the image on the retina remains stationary.

In 1950, two researchers independently set out very similar models of the neural mechanism underlying these phenomena. Von Holst [1] and Sperry [2] were both studying the effects of surgically inverting the eyeball: Von Holst in insects and Sperry in fish. Following this surgery, any attempt by the animal to turn to the left or right results in rapid spinning in that direction until exhausted. Both researchers realized this behaviour could be explained by the presence of a cancellation signal that normally maintains visual stability during movement but, due to the inversion of the eye, now acted to exaggerate any retinal shift caused by the animal's motion. According to Von Holst's 'principle of reafference', when the motor areas of the brain generate a motor command signal to move the eyes they also send a copy of the command to the visual areas. Von Holst proposed that this 'efference copy' acts as a cancellation signal, compensating for changes to the visual input caused by the eye movement and hence maintaining visual stability.

Simple subtraction of one signal from the other is not possible, as the efference copy signal is in motor coordinates whereas the visual input is in sensory coordinates. According to modern formulations of the theory, therefore, the efference copy signal is used to *remap* the presaccadic visual input in memory, generating a prediction of the expected visual input following the saccade (Fig. 1). This prediction can then be compared with the actual postsaccadic visual input: if no discrepancy is found, the visual scene is perceived as stable. If the predicted and actual visual input differ, the discrepancy is interpreted as the result of external motion: as when the eyeball is moved by a fingertip (in which case there is a shift in the retinal input but no matching efference copy). Conversely, when the eye is paralysed, efference copy signals are generated but now there is no matching retinal shift.

Neurophysiological evidence for remapping

Studies in monkey parietal cortex were the first to reveal neuronal activity that might be related to spatial updating across eye movements. It has been known for some time that both visual stimuli and eye movements elicit activity in parietal area LIP (lateral intraparietal area), a brain region in the dorsal visual pathway. Duhamel and his colleagues [3] devised experiments to test whether this region might also be involved in remapping object locations across saccades. LIP neurons have visual receptive fields which – like classical receptive fields throughout the visual system – are tied to retinal coordinates. Thus, whenever a stimulus is

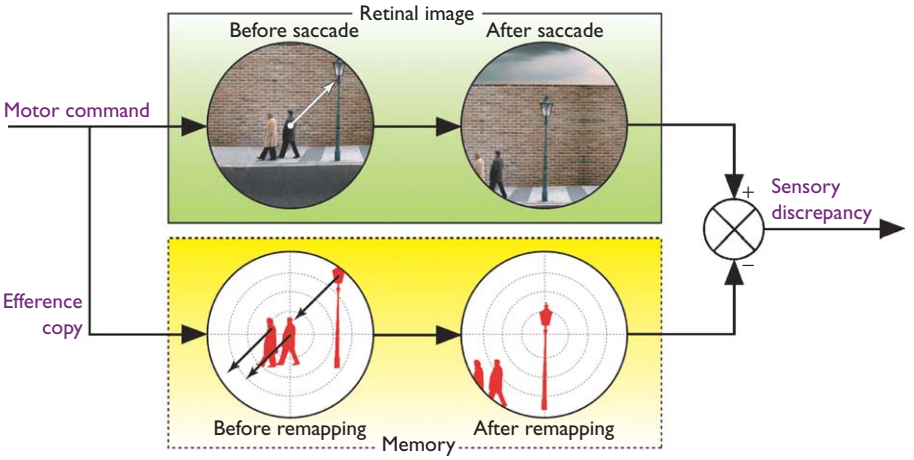

Fig. 1 Detecting intrasaccadic changes to visual input. An eye movement (indicated by the white arrow, top) causes a global shift in the image falling on the retina. According to the spatial remapping model, an internal retinotopic representation of the visual scene (bottom) is translated by a distance and direction specified by an efference copy of the saccadic motor command. This remapping (indicated by the black arrows) results in a prediction of the expected visual input following the saccade. Comparison with the actual postsaccadic input reveals any discrepancy due to external motion (e.g. in this scene, movement of the two walkers). Where there is no discrepancy, the visual scene is perceived as stable.

flashed in that part of the visual field which constitutes a neuron's receptive field, it fires, regardless of where the eyes are looking. Remarkably, Duhamel *et al.* found that the visual receptive fields of some LIP neurons appeared to shift just before a saccade, from their normal retinal location to the location the receptive field would occupy *after* the saccade.

For example, if a neuron had a receptive field that was located just below fixation (Fig. 2a) and the monkey was about to make a saccade up and to the right, the neuron would become responsive to a flashed stimulus that occupied its *future* receptive field location (denoted FF in Fig. 2b). The receptive field had become temporarily untied from retinotopic coordinates, having undergone a remapping that was specific to the forthcoming eye movement: the neuron fired only if the stimulus occupied the expected receptive field location following completion of the saccade. It appears therefore that this population of parietal cells use an efference copy signal to predict the sensory consequences of gaze shifts, as in the remapping model shown in Fig. 1.

Subsequent studies (reviewed in Ref. [4]) have shown similar remapping activity in two other critical brain regions associated with eye movements: the superior colliculus (SC) and the frontal eye field (FEF). Both LIP and FEF receive dense projections from the SC, which is thought to generate efference copy signals related to forthcoming eye movements. A series of elegant studies performed by Sommer and Wurtz has focused on the pathway between the SC and the FEF, which passes in monkey through the mediodorsal nucleus (MD) of the thalamus. These experiments have demonstrated that the colliculothalamic pathway to FEF is a critical route via which remapping occurs in the brain. Figure 2c shows the shift in responsivity from current receptive field to future receptive field in one FEF neuron. Following transient inactivation of the MD thalamic nucleus using the γ-aminobutyric acid agonist muscimol, such remapping activity was severely diminished in FEF cells [5].

Inactivation of the MD nucleus also led to impairments on the double-step saccade task [6]. In this paradigm, two stimuli are flashed rapidly in succession and observers have to make saccades to their *remembered* positions in the correct sequence (Fig. 3). Although the first saccade might be coded in retinotopic coordinates (relative to original fixation), the second eye movement has to take into account the new eye position after the first saccade. The original retinal position of the second target is insufficient on its own to compute the required second saccade. Instead, both the original retinal position of the second target and the updated position of the eyes after the first saccade have to be used to compute the vector required to shift gaze correctly to the remembered location of the second target. Monkeys with unilateral MD inactivation had no difficulty making the first saccade on this task, but made significant errors on the second saccade when the first eye movement was in the contralesional direction. This result is consistent with impaired updating of gaze location after the first saccade.

In humans, too, thalamic and frontoparietal lesions can lead to deficits on the double-step saccade task in keeping with impaired remapping of the location of the second target after the first saccade has been executed [4,7]. Neuroimaging experiments using functional MRI to investigate brain regions involved in spatial remapping in healthy human volunteers have not been easy to design. Given the protracted time course of the haemodynamic response, the delay between the first and second saccade has to be long to disambiguate the response associated with each eye movement. Nevertheless, some findings point to a role of dorsal parietal regions, perhaps homologous to monkey area LIP, in such a function [8,9]. Such studies suggest remapping of remembered spatial locations across the hemispheres, from one parietal region to its contralateral homologue, when an intervening saccade reverses the location of a remembered location relative to fixation. Thus, if a remembered location was initially left of fixation it would be encoded within right parietal cortex. But, if a large amplitude leftward gaze shift occurred so that the original remembered location was now to the right of current fixation, it would be remapped to the left parietal cortex. The recent application of magnetoencephalography to delayed double-step saccades might be a useful way to extend such studies [10].

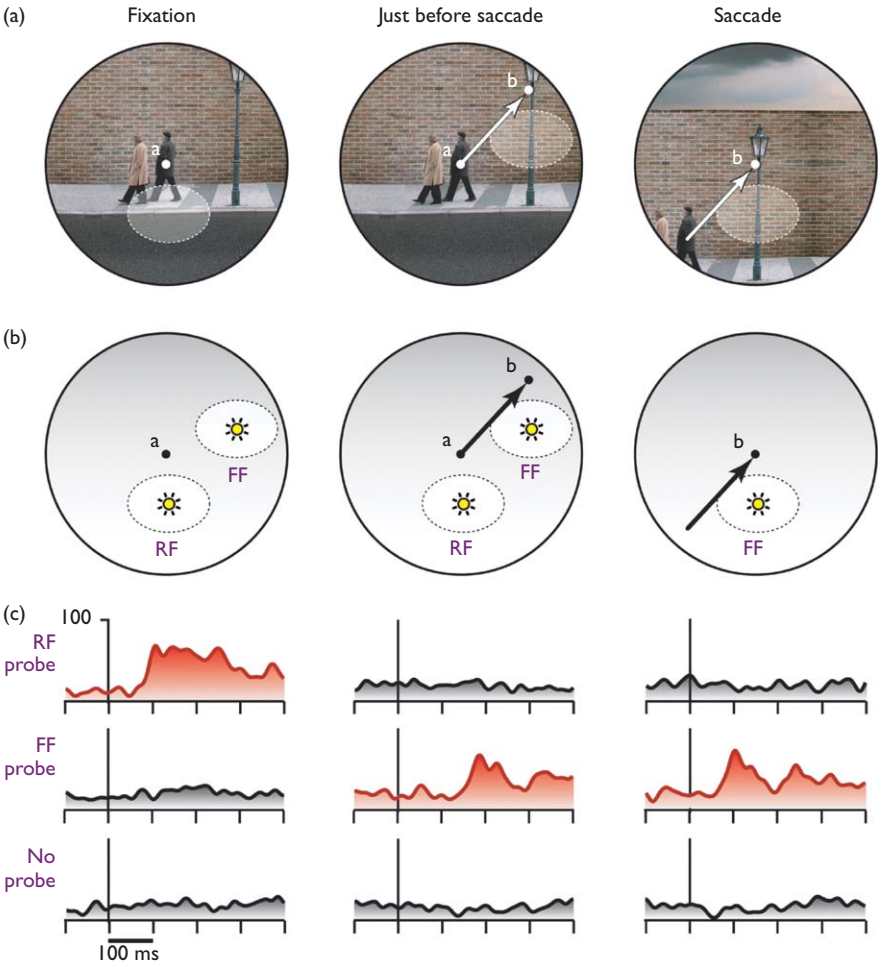

Fig. 2 Neural basis of spatial remapping. (a) An example visual neuron has an RF located just below fixation (left). Immediately before a saccade is made from 'a' to 'b' the neuron's RF shifts to a new position (centre). This position corresponds to the expected location of the RF following the eye movement (right). (b) Remapping is probed experimentally in monkeys by flashing a probe in either the presaccadic RF or the postsaccadic RF (FF), at various times relative to the saccade. (c) Activity of an example neuron in the frontal eye field. Firing rate (in spikes/s) is aligned with probe onset, for probes presented at the times illustrated in (a) and (b) above. The visual response shifts from the RF to the FF just before the saccade. FF, future field; RF, receptive field. (Adapted with permission from Ref. [5]).

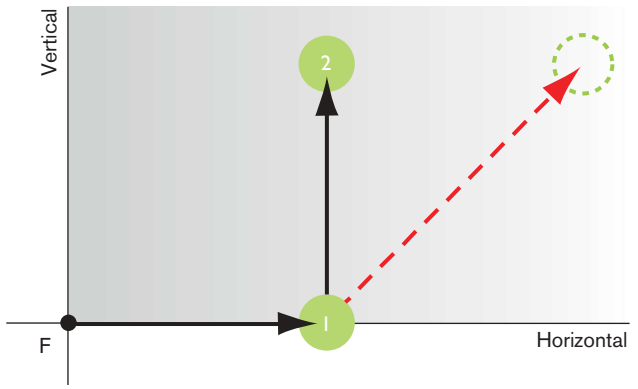

Fig. 3 The double-step saccade task. Two targets (1 and 2) are briefly presented in quick succession. The participant must then saccade from the initial fixation point (F) to the remembered target locations in the correct sequence (black arrows). Motor planning of the second saccade must take into account the change in eye position owing to the first saccade. Planning the second eye movement without remapping the retinotopic representation of target 2 would result in an erroneous horizontally deviated saccade (red arrow) computed on the basis of the second target's retinotopic position as viewed originally at fixation point (F).

Although neuronal populations have been identified that predict the effect of forthcoming saccades, it is not clear how or where the comparison is made between this prediction and actual visual input. As remapping neurons appear to predict *their own* postsaccadic activation, this comparison may require some form of delay mechanism that brings the prediction signal into temporal alignment with the postsaccadic signal. Studies of efference copy mechanisms in nonvisual modalities suggest the cerebellum as a candidate location for the comparison mechanism [11–13], but an involvement of this structure in spatial remapping has not yet been established.

Influence of remapping on perception

As we have seen, Von Holst's theory of efference copy has found strong support in the neurophysiology of the primate visual system. Furthermore, similar efference copy mechanisms have now been demonstrated in electrosensory [11], vestibular [13], auditory [14], and tactile [15] sensory processing. Ironically, the phenomenon for which an efference copy model has been hardest to substantiate is the one it was designed to explain: the perceived stability of the visual world.

Underlying early cancellation theories of visual stability was the assumption that a detailed visual representation of the retinal image is retained from one fixation to the next. Once the self-generated retinal shift was accounted for, comparison of presaccadic and postsaccadic images would allow accurate detection of any actual change or movement in the visual scene. Subsequent research, however, has shown that the ability to compare visual input across eye movements is in fact very limited. Indeed, it is possible to make remarkably large changes to a visual scene during a saccade without the viewer becoming aware of them, including displacing items, changing their forms, and even deleting them entirely (see Ref. [16] for a review). Studies of transsaccadic memory have revealed a number of characteristics that may explain this 'change blindness'.

First, it appears that transsaccadic memory has a very limited capacity. Studies by Irwin and colleagues [17,18] have attempted to estimate the number of visual items that can be retained in memory across a saccade. In these studies, an array of letters was presented along with a saccade target. When the participant initiated a saccade to the target, the letter array disappeared and was replaced by a probe marker. On the basis of participants' ability to recall the letter indicated by the probe, Irwin estimated that memory for at most three or four objects could be retained across a saccade. This is equivalent to estimates of the capacity of visual short-term memory (vSTM), the information maintained when static visual input is briefly interrupted by a temporal gap or a mask, without any intervening eye movement being made (e.g. Ref. [19]). Irwin proposed that no additional information is maintained across a saccade beyond that stored in vSTM.

Certainly, transsaccadic memory shares several further features with vSTM. For one, transsaccadic memory appears not to consist of veridical images of remembered items, but rather of abstract representations of object properties. Thus, it is not possible to perceptually fuse two patterns presented in successive fixations into a composite image, as one can within a single fixation (e.g. Ref. [20]). Naming of words or objects is facilitated if semantically similar items are presented in the previous fixation, but this facilitation is unaffected by intrasaccadic changes to visual features such as size or letter-case. In fact, the letter-cases of words can even be changed during an eye movement without participants' awareness and without disrupting reading (reviewed in Ref. [21]).

Most importantly for the role of remapping in visual stability, detection of object displacement during a saccade appears to depend not on absolute shifts in retinal location, but rather on changes in position *relative to other objects* in the scene [22–24]. For example, Germeys *et al.* [24] presented participants with a random array of dots, one of which changed position during a saccade. Detection of the location change was shown to depend on the continued presence of other array items that could act as visual landmarks, particularly the item fixated immediately before the saccade. These results are consistent with previous research in which stationary landmarks were dispensed with altogether by shifting the entire visual scene during a saccade. With no fixed reference points, displacements of several degrees of visual angle could be made without the viewer's awareness [25].

It would appear therefore that in our day-to-day experience, the perception of a visually stable world is *not* the result of an active process of spatial remapping, but rather a consequence of the abstract and configurational encoding of the limited information retained across a saccade. This is borne out by a reexamination of some of the visual illusions that were taken by early investigators as evidence for an efference-copy account. Matin [26] confirmed that paralysis of the eye muscles with curare caused illusory visual shifts of a single target viewed in darkness; however, this effect disappeared when the room was normally illuminated. Similarly, while remapping accounts predict that a retinal afterimage viewed in darkness will appear to jump with each saccade, this in fact holds true only for small afterimages and is not observed for an extended afterimage of a complex scene [27].

Is the role of spatial remapping therefore only to maintain stability in a sparse visual environment, for instance when following a single point of light in darkness? In this situation, the change in the retinal image caused by a saccade is identical to the change caused by a contrary movement of the light source, so the only means of discriminating between eye and target motion is by taking into account extraretinal signals. Our ability to make this discrimination has been investigated in detail by a number of researchers (e.g. Refs [28–30]).

In a typical experiment (Fig. 4a), participants are instructed to make an eye movement to a small target presented against a blank background. At saccade onset, the target is shifted by a variable distance either towards or away from the initial fixation point; participants must report the direction of target displacement. As was the case with studies that shifted the entire visual scene, detection of these intrasaccadic displacements is found to be remarkably poor. Nonetheless displacements can be detected if they are sufficiently large: the detection threshold is variously estimated at between 10 and 30% of the size of the saccade. This has been taken to imply that some information about absolute target location is retained across an eye movement, but that it is ignored in favour of an assumption that object positions remain constant during a saccade. Only when a very large discrepancy is detected between presaccadic and postsaccadic images is this assumption overcome [25].

Niemeier *et al.* [31] have formalized this hypothesis within a Bayesian framework, proposing that perceived target motion results from an optimal integration of sensory input, efference copy, and a *prior expectation* of object stability. A prediction of postsaccadic target location based on a spatial remapping of the presaccadic input will inevitably be imprecise, because variability in motor execution creates a discrepancy between intended and actual eye movements (Fig. 4c). According to Bayes theory, the optimal way to combine multiple sources of information is to weight each according to its precision [32]. The greater the uncertainty in the predicted target location, the more a Bayesian mechanism will rely on the prior expectation that the target is stationary during the saccade. This model successfully predicts two well-documented features of intrasaccadic displacement detection. First, detection threshold increases linearly with saccade magnitude [29]. This is predicted by a Bayesian model because variability in saccadic endpoint also increases in proportion to the size of the saccade [33]. Second, saccadic variability is greatest in the direction of the saccade [33], so the Bayesian model correctly predicts that it will be easier to detect target shifts perpendicular to the saccade than shifts parallel to it [31].

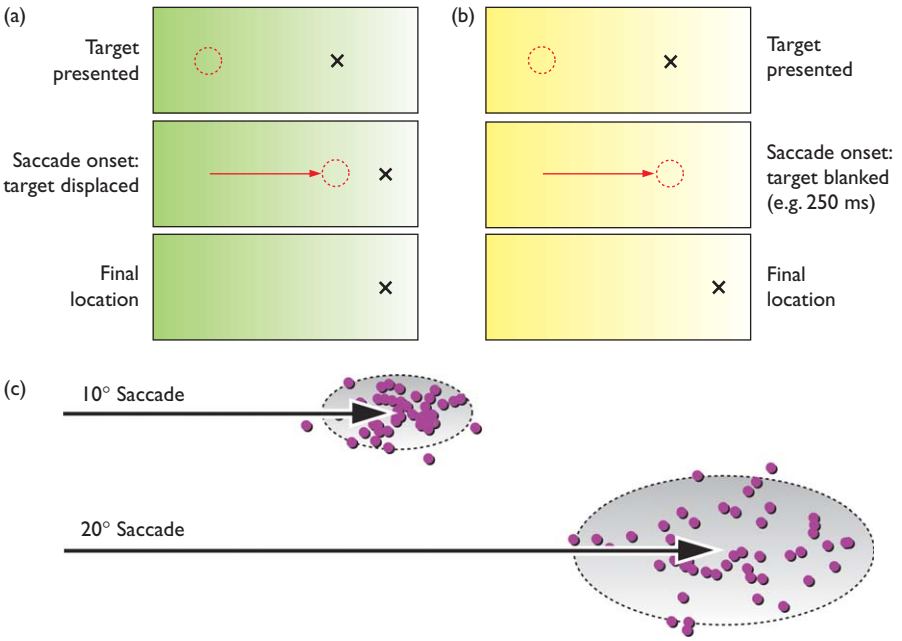

Fig. 4 (a) A typical experimental procedure demonstrating saccadic suppression of displacement. A saccade target (X) is presented on a blank screen at a location peripheral to fixation (red circle). Triggered by onset of the saccade, the target shifts to a new location. Participants are generally very poor at detecting the intrasaccadic displacement. (b) In a variant of this procedure, the saccade target is briefly blanked at saccade onset, reappearing in its new location only after the saccade is complete. This manipulation greatly improves participants' ability to detect the displacement [28]. (c) Saccades are variable in size and direction owing to noise in motor output. Typical distributions of actual saccadic endpoints (purple dots) about the intended endpoint (arrow tip), are shown for saccades of 10 and 20° (arrows not to scale). Grey ellipses indicate areas expected to contain 95% of endpoints. Uncertainty over saccadic endpoint is greater in the direction parallel to the saccade than perpendicular to it, and scales with saccade size.

Is it possible to break the assumption of object stationarity? Deubel *et al.* [28] introduced a simple manipulation to the intrasaccadic displacement paradigm: at eye movement onset the saccade target is blanked, reappearing in a shifted position only after completion of the eye movement (Fig. 4b). Counterintuitively, the brief disappearance of the target makes its displacement much easier to perceive, with reliable detection of shifts as small as 5% of the saccade size. In terms of the Bayesian model, the absence of the target in the immediate postsaccadic image causes the prior assumption of object stability to be dropped [31]. Under these circumstances, when the target reappears the perceived shift is the result of a direct comparison between its actual retinal location and the location predicted by a remapping of presaccadic position.

Alternative functions of spatial remapping

The blanking paradigm provides the first clear psychophysical evidence that a high-resolution memory for absolute spatial location is indeed maintained and remapped across a saccade – at least for the location of the saccade target. Its relevance to normal visual perception, however, is doubtful. The psychophysical evidence reviewed above, combined with the fact that remapping neurons are located within the dorsal rather than ventral visual pathway [34,35], suggests a new perspective: that the primary role of spatial remapping may be to support action rather than perception.

The first evidence that spatial remapping might contribute to accurate motor control came from studies in which participants were asked to point to targets that shifted during an eye movement (e.g. Ref. [36]). Despite failing to perceive the intrasaccadic displacement, participants made rapid online corrections to their arm movement to account for the change in target location. This suggests that the motor system may have direct access to the sensory discrepancy detected by a remapping mechanism (Fig. 1). Alternatively, this result could reflect an egocentric representation of target position updated using eye-in-head proprioception. However pointing to the *remembered* location of visual targets is biased by a change in eye position [37], supporting the neurophysiological evidence for a retinotopic representation that is remapped with each saccade. Surprisingly, the same effect is found even when the targets are auditory or proprioceptive [38], suggesting that the primary coordinate system for all internal representations of space may be retinotopic (but see Ref. [39] for a contrary view). If this hypothesis were to prove correct, spatial remapping – originally invoked to explain perceptual stability – would be a critical component of *motor* control, whether visually guided or not.

A remapping mechanism may also play an important role in sensorimotor adaptation. The motor signal needed to generate a particular eye movement changes over one's lifetime, as the eye grows and ocular muscles strengthen and weaken. Accuracy is maintained by a process of saccadic adaptation, in which persistent errors in saccadic endpoint are corrected by changes to motor output [40]. The simplest model for this adaptation suggests that it is triggered by a purely retinal discrepancy between the saccade target and the fovea following an eye movement. Some results, however, are difficult to account for within this model: saccadic adaptation can be induced even when the saccade target is not a clearly defined object [41] or when

participants intentionally fail to foveate the target [42]. A comparison of predicted and actual postsaccadic input based on a remapping mechanism could provide a more accurate error signal for adaptation. Similarly, adaptation of the vestibuloocular reflex (which stabilizes gaze direction during head movements) is often assumed to result from visual motion transients induced by gain errors, but could also be explained in terms of an efference copy mechanism.

Further roles for spatial remapping are suggested by recent human lesion studies. Damage to the right inferior parietal lobe and adjacent regions in the intraparietal sulcus frequently results in hemispatial neglect, a condition in which patients have difficulty perceiving, attending and directing movements towards objects in the contralesional hemispace [35]. In the last few years, however, patient studies have begun to identify additional nonlateralized deficits in spatial memory, particularly following parietal damage. In a visual search task, individuals with parietal lesions were found to have difficulty maintaining memory for previously examined locations, repeatedly refixating the same items (Fig. 5). Moreover, these patients failed to identify the refixated items as ones they had seen before [43–45]. Similarly, parietal patients are specifically impaired in maintaining memory for object locations in a change detection task, an impairment not observed for object shape or colour [46].

It is possible that these memory deficits reflect damage to parietal networks that remap object location information across eye movements. A recent study has tested this hypothesis directly by examining memory for object locations with an intervening eye movement [47]. Unusually, right hemisphere patients showed a specific deficit on this task when the saccade was made into *right* hemispace (the 'intact' ipsilesional side). This seemingly paradoxical result can be explained by considering that such a saccade causes the representation of a centrally located object to be remapped retinotopically *into a damaged representation in the lesioned hemisphere*, hence the difficulty in maintaining its memory. By contrast, a leftward saccade towards the neglected side of space means that objects that were at fixation now fall into right hemispace and therefore are remapped into the intact left hemisphere.

A further role for parietal representations of object location may be to enable the binding together of object features such as colour and motion, analysed in disparate specialized regions of visual cortex, into a unified object representation. Bilateral damage to parietal cortex can result in simultanagnosia, the inability to perceive simultaneously multiple objects, which may be the consequence of a deficit in object binding [48]. As yet the role of eye movements in simultanagnosia has not been systematically investigated: if the location information used for feature binding is remapped across each saccade, we would expect binding to be particularly vulnerable in these patients when they make eye movements.

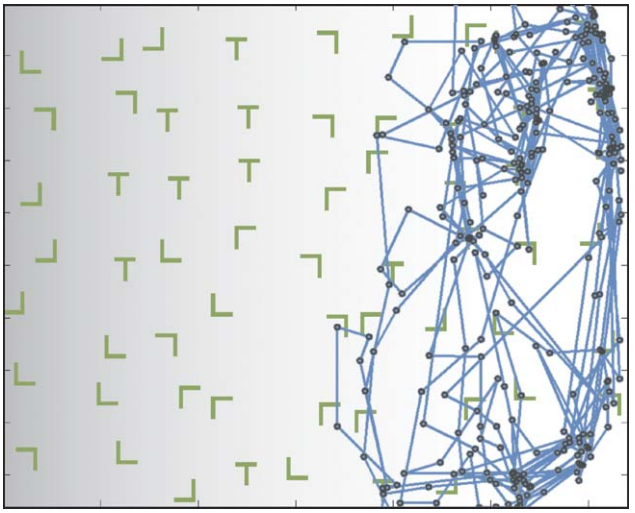

Fig. 5 Typical scanpath of a patient with a right parietal lesion searching for target letter Ts among distractors. Note both the neglect of the left side of the search array and the many refixations of stimuli on the right. (Adapted with permission from Ref. [44]).

Conclusion

Spatial remapping based on an efference copy of eye motor commands is frequently cited as a mechanism for maintaining the perception of a stable visual world across eye movements (e.g. Ref. [5]). There is, however, considerable evidence that efference copy has only minimal involvement in generating this perception, at least in everyday experience. Nonetheless convergent findings from monkey neurophysiology and human psychophysics clearly support the existence of a remapping mechanism that predicts the sensory consequences of a gaze shift. Here, we have set out some of the possible functions such a mechanism could have aside from influencing perception: in motor control, sensorimotor adaptation, and spatial memory. Our understanding of the involvement of spatial remapping in each of these functions is at a relatively early stage, and clearly in need of further development if we are to fully appreciate its contribution to these critical aspects of behaviour.

Acknowledgement

This work was supported by the Wellcome Trust and NIHR CBC at UCLH/UCL.

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