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Abstract
Recent evidence has implicated posterior parietal cortex (PPC) in adaptation to optical displacing prisms. It has been suggested that PPC contributes to the strategic component of prism adaptation necessary for perceptual realignment (true adaptation). It has also been suggested, however, that the part of PPC responsible for corrections to ongoing movements (a putative strategic component) may not be necessary for successful adaptation. A patient presenting with bilateral posterior parietal damage (patient JJ) was tested with both hands on two versions of a prism adaptation task—one using prism goggles and one using a virtual prism arrangement. JJ displayed independent deficits: his right hand failed to show strategic control, yet adapted fully to the prisms whereas his left hand showed evidence of strategic control without subsequent adaptation. The data indicates that the ability to implement control strategies may not be necessary for successful adaptation to prisms. A proposed model for the role of posterior parietal cortex in prism adaptation is also presented.

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I. Introduction
Despite the fact that optical displacing prisms have been in experimental use for many years the processes and neural mechanisms that enable prism adaptation remain poorly understood. Until relatively recently it was thought that prism adaptation was a type of motor learning largely undertaken by the cerebellum. In the monkey, ablation of regions of the cerebellum that receive ‘mossy-fibre’ cortical input via the pontine nuclei abolishes prism adaptation (Baizer, Kralj-Hans, & Glickstein, 1999; Stein & Glickstein, 1992), and humans with cerebellar lesions are also impaired at prism adaptation (Martin, Keating, Goodkin, Bastian, & Thach, 1996; Morton & Bastian, 2004; Weiner, Hallett, & Funkenstein, 1983). More recent evidence however has started to reveal an important role for the parietal (Clower et al., 1996) and premotor areas (Kurata & Hoshi, 1999) in prism adaptation. In particular, parietal areas have been implicated by functional neuroimaging (Clower et al., 1996) and lesion studies (e.g. Newport, Brown, Husain, Mort, & Jackson, in press; Pisella et al., 2004; Rossetti et al., 1998). Before examining a putative role for each of these structures in prism adaptation, it is useful to provide a brief account of prism adaptation and the generally accepted processes that give rise to it.

The effects of prism adaptation are striking. Wearing wedge or fresnel prisms makes objects appear to be to one side of where they are actually located (see Fig. 1a). When the wearer reaches out to touch or pick up an object viewed through prisms, they misreach in the direction of the visual deviation. After a few such reaches, however, their accuracy improves rapidly, and they are soon able to accurately reach directly to the target location. This is known as prism adaptation or the prism adaptation effect. If, once an individual has adapted to optical prisms, the prisms are removed, then the first few reaches made without prisms will also be inaccurate. This time, however, subjects will misreach in the opposite direction to the visual deviation caused by the prisms (Fig. 1b). This is known as the negative aftereffect or the prism aftereffect, and it is generally regarded as the ‘true’ test of adaptation because it gives an indication of the amount of realignment that has taken place while wearing the prisms (realignment will be discussed shortly). It should be noted that for rapid and efficient prism adaptation to occur, certain conditions must apply. Negative aftereffects are greatest when
his shots more accurate: he can aim approximately $10^\circ$ to the left or he can adjust the scope until it is properly realigned. The first solution (analogous to strategic control) might be effective in the short-term, but is unlikely to provide long-term stability to his marksmanship whereas the second (spatial realignment) might take a little longer, but will provide a permanent solution.

Unfortunately, this is as far as our simple analogy can take us as prism adaptation turns out to be a little more complicated as the two processes are thought to rely on one another (Redding & Wallace, 1996, 2000). Specifically, realignment is thought to depend upon strategic control for the detection of misalignment, but strategic control can also interfere with the realignment process by reducing the apparent misalignment (thus a side-point estimate that is immediately accurate produces the illusion of no misalignment and dramatically reduces adaptation). Moreover, as spatial realignment progresses, strategic control must be reduced in order to avoid ‘overcompensating’ for the prism-induced displacement. Strategic control involves such stratagems as side-pointing (deliberately planning a reach to miss in the direction opposite to the displacement), ‘on-line’ corrections to the path of the movement (when a selected motor plan proves erroneous) and recalibration. Recalibration provides a temporary and local rearrangement of spatial representations within a specific workspace for a specific task and might be employed, for example, when learning to use a mouse to control a cursor on a computer screen. Prism studies employing terminal exposure conditions rarely allow much time for in-flight corrections and it is likely that recalibration (of which deliberate side-pointing is sometimes considered a part) is the primary strategy employed under such circumstances. Spatial realignment on the other hand involves the gradual realignment of visual and proprioceptive spatial maps so that the seen and felt positions of the reaching limb are in concert. It is a slow and iterative process that develops gradually and can be contrasted with strategic correction, which is much faster and can be observed to operate even within the first few trials.

In summary, there are two processes at work during exposure to prisms: one that operates very quickly to reduce the apparent reaching error and one that operates very slowly to produce long-term accuracy through accurate correspondence between spatial maps. The rapid reduction in reaching error (see Fig. 1b which illustrates typical prism adaptation) usually takes somewhere between 5 and 15 trials depending on the particular conditions employed. If the exposure phase were to end here (i.e. prisms removed) participants would not normally exhibit aftereffects. Typically, participants must be exposed to the prism deviation for a minimum of 30 trials. During this time reaching does not become any more accurate—indeed, paradoxically, occasional apparent temporary regressions in accuracy can be seen during the later stages of prism adaptation. Although they both operate simultaneously the strategic component is evidently not fully dependent on the progress of the realignment process, and over or under use of strategic corrections, relative to the progress of spatial realignment, can produce the mildly inaccurate reaches often seen during late prism exposure trials (Fig. 1b).

Prism adaptation seems therefore to involve multiple processes. It has been suggested that strategic control processes may

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**Fig. 1.** (a) When viewed directly, without prisms, an image of target T falls on the retina when it is fixated (left panel). If rightward deviating prisms are worn the image of T will fall to the left of the retina if the eyes are not rotated from their original position and the target will appear to be to the right of fixation at T′ (middle panel). In order to fixate the target when wearing prisms the eye must be rotated clockwise as far as T′, to the right if the actual target location (right panel). (b) Typical prism adaptation behaviour. In this example positive terminal errors are given as being in the direction of the prism perturbation. Typically accurate pre-exposure (without prisms) performance is shown in the left panel. During early exposure trials (middle panel) reaching errors are large and in the direction of the prism perturbation, but rapidly reduce in magnitude until they hover around pre-exposure levels of accuracy. Occasional regressions in accuracy are often seen during this phase. Following removal of the prisms again (right panel) errors can be seen in the direction opposite to the prism perturbation, but are typically not as large and as long lasting as exposure (test phase) errors.
occur within the posterior parietal cortex (PPC) while realignment processes are dependent upon cerebellar mechanisms. The main focus of this article will be to consider further how PPC and its connections might give rise to the strategic component of prism adaptation.

Several lines of evidence implicate the parietal lobes in the process of prism adaptation. Firstly, various brain imaging studies have highlighted activation in the PPC during visuomotor adaptation (Clower et al., 1996; Inoue et al., 1997, 2000). In particular, the study by Clower et al. (1996), using positron emission tomography (PET) directly examined the brain areas active during manual pointing while subjects wore optical prisms. Activation was observed in area PEG on the lateral bank of the intraparietal sulcus (IPS) contralateral to the reaching limb (although the precise location of this activation has since been questioned and now looks to be more accurately localised to the supramarginal gyrus [BA 40] see Pisella et al., 2004) while subjects pointed to targets that were displaced by leftward or rightward deviating prisms that alternated every four trials. This study provides supporting evidence for the idea that the rapid strategic component of prism adaptation involves PPC as, due to the alternating nature of the prism task, subjects were never permitted to adapt using spatial realignment mechanisms, which also explains the absence of cerebellar activity in this study.

A second source of evidence comes from patients with visuospatial neglect, a disorder that is frequently the result of right posterior parietal damage. Neglect patients typically fail to explore, orient towards, or otherwise respond appropriately to stimuli presented in left space. However, in many cases neglect can be improved by exposing patients to rightward displacing prisms (Rossetti et al., 1998; Rossetti and Rode, 2002; Rode et al., 2003). Although the precise mechanisms through which prism adaptation reduces the behavioural signs of neglect remain unclear, it is thought that exposure to prisms may promote the realignment of dysfunctional spatial maps and that the amelioration of neglect symptoms is the result of a long-lasting negative aftereffect that shifts the body-centred reference frame toward neglected space. Another point to consider is why neglect patients tend to show much larger and longer-lasting aftereffects than neurologically normal subjects (Rode, Rossetti, Li, & Boisson, 1998/1999; Rossetti et al., 1998). It has been suggested (Redding & Wallace, 1997) that strategic control is not only necessary for the detection of the misalignment that produces realignment, it might also impose a limit on realignment. Once strategic control is no longer necessary, realignment must be complete. If strategic control in parietal patients is faulty then this limiting factor may be removed, resulting in ‘over-realignment’ of spatial maps.

Another reason why the PPC is a likely candidate for the strategic control component of prism adaptation is that it has been shown to be involved in the correction of movements during their execution (so-called ‘on-line’ correction) (Desmurget et al., 1999; Pisella et al., 2000; Gréa et al., 2002). On-line corrections contribute to the rapid reduction of error that is a key component of strategic control. Transcranial magnetic stimulation (TMS) over PPC shortly after movement onset disrupts corrections to reaches made to unseen target jumps (Desmurget et al., 1999). Moreover, neuropsychological evidence gathered from patient IG (Pisella et al., 2000; Gréa et al., 2002), who suffered extensive bilateral PPC damage, has demonstrated that IG is unable to make corrective on-line movements when reaching to a target that unexpectedly displaced to the left or right. Instead, IG completed her initial reaching movement (to the old target position) and then executed a secondary movement in order to achieve the new target location. Not all prism adaptation paradigms allow time for online error corrections, however, and such corrective movements are probably not a necessary component of prism adaptation (although they may contribute to strategic control). This point is borne out by the testing of patient IG under conditions of prismatic displacement. Despite being unable to perform online corrections patient IG was nevertheless able to achieve movement accuracy under exposure conditions in a similar time-frame to that of controls (i.e., five trials for IG versus an average of four trials for controls) and her adaptation to prisms, as measured by the magnitude of a negative aftereffect, was not different to that of controls (although the transfer of adaptation to the unexposed limb was much greater). Therefore, while online control is considered to be part of the strategic control process, it does not appear to be necessary for true adaptation. As stated earlier, on-line correction is only one of the many strategies that could be employed during strategic control and under terminal exposure conditions the most useful strategy might be that of side-pointing.

Jackson et al. (2005a) reported another neurological case (JJ) with bilateral PPC damage who, unlike IG, exhibited a clear impairment of prism adaptation. JJ presented with Balint’s syndrome and had a profound optic ataxia as part of the triad of disorders associated with this syndrome. When asked to pick up objects presented in peripheral vision JJ tended to reach towards the point of fixation instead of the target, especially with his right hand. The authors suggested that JJ’s prism adaptation failure was a consequence of this deficit and that he was unable to plan reaches away from the point of fixation. Thus, JJ could not employ the side-pointing strategic control necessary to trigger spatial realignment. JJ’s inability to side-point and his impaired adaptation therefore suggest that side-pointing is a necessary component of prism adaptation.

The current paper presents new data from patient JJ that casts doubt on that assumption. JJ was tested on an extended and modified version of the original prism study. In the first study JJ showed little sign of adaptation after 48 exposure reaches (normal participants adapt comfortably within this number of exposures). In the current study JJ was allowed 100 exposure reaches. Furthermore, using mirrors and a projector set-up, JJ viewed a representation of his hand through the prisms rather than his actual hand. This allowed a comparison between an optical prism version of the experiment and a ‘virtual’ prism version, in which visual feedback from the hand was displaced by computer software instead of prisms. In all other respects the two experiments were identical; the main difference of interest being that JJ was not required to rotate his eyes in order to view the target in the virtual prism condition. In the previous prism study JJ’s endpoint errors during the exposure phase were more...
than twice that expected by the prism deviation. Given that a narrowed functional visual space is a common consequence of Balint’s syndrome (e.g. Hauser, Robert, & Giard, 1980; Pierrot-Deseilligny, Gray, & Brunet, 1986), large enforced rotations of the eye might produce abnormally large misrepresentations of visual information in an oculocentric system. If this is the case with JJ then reaching errors during the exposure phase of the virtual prism experiment should be smaller than those in the prism experiment despite the absolute magnitude of the perturbation being the same.

2. Methods

2.1. Participants

2.1.1. Controls

Twelve young neurologically healthy adults (six male, six female; mean age 20.5 years) and one age-matched male control (65 years) gave informed consent to participate in the study. None of the participants had any known history of neurological disorder or head injury and all had normal or corrected to normal vision. All were right-handed and all were naive as to the purpose of the study.

2.1.2. Patient JJ

At the time of testing, JJ, a right-handed male, was 67-years-old. A T1weighted MRI scan revealed asymmetrical bilateral atrophy predominantly of the parietal lobes with the damage in the left parietal cortex extending further superior than in the right hemisphere. Damage to the left hemisphere involved the SPL and IPS, and angular gyrus. In addition, there was signal change consistent with degeneration of the white matter in the occipital lobe. Damage to the right hemisphere involved the IPS and the posterior aspect of the angular gyrus. There was also signal change within the white matter of the occipital lobe. Both hemispheres showed evidence of damage to white matter tracts underlying the parietal cortex. Patient JJ gave his informed consent prior to his participation in the study.

A more comprehensive account of his deficits has been published elsewhere (Jackson et al., 2005a; Jackson, Newport, Mort, & Husain, 2005b), but briefly, JJ was impaired in the identification of simultaneously presented multiple or complex patterns, had extreme difficulty in making accurate saccades towards visual objects in space and was impaired when reaching towards visual targets presented in extra-foveal vision, particularly with his right hand. Jackson et al. (2005b) provided a detailed case study of this patient describing a limb-dependent form of optic ataxia. JJ could reach accurately with either hand to objects that he could foveate, but showed profound misreaching when executing movements using his right limb, and only for movements that were directed to objects that he could foveate, but showed profound misreaching when executing movements using his right limb, and only for movements that were directed to extrasaccadic targets. They ruled out explanations based upon simple perceptual or motor deficits and suggested an impairment in the ability to disassociate the eye and limb visuomotor systems when appropriate. In essence, JJ could not effectively decouple reach direction from gaze direction for movements executed using his right arm.

2.2. Apparatus and procedure

Participants sat at a table in a dimly lit room. A mirror, positioned above the table at chin height, reflected images projected onto a semi-opaque screen mounted equidistant above the mirror. Thus, images presented on the screen, when viewed in the mirror, appeared to be in the same plane as the tabletop (see Fig. 2). In this way a computerised representation of the participant’s index finger (a 10 mm black circle) and target (20 mm green circle) appeared to be in the same plane as the limb movement. Limb movements were recorded at 80 Hz using a Membrad electromagnet sensor (Ascension Technologies Corp.) attached to the nail of the index finger. Position data were filtered on-line using a Butterworth filter with a cut-off frequency of 10 Hz. Throughout the experiment the representation of the location of the participant’s finger was displayed with a delay of 11 ms. With the participant’s finger positioned on a tactile marker centred on their midline, a tone, coincident with target onset,
2.4 Virtual prism condition

The virtual prism condition also consisted of pre-, test- and post-exposure phases performed in that order. Participants made 40 reaches in each phase except JJ who completed 100 reaches in the test phase. During the test phase a lateral perturbation was applied to the representation of the finger position by computer software. The magnitude of the shift was 14.7°, equivalent to that produced by 25 dioptre prisms. In the pre- and post-exposure phases no perturbation was applied and the representational dot reflected the true position of the finger. Participants were informed prior to each phase whether or not a perturbation would be applied. This was to reflect the fact that participants are aware of the application and removal of prism goggles. The nature of the mirror set-up ensured that only the last 50% of the movement was visible to the participant.

It is important to note the differences and similarities between the two experiments. In both experiments only a representation of the finger could be seen, not the finger itself, and in both test phases the apparent position of the finger was perturbed by the same amount (14.7°). The primary difference between the experiments was that in the prism experiment both the target and the hand representation were perturbed and a rotation of the eye in the direction of the prism perturbation was necessary in order to fixate the target, whereas in the virtual experiment only the finger representation was perturbed and no optical rotation was required.

Half of the control group were tested using their right hand only and half with their left. The order of presentation between the prism and virtual conditions was counterbalanced. Both the age-matched control and the patient completed both experiments with both hands in the following order: virtual left hand, prism right hand, virtual right hand, prism left hand. Each condition was conducted on separate occasions to minimise carry-over effects.

3. Results

The angle in degrees subtended between a straight line drawn from movement startpoint to movement end point and a line drawn from movement startpoint to the target location formed the terminal direction error score (DE) for each trial. Leftward deviations were expressed as negative scores and rightward deviations as positive. Movement start and endpoints were defined as the time at which the tangential velocity of the movement rose above or fell below 50 and 100 mm/s, respectively. The slightly higher cut-off value for the end point was to reduce the possibility of secondary corrective movements being included in the analysis.

Trials were placed into bins of four trials in the order in which they occurred. Due to the pseudo-randomisation procedure, one trial to each target location appeared in each bin. Means were calculated for each bin and 95% confidence intervals were produced for each bin for the control group. See Fig. 3 for details of the prism experiment and Fig. 4 for the virtual prism experiment.

3.1 Prism experiment

3.1.1 Control group performance

The graphs clearly indicate that for both hands in the prism condition the control group performed in the manner expected. Baseline reaches achieved a high level of accuracy throughout the pre-exposure phase whereas initial test phase reaches were highly inaccurate, averaging 8.9° for the right hand and 12.6° for the left hand in the first bin – a level within normal limits for a 14.7° prismatic shift. Inaccurate reaching was short-lived and baseline levels of accuracy were re-established by the third bin – again within normal limits. In the post-exposure condition the group displayed the classic negative aftereffect, misreaching to the left (the direction opposite to the prism displacement) by an amount less than in the exposure condition for the first bin, but becoming accurate again soon after.

3.1.2 Age-matched control performance

The performance of the age-matched control was almost identical to that of the younger control group and was within the 95% confidence limits throughout all three phases of the experiment with both hands.

3.1.3 Patient JJ right hand

In the pre-exposure phase JJ was within normal limits with both his left and right hand. In the test- and post-exposure phases,
however, his performance was very much different to that of controls. Right hand misreaching was far greater in magnitude than the controls, being 22.8° in the first bin. In contrast to controls, JJ’s performance did not improve rapidly and, rather than becoming accurate after only a few bins, JJ was still missing the target by 16.2° after 100 trials (25 bins). The reduction from almost 23°–16° over the course of the exposure phase was slow, but consistent. Despite this failure to achieve accuracy, JJ nevertheless showed a negative aftereffect that was much larger (−13.0°) and longer-lasting than that shown by controls, persisting for four bins.

3.1.4. Patient JJ left hand
JJ’s left handed reaches showed an entirely different pattern of effects. During the exposure phase reaches were again highly inaccurate (27.8° in bin 1), but errors reduced quite rapidly (more than 5° in bin 2) until the 7th bin when they suddenly reversed direction and returned to levels of inaccuracy greater even than in bin 1. Again, in contrast to his right hand, and also to the control group, JJ showed no evidence of a negative aftereffect in the first bin or indeed even in the first trial (3.8° positive error). Instead his reaches immediately returned to pre-exposure baseline levels of accuracy.

3.2. Virtual prisms experiment
3.2.1. Control group performance
The control group displayed very similar effects to that shown in the prism experiment, but the magnitude of the exposure effect and negative aftereffect were slightly reduced (7.5° exposure effect for both hands and −4.5° and −4.8° negative aftereffects for the right and left hands, respectively).

3.2.2. Age-matched control performance
Again, the age-matched control’s performance was almost identical to that of the younger control group and very similar to that in the prism experiment, but with smaller magnitude. Misreaching for the right hand started at 14.6°, gradually reducing to 8.5° and the negative aftereffect in bin 1 was −10.3°, reducing to baseline levels by bin 4.

4. Discussion
In this experiment we measured the ability of JJ, a patient with bilateral PPC damage and suffering from Balint’s syndrome, to adapt to two motor learning tasks with either hand. Patient JJ displayed a unique double dissociation: With his right hand he showed no evidence of being able to use strategic control in order to improve reaching accuracy under prism or virtual exposure, yet he still showed very large negative aftereffects—an indication of true adaptation. In contrast, with his left hand he did show signs of being able to use some form of cognitive strategy in order to reduce reaching errors, however unsuccessfully, yet there were no negative aftereffects, even after 100 exposure trials. These results confirm the hypothesis that the processes of strategic control and spatial realignment are functionally separate, but ask questions about the correspondence between the two.

4.1. Differences between real and virtual prism exposure
The results of this experiment also show two interesting features in relation to the similarities and differences between the actual and virtual prism conditions. Firstly, the overall pattern of results in the virtual prism condition is broadly similar to those in the actual prism condition: normal participants become accurate within the same number of trials and show a similar magnitude and duration of aftereffect. This may be explained by the fact that in both conditions there is a very strong proximity between the felt position of the hand and the seen representation of the position of the hand. In both prism and virtual adaptation phases the representation of the hand appears shifted by the same amount. The similarity of behavioural patterns could have far reaching consequences for the rehabilitation of neglect following parietal damage. Although prisms have been shown to be effective in the amelioration of neglect symptoms, they are limited insofar as the perturbation they produce is linear whereas the visuo-spatial distortion seen in neglect may not be. Using computerised feedback it should be possible to produce perturbations that are individually tailored to patients’ needs and thereby improving the efficacy of adaptation techniques.

Patient JJ’s performance between the two conditions differed however: the magnitude of his errors in the virtual prism adaptation phase was much smaller than those in the equivalent prism adaptation phase and aftereffects were also noticeably smaller. As mentioned in the introduction, the primary difference between the two types of adaptation was that in the virtual condition, in which the discrepancy between seen and felt hand position is generated by computer software rather than by an optical shift, the eyes do not have to be rotated in order to fixate the target. A constricted functional or attentive visual field is a common result of bilateral PPC damage in Balint’s syndrome (Haussser et al., 1980; Pierrot-Deseilligny et al., 1986) and might be thought of as being compressed bilaterally in the same way that visual space may be compressed unilaterally in neglect. If the whole of visual space is compressed into a narrow band then it follows that any visual targets located by enforced large rotations of the eye will be (mis) represented in
4.2. Differences between the two hands

Dealing firstly with the right hand, JJ showed no capacity for strategic correction, but nevertheless exhibited large and persistent aftereffects. We have previously suggested that JJ’s sensorimotor transformations are impaired. Specifically we have suggested that his reaches planned using his right hand are trapped in oculocentric coordinates: when JJ was required to perform reaches away from fixation his right hand always erred towards the point of fixation rather than reach to the target. The same was not true of his left hand, which consistently reached to the target location. This was true for both unimanual and bimanual reaches (Jackson et al., 2005a,b). With his right hand JJ seemed compelled to plan reaches specified by the direction of his gaze. Such an impairment would mean that no matter how much JJ tried to reach away from fixation (required for accurate strategic prism performance), he would always reach to a location specified (erroneously in this case) by eye direction. Endpoint error information, however, might still be available to the neural components necessary for spatial realignment allowing a very gradual improvement in accuracy seen over the 100 trials, a true adaptation that is demonstrated by the aftereffect. In normal participants accurate strategic correction may modulate spatial realignment, something absent in JJ and leading to abnormally large aftereffects.

In contrast, JJ’s left hand performance is entirely different. His errors rapidly reduce initially in line with normal strategic correction, but perhaps because the magnitude of the errors are so large and strategic corrections not sufficient, the strategy is abandoned or replaced and errors veer away again. Despite the ability to attempt strategic corrections (side-pointing) no spatial realignment takes place with the left hand. Endpoints are no nearer the target at the end of 100 trials than at the start and there is no evidence of any aftereffect. Here then, in a single patient, is a clear double dissociation between the two primary components of prism adaptation: strategic control and spatial realignment. The data also suggest that strategic control is not a necessary prerequisite for spatial realignment.

It was previously thought that strategic control processes were necessary to highlight the misalignment between the seen and felt position of the limb. Although it does not rule out the possibility that there are other strategic processes at work, the current data in combination with the results from patient IG suggest that neither deliberate side-pointing nor on-line corrections are necessary for prism adaptation. Crucially, both of these strategies are thought to be parietal functions so questions remain over the precise role of the parietal lobes in prism adaptation.

4.3. PPC and the dissociable components of prism adaptation: a model

Presented in Fig. 5 is a proposed model for the role of posterior parietal cortex in prism adaptation. Readers should note that anatomical connections are primarily inferred from monkey studies and that no attempt has been made for the precise localisation of hypothetical functions or connections within their respective brain regions. Furthermore, readers are reminded that this model is purely speculative in its attempt to reconcile the current patient data with a role for PPC in prism adaptation and is only provided to promote debate.

Prism adaptation involves two distinct processes, one (spatial realignment) thought to involve the cerebellum and another (strategic control) thought to primarily involve PPC. These two processes have their own functionally distinct circuitry, but must be able to communicate for fast and efficient adaptation. The ventral premotor area (PMv) is a likely candidate that forms the communicative link between the PPC and the cerebellum as it has reciprocal links with both structures and has been shown to be involved in the adaptation process (Kurata & Hoshi, 1999).

4.3.1. The cerebellar-PMv loop for spatial realignment

Spatial realignment under prism exposure is not a one-trial process and requires gradual modification of inverse models used to plan reaches to the apparent location of the target. The
4.3.3. The parieto-PMv loop for strategic control

The final circuit involves reciprocal parieto-frontal links between the PPC and PMv (Cavada & Goldman-Rakic, 1989; Closer, West, Lynch, & Strick, 2001; Fogassi et al., 1996; Graziano, Hu, & Gross, 1997; Kurata, 1991; Neal, Pearson, & Powell, 1990a,b). Errors signalling the discrepancy between the final (rather than during the reach) finger position and the target location are relayed to PMv and used to override goal specification provided by PPC sensorimotor transformations in order to bring about a new reach that will bring the hand closer to the target on the next trial. This aspect of the model is crucial. Prism adaptation cannot be achieved within one trial and must be achieved by bringing about changes in response to endpoint errors on each subsequent trial.

How does the current data from JJ fit into this model? Remember that JJ was impaired at strategic control, but not spatial realignment, with his right hand and vice versa with his left hand. Remember also that JJ has asymmetric parietal damage and substantial white matter damage so that any links drawn here between the model and JJ’s damage are both possible and purely speculative. Looking at Fig. 5 it can be seen that a lesion that impairs PPC goal specifications or the bi-directional communication of that information to PMv would mean that no matter how much JJ tried to reach away from fixation, he would not be able to do so. Endpoint error information, however, could still be relayed to the cerebellum via PMv. Thus, the cerebellar-PMv loop has the information necessary to start spatial realignment, but the parietal circuits are unable to implement the side-pointing that strategic control demands.

In contrast, disruption of the link between PPC error detection and PMv could account for the data from his left hand. Disruption of this link would deprive the cerebellar-PMv loop of the information necessary to initiate realignment, but strategic control would still be possible. Such strategic control, without effective error information however, would be rather aimless and inefficient. JJ’s left hand performance could be described in these terms: his errors reduce initially, but when that doesn’t work the strategy is abandoned or replaced and errors veer away again. His on-line control should be unaffected, however, and previous studies suggest that this may be the case (Newport et al., in press).

In any case, any online corrections could not possibly make JJ accurate though as there would not be time under terminal exposure conditions to correct his enormous initial errors. Further testing of this model is clearly required including examination of JJ’s white matter connections using diffusion tensor imaging.

In conclusion, the current experiment highlighted the dissociable strategic and realignment processes of prism adaptation in a single patient with bilateral posterior parietal damage. Although the precise nature of its contribution is as yet unclear, this study presents supporting evidence for the involvement of PPC in prism adaptation. The data also indicates that, like the ability to perform on-line corrections, the ability to implement control strategies may not be necessary for successful adaptation to prisms.

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