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RESEARCH ARTICLE

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After-effects of visuo-manual adaptation to prisms on body posture in normal subjects

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Abstract Postural equilibrium is known to be controlled by sensorimotor reflexes and automatic control loops but also depends on high-level body representation in space, probably implicating the right temporoparietal cortex. Indeed, short-term prism adaptation to a 10° rightward visual shift has been shown to reduce predominant postural imbalance in patients with right hemisphere damage, as it did for neglect symptoms. These effects are likely to be explained by a high level effect of prism adaptation on body and space representation, rather than by a sensorimotor effect. Cognitive after-effects of prism adaptation to a leftward visual shift, suggesting neglect-like symptoms, have also recently been shown in normal subjects on line bisection tasks. In the present study, we investigated the effect of wedge prism adaptation on postural control in normal subjects. Two groups of seven healthy subjects were either adapted to a leftward or a rightward visual shift. Results showed that our procedure induced changes in lateral postural control in normal subjects. Furthermore, this lateral postural after-effect was dependent on direction of prism adaptation. Indeed, only adaptation to a leftward visual shift induced significant rightward postural bias in normal subjects. The rightward postural lateral displacement was negatively correlated with the visual vertical. Both transfer and direction specific effect of visuo-manual adaptation to prisms on postural control suggest that effects of adaptation act more on high-level postural control linked to body representation in space or at least reveal close interaction between sensorimotor plasticity and body representation.

Keywords Prism adaptation · Space representation · Postural control · Body representation · Subjective visual vertical

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Introduction

Symmetrical sensorimotor after-effects of wedge prism adaptation are commonly described in classical literature (see Welch 1986). However, recent results in neglect patients raise a new issue about the cognitive nature of prism after-effects (for review see Rossetti and Rode 2002). Indeed, it has recently been shown that a single, short adaptation period of a 10° rightward prismatic shift of the visual field improves various cognitive symptoms of patients with unilateral spatial neglect for at least several days (Farné et al. 2002; McIntosh et al. 2002; Pisella et al. 2002; Rossetti et al. 1998, 1999). Unilateral spatial neglect is a neuropsychological disorder commonly observed following right hemisphere injury. Neglect of contralesional space is characterized by a failure to report, respond to, or orient to novel or meaningful stimuli, even when this deficit cannot be attributed to either sensory or motor defects (Halligan and Cockburn 1993; Heilman and Valenstein 1979). For many authors, neglect might be attributed to a distortion of space representation linked to dysfunction of right posterior parietal cortex (Bisiach et al. 1983). Sensorimotor manifestations of this condition, such as the rightward displacement of the perceived midline with respect to the body, are improved by prism adaptation (Rossetti et al. 1998). Furthermore, prism adaptation is able to markedly improve cognitive levels of neglect as assessed by standard neuropsychological tests such as line bisection, line cancellation (Pisella et al. 2002; Rossetti et al. 1998) and simple drawing from

memory, as well as on non-manual tests such as reading, object recognition (Farné et al. 2002; Rossetti et al. 1999) or even mental imagery (Rode et al. 1999, 2001, 2003).

In parallel, adaptation to a 10° rightward prismatic shift improves postural symptoms in right brain-damaged patients with left hemiparesia (Tilikete et al. 2001). These patients show predominantly lateral displacement of the centre of pressure toward the ipsilesional side relative to right hemiparetic patients (Rode et al. 1997, 1998). This asymmetry might be partly attributed to distortion of body representation linked to the right posterior parietal lesion (Pérennou et al. 2000). Indeed, several authors have proposed that posture is not only controlled by a system based on reflex and automatic action (Kolb et al. 2001; Massion and Viallet 1990) but also depends on higher level functions through the elaboration of an internal model of the body (Gurfinkel and Levick 1991; Timmann and Horak 2001). The body scheme is progressively built up from sensory and motor experiences; nevertheless, several pieces of evidence suggest that body representation is partly governed by implicit knowledge of body structure (Berlucchi and Aglioti 1997). One example is the phantom sensations reported by phocomelic children who were born without one or more limbs (Melzack 1990). Even though the limbs of phocomelic children did not develop physically, they are nevertheless represented in sensory and motor areas (Brugger et al. 2000). Furthermore, imitation of the movements by neonates suggests an innate knowledge of the body that antedates the adult body scheme (Berlucchi and Aglioti 1997).

The above-reviewed results obtained from neglect and left hemiparetic patients show that prism adaptation can induce improvements of neurological deficits resulting from a right brain lesion. In addition to sensorimotor after-effects (such as subjective perception of the body midline), the improvement of cognitive neglect manifestations (Rode et al. 1999) and postural control in patients with right brain damage (Tilikete et al. 2001) suggest an effect of prism adaptation on internal maps used for space and body representation. Common mechanisms for, or at least close interaction between, sensorimotor plasticity and higher levels of space and body representation (Rossetti et al. 1999) may explain this set of concordant results. Along this line, if prism adaptation acted on the body scheme by modifying body representation, a postural adjustment should be observed not only in right brain damage patients with postural deficits (Tilikete et al. 2001) but also in normal subjects. This hypothesis could be sustained by the modification of space representation in normal subjects. Indeed, evidence for cognitive after-effects of prism adaptation has already been provided by studies in normal subjects performing line bisection tasks (manual bisection and Landmark task). Prism adaptation to a leftward optical shift emulates moderated neglect-like symptoms in the form of rightward biases measured by line bisection tasks (Berberovic and Mattingley 2002; Colent et al. 2000; Michel et al. 2002).

In the present study we investigated the effect of prism adaptation on postural control in normal subjects by recording the projection of the centre of pressure and subjective visual vertical. If after-effects of prism adaptation on postural control were sensorimotor then a symmetrical bias would be expected. However, if there were an effect on body scheme then the main effect would be a rightward postural bias after adaptation to a leftward optical shift. The following experiments expand on the exploration of after-effects of prism adaptation in normal subjects. We aimed not only at uncovering properties of the normal system and further exploring the properties of the spatial maps altered by prism adaptation, but also at the better understanding of the nature of the internal maps involved in postural maintenance.

Material and methods

Subjects

Fourteen right-handed and normal-sighted healthy subjects participated in the experiment. The group exposed to a rightward optical deviation was composed of three females and four males ranging in age from 17 to 42 years (mean 27 years, SE 3.0 years), and the group exposed to a leftward optical deviation was composed of four females and three males ranging in age from 21 to 28 years (mean 24 years, SE 0.8 years). All subjects gave their informed consent prior to their inclusion in the study, in accordance with the local ethics committee.

Experimental procedure

Posturographic evaluation

The posturographic evaluation was performed with a Dynatronic statokinesimeter. It consisted of a statokinesimetric platform measuring 45×45 cm supported by three strain gauges. The room was normally illuminated and a 75-cm long rod, placed 60 cm in front of the subject (64° angle of vision), was presented on the vertical axis and aligned with body midline. The available visual cues were the vertical rod and the vertical wall of the box in which the posturographic evaluation was performed. During the test, subjects stood barefoot on the platform in the upright position with their arms alongside their body and with their feet placed 10 cm apart on the designed site, centred in relation to the antero-posterior and lateral axis. The analogue signal from the three gauges, digitalized at a sampling frequency of 5 Hz for 53 s, was used online to calculate the successive positions of the centre of pressure along the forward-backward (Y-axis) and the left–right axis (X-axis). Three main parameters were extracted from these data: the mean lateral projection of the centre of pressure (Mean X), the mean antero-posterior projection of the centre of pressure (Mean Y) and the surface area (S). For Mean X, a rightward bias from mid-sagittal plane was assigned a positive value and a leftward bias was assigned a negative value. For Mean Y, a forward bias from frontal plane was assigned positive value and a backward bias a negative value. Surface area was a measure of the area of the confidence ellipse of the distribution of the centre of pressure. Two sessions were performed: in the first one, subjects kept their eyes closed (closed-eye condition), and in the second they kept their eyes open (open-eye condition). Postural measures were recorded before (pre-test) and after (post-test) prism adaptation.

Subjective visual vertical (SVV)

The visual perception of verticality was recorded for all subjects. Using a remote control, subjects were asked to align a 31-cm long

phosphorescent rod with their subjective perception of the verticality (12 trials). The rod was placed 175 cm from the subjects' eyes (10° angle of vision). Before each trial, the phosphorescent rod was presented alternately with various rightward or leftward angular deviations from the vertical within the frontal plane by the examiner. The task was performed in total darkness before (pre-test) and after (post-test) prism adaptation.

Sensorimotor effects

In order to measure sensorimotor after-effects, effective development of the adaptation was assessed with two tasks performed before (pre-test) and after (post-test) prism adaptation. (1) In the "open-loop" pointing task, subjects were required to point toward visual targets with the right hand and without visual feedback (total after-effect). The total lateral error from the visual target (TLE) was the parameter recorded in this task. (2) In the proprioceptive estimation of body midline task (proprioceptive after-effect), the subjects had to point with the right hand in alignment with their mid-sagittal axis in darkness (e.g. see Kornheiser 1976; Rossetti et al. 1998). The proprioceptive lateral error from the mid-sagittal axis (PLE) was the parameter calculated in this task.

Prism adaptation

Following the pre-tests, subjects sat at a table and wore prismatic goggles producing a shift of the visual field. With goggles on, the total visual field was 105°, the monocular visual field was 75° and the binocular visual field was 45°. The relative locations of the visual stimuli were conserved whereas the absolute location was shifted in the direction of the optical deviation. Subjects were asked to point, as fast and as accurately as possible, with the right arm to each of the ten visual targets that were placed on the table equitably distributed relative to the subject's body midline. The adaptation procedure lasted 20 min during which the subjects wore prisms producing a 15° (leftward or rightward) lateral visual shift. Vision of the starting position of the hand was occluded to ensure optimal development of the adaptation (Redding and Wallace 1997a). This procedure was identical to that used in a previous study (Colent et al. 2000). Seven subjects were exposed to a 15° leftward visual shift (group L) and seven others to a 15° rightward visual shift (group R).

The sensorimotor effects, the posturographic data and the subjective visual vertical were evaluated in that sequence before and after prism exposure. Subjects were asked to keep their eyes closed between trials and were transported in a wheelchair between the table (used for prism adaptation) and the statokinesimeter in order to minimize de-adaptation.

Statistical analysis

To estimate the after-effects of prism adaptation we analysed the TLE and the PLE of subjects using a two-way analysis of variance. This analysis evaluated the condition (pre-test and post-test) as within-subject factors, and the effect of the group (group L and group R) as between-subjects factors. The specific effects of each factor were analysed with least significant difference (LSD) post hoc comparison.

To evaluate the effect of prism adaptation on postural equilibrium in groups L and R, we performed a two-way analysis of variance for S, Mean X and Mean Y with eyes open and eyes closed. The analysis evaluated the condition (pre-test and post-test) as within-subject factors, and the effect of the group (group L and group R) as between-subjects factors. The specific effects of each factor were analysed with LSD post hoc comparison.

In both groups, a correlation analysis was performed between the effects of prism adaptation and all others parameters.

All statistics were performed by the STATISTICA software package (release 4.5, 1993; StatSoft, Tulsa, OK, USA). The 95% confidence interval was used to establish statistical significance. Mean and standard error are presented in parentheses.

Results

Open-loop pointing task

The statistical analysis revealed a significant group effect on TLE [$F_{(1,12)}$=25.8, P<0.001], and a group × 'pre-post' interaction [$F_{(1,12)}$=39.7, P<0.0001]. As expected, group L showed a significant rightward TLE (mean ±SE 13.7±2.7°) during the open-loop pointing task when the post-test (11.8±2.1°) and the pre-test (–1.9±1.3°) values were compared (P<0.001). Group R exhibited a significant leftward TLE (–14.4±3.6°) when the post-test (–13.1±3.2°) and the pre-test (1.3±1.5°) values were compared (P<0.007). Sensorimotor after-effects of prism adaptation, observed in open loop pointing task, were symmetrical; they had similar amplitudes (P>0.8) and were each oriented in the direction opposite to the optical deviation.

Proprioceptive straight-ahead task

Statistical analysis revealed a significant group × pre-post interaction on PLE [$F_{(1,12)}$=15.14, P<0.005]. A significant rightward PLE was detected in group L (13.3±4.6°) when the post-test (10.3±4.6°) and the pre-test (–3.0±1.7°) values were compared (P<0.015). Symmetrically, a leftward PLE (–9.9±3.6°) was observed in group R when the post-test (–7.1±3.1°) and the pre-test (2.8±4.6°) were compared (P<0.03). The sensorimotor after-effects assessed by the proprioceptive straight-ahead demonstration task, were symmetrical; they had similar amplitudes (P>0.5) and were oriented in the direction opposite to the optical deviation.

Postural after-effects

Surface area (S)

Closed-eye condition. The statistical analysis revealed a significant pre-post effect on S [$F_{(1,12)}$=6.70, P<0.03] without group effect. For group L, S was 231±29 mm^2 during the pre-test and 296±45 mm^2 during the post-test. For group R, S was 257±58 mm^2 during the pre-test and 385±94 mm^2 during the post-test. Therefore, prism adaptation induced a symmetrical increase of the surface area in the two groups for the closed-eye condition.

Open-eye condition. There was no group effect, no pre-post effect and no significant interaction (P-values >0.3).

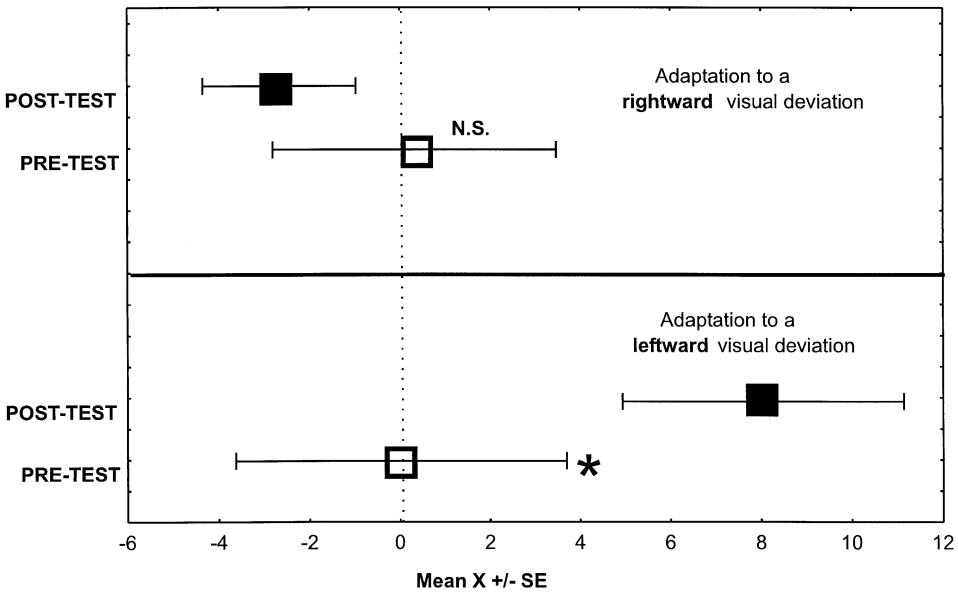

Fig. 1 Mean lateral displacement of the centre of pressure (Mean X, in mm) before (pre-test) and after (post-test) the prism adaptation procedure in groups adapted either to a leftward or rightward visual deviation, under the closed-eye condition. A significant lateral displacement was observed only in the group adapted to a leftward optical deviation. *SE* standard error, *N.S.* non-significant ($P>0.05$), *significant ($P<0.05$)

Mean X

Closed-eye condition. Statistical analysis revealed a significant group × pre-post interaction [$F_{(1,12)}=9.90$; $P<0.009$] (Fig. 1). For group L, Mean X was shifted significantly to the right ($P<0.008$) (Fig. 2). The rightward shift (8.0±2.9 mm) observed between the post-test (8.0±3.1 mm) and the pre-test (0.0±3.6 mm), ranged from –0.1 mm to 23.5 mm. For group R no significant effect was observed ($P>0.23$). Mean X was localized at 0.4±3.1 mm in the pre-test and at –2.7±1.8 mm in the post-test.

Open-eye condition. No pre-post effect, no group effect and no interaction was observed (P-values >0.9). For group L, Mean X was 4.2±2.7 mm in the pre-test and 7.1±2.8 mm in the post-test. For group R, Mean X was 0.6±2.0 mm in the pre-test and –0.24±1.7 mm in the post-test.

Therefore, the effect of prism adaptation on Mean X position was asymmetrical: only adaptation to a leftward optical deviation induced a rightward postural imbalance in experimental closed-eye condition (Fig. 3). This result provides support for a direction-specific effect of prism adaptation on postural control in the frontal axis in normal subjects.

Mean Y

Closed-eye condition. There was a significant pre-post effect without group effect. Mean Y was shifted forward after prism adaptation [$F_{(1,12)}=5.87$, $P<0.04$]. For group L, Mean Y was –27.5±6.9 mm in the pre-test and –21.1±8.4 mm in the post-test. For group R, Mean Y was –33.1±4.8 mm in the pre-test and –27.6±5.9 mm in the post-test.

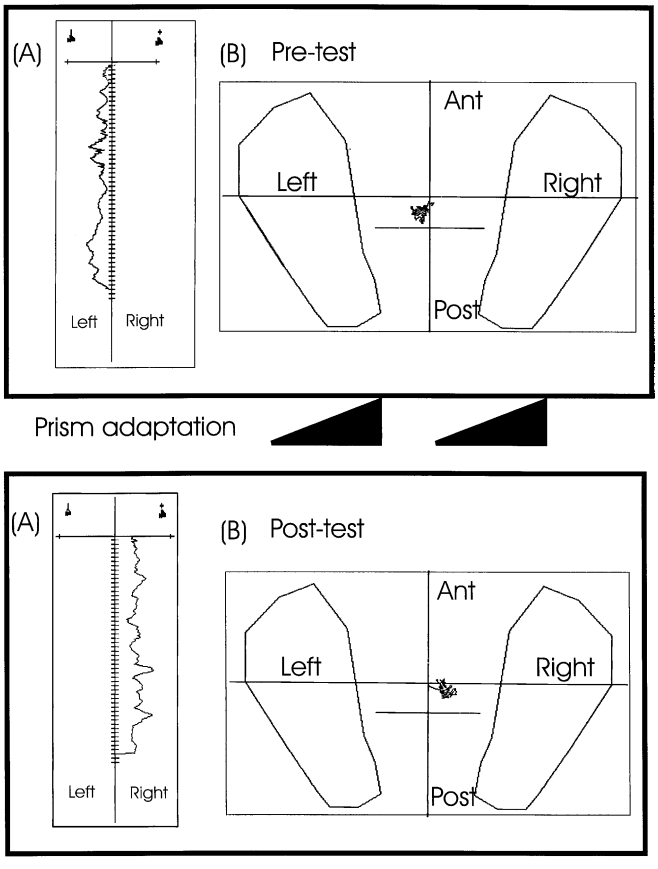

Fig. 2A–B Displacement of the centre of pressure on the lateral axis (**A**, in cm) and projection of the centre of pressure on the ground (**B**) for one subject adapted to a leftward optical deviation, before (pre-test) and after (post-test) adaptation under the closed eye condition. Note the rightward deviation of the projection of the centre of pressure in the post-test (*Ant* anterior, *Post* posterior)

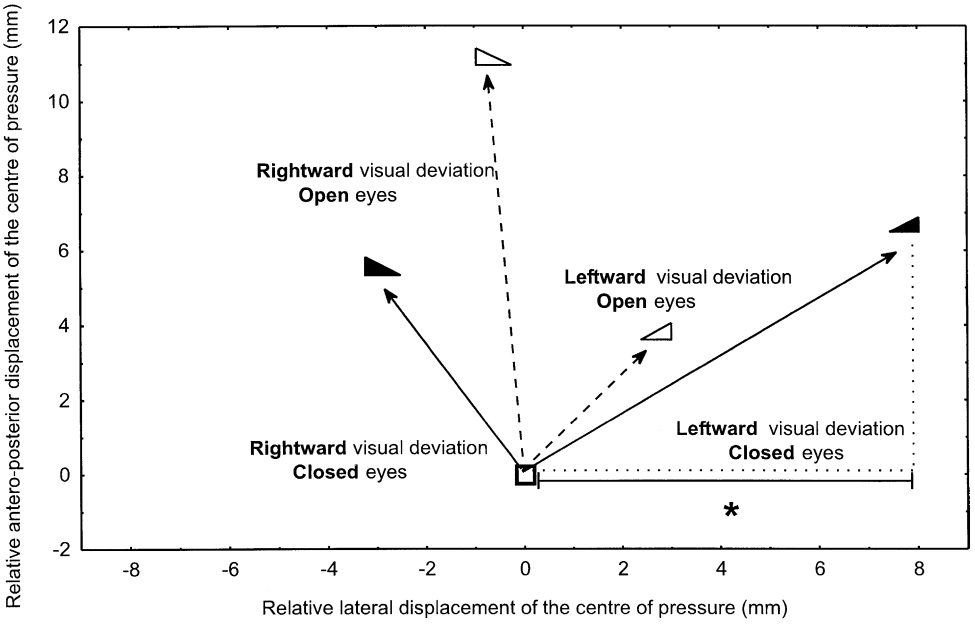

Fig. 3 Relative displacement of mean position of the centre of pressure in groups adapted either to a leftward or rightward visual deviation in the two experimental conditions (eyes open and eyes closed). Performances in the pre-test were undifferentiated between groups or experimental condition ($P>0.45$). There was a global significant forward effect in both rightward and leftward visual group for both conditions. Note the significant rightward asymmetrical lateral effect in the leftward visual group in the closed-eye condition (*$P<0.05$)

Open-eye condition. There was a significant pre-post effect without group effect. Mean Y was shifted forward after prism adaptation [$F_{(1,12)}=9.22$, $P<0.02$]. In the group L, Mean Y was localized at –30.2±7.6 mm in the pre-test and at –26.2±8.2 mm in the post-test. For group R, Mean Y was –35.6±5.6 mm in the pre-test and –24.5±4.3 mm in the post-test.

There was a constant forward displacement on Mean Y after prism adaptation, irrespective of the direction of the optical shift (rightward versus leftward) and the experimental condition (closed or open eyes) (Fig. 3).

Subjective visual vertical (SVV)

The statistical analysis revealed no effect of group (L versus R), no pre-post condition effect and no group × pre-post condition interaction ($P>0.08$). However, in group L the correlation analysis revealed a significant negative correlation between the mean effect of prism adaptation on X-axis and the effect of prism adaptation on SVV (correlation coefficient –0.90, $P<0.05$). Whereas the mean position of the centre of pressure was laterally shifted to the right after adaptation to a leftward optical deviation, the SVV was shifted counterclockwise (–0.26±0.18°; pre-test –0.43±0.25°, post-test –0.69±0.36°).

Discussion

Whereas sensorimotor after-effects of prism adaptation have been extensively described (e.g. Redding and Wallace 1997b, Welch 1986), it is only recently that several cognitive after-effects have been demonstrated in unilateral neglect patients (reviewed in Rossetti and Rode 2002) and, to a lesser extent, in normal subjects during line bisection tasks (Berberovic and Mattingley 2002; Colent et al. 2000; Michel et al. 2002). The present work investigated whether after-effects of wedge prism adaptation can extend to the postural control of normal subjects, as has been reported for patients with right hemisphere lesions (Tilikete et al. 2001). The main results of the present study showed that wearing prisms that shift the vision horizontally during a pointing task with the right arm might alter the lateral postural control in normal subjects. Furthermore, the lateral postural bias was dependent on direction of the prism adaptation. Indeed, only adaptation to a leftward (not to a rightward) visual shift induced a significant rightward shift of the vertical projection of the centre of pressure, which was negatively correlated with the visual vertical. The secondary results showed an increased surface area and a forward displacement of the body weight, in both rightward and leftward group conditions, which might be explained by a postural compensation to the backward postural adjustments required during the pointing movements performed with the prisms or by a reaction to the slight 'magnifying' effect of the prisms. Another secondary observation was that postural after-effects of prism adaptation were observed only in the closed-eye condition. Visual cues are known to be crucial for the control of body posture (Paulus et al. 1984). In our experiment, vertical cues available in the environment when the eyes were open may explain absence of significant effects with eyes open. The discussion following will attempt to provide explanations for our main results. The discussion is based on three principal points: (1) the extension of current literature of prism adaptation transfer, (2) the link between the present asymmetrical results in normal subjects and the symptoms known in right brain-damaged patients, and (3) the anatomical hypothesis to explain how

prism adaptation could qualitatively simulate right-brain damage deficits in normal subjects.

These present results extend the current literature about prism adaptation on several points. Firstly, for many years the after-effects of prism adaptation in normal subjects have been found to have incomplete or no generalization across different tasks. For example, adaptation with head fixed cannot be generalized to the contralateral arm (Taub and Goldberg 1973), and can even be specific to the velocity (Kitazawa et al. 1997) or to the type of arm movement performed during the exposure period (Martin et al. 1996b). Nevertheless, experimental conditions with head free may produce inter-manual (e.g. Choe and Welch 1974; Redding and Wallace 1988) or inter-limb (Elliot and Roy 1981) transfer. Some other experiments also showed inter-modal after-effects of visuo-manual adaptation such as auditory mislocation (Lackner 1973, 1976). As for transfer of prism adaptation across different tasks, interestingly our results showed that a manual adaptation to a visual shift could transfer significantly to the postural system. Secondly, sensorimotor after-effects of wedge prism adaptation are known to be symmetrical (see Kornheiser 1976; Redding and Wallace 1992; Welch 1986). However, our results show that the postural after-effects of prism adaptation are asymmetrical and thus of a specific nature. These effects appear significant only after adaptation to a leftward optical deviation and are rightward oriented. Both transfer and direction-specific effects of prism adaptation on postural control suggest that effects of adaptation arise more from higher levels of postural control rather than from lower level sensorimotor reflexes. These results extend on our previous work (Colent et al. 2000; Michel et al. 2002) by showing that adaptation to a leftward optical shift modifies not only the extrapersonal space representation but also the body representation at a high level of processing.

The links between the present asymmetrical results in normal subjects and the symptoms known to exist in right-brain-damaged patients need to be discussed now. Firstly, the holistic processing of sensory information for the control of body postural activities has been attributed to the right parietal cortex (Pérennou et al. 1997). Furthermore, clinical analyses show greater postural difficulties after right hemisphere damage than after left hemisphere lesion (Bohannon et al. 1986; Rode et al. 1997). More precisely, the right temporoparietal junction (TPJ) has been found to markedly contribute to body stability by composing internal models (Pérennou et al. 2000), by resolving sensory ambiguities, synthesising information from disparate sensory modalities and combining efferent and afferent information (Mergner et al. 2001). In neglect patients, where damage predominates in the right parieto-occipito-temporal junction, there is a dramatic postural instability (Pérennou et al. 2000) and an ipsilateral rightward displacement during standing. A similar but moderate rightward postural bias was observed in our experiment in normal subjects after adaptation to a leftward visual shift, suggesting an influence, at least indirectly, of prism adaptation on the right parietal cortex.

Secondly, to regulate the postural control of the body, the central nervous system must rely on an internal representation of the vertical axis. Clinical studies show that deficits of general spatial direction are more frequent and more severe following right hemisphere damage than after left hemisphere lesion (Benton et al. 1975; Taira et al. 1998). The perception of the subjective visual vertical is oriented in the opposite direction to the lesion (Brandt et al. 1994). Indeed, neglect patients show a counter-clockwise perception of the vertical orientation (Kerkhoff 1999), negatively correlated with the postural bias in upright position. Even if pre-post measurement did not show any significant change in subjective visual vertical, our results in normal subjects reinforce these data since the rightward bias in upright posture was negatively correlated with the subjective visual vertical after a visuo-motor adaptation to a leftward optical shift.

Thirdly, neglect seems to be associated with failure to attend to the body scheme (Pérennou et al. 2001), which suggests that neglect is in part attributable to a deficit of spatial cognition (Halligan 1995; Rode et al. 1999, 2003; Rossetti et al. 1998). A recent study by Richard et al. (2000) investigating extrapersonal projection of the body in neglect patients suggests a compression of the body projection in extracorporeal space in which the projection of the left hemi-body is narrower than the right one. Such a remapping of the body scheme in space leading to "an amputation of the left part of the 'normal' body scheme" transiently induced by prism adaptation in normal subjects might be responsible for postural neglect-like imbalance.

With our results showing qualitative simulation of right brain deficit in normal subjects, we have now to consider the anatomical substrates that could explain how adaptation to a leftward optical deviation can modify inter-hemispheric balance to the detriment of the right hemisphere. Anatomical structures involved in prism adaptation are not well known. The involvement of the cerebellum in prism adaptation is crucial (e.g. see review of Jeannerod and Rossetti 1993; Thach et al. 1992; Weiner et al. 1983). Prism adaptation in humans and monkeys is abolished by disruption of visual afferents to the cerebellum (Baizer et al. 1999; Martin et al. 1996a). Whereas the parietal cortex is not necessary for prism adaptation (L. Pisella, C.Michel, C. Tilikete, A. Vighetto and Y. Rossetti, submitted for publication), it has been shown in brain imaging studies to be involved during the prism adaptation process (Clower et al. 1996) and in after-effects (Sekiyama et al. 2000). It is now necessary to consider how the action of prism adaptation at a cerebellar level could produce a parietal-like deficit (as in the present experiment), and how it can be responsible for a therapeutic effect on neglect patients (reviewed in Rossetti and Rode 2002). Firstly, anatomical connections from the cerebellum to the parietal cortex underlying the recalibration during the adaptive process have been recently demonstrated (Clower et al. 2001). Secondly, a

recent positron emission tomography (PET) investigation showed that the parietal cortex and cerebellum seem to be involved in the therapeutic effect of prism adaptation in neglect patients (Luauté et al. 2002). Therefore, specific cerebello-parietal pathways could provide an anatomical substrate to enable higher level effects of prism adaptation (Rossetti and Rode 2002; Schmahmann 1998). Apart from parietal cortex, lesions of ventral premotor cortex have been shown to reduce the development of adaptation in monkeys (Kurata and Hoshi 1999). Cerebellar projections to pre-motor areas of the frontal lobe may also represent part of the neuronal substrate for prism adaptation (Kurata and Hoshi 1999).

At a neuronal level, effects of prism adaptation could be mediated by polymodal neurons of the parieto-temporal cortex (Bremmer et al. 2001). In the monkey, neurons of the temporoparietal junction (TPJ), classified as polymodal vestibular units responding both to vestibular and somatosensory stimulations (Grüsser et al. 1990a, 1990b), could provide the anatomical substrate for transfer of prism adaptation to postural control. Furthermore, we have to consider the possible influence of prism adaptation interfering with projections of the parietal cortex onto primary target areas via feedback projections (Hyvarinen 1982) to modify the elementary processing of sensory information. Indeed, it as been shown that mechanisms of neuronal plasticity might even involve the primary visual cortex in monkeys (Sugita 1996).

Possible mechanisms of the postural after-effect induced by prism adaptation will have to be considered in further studies. For example, the interaction between the direction of the visual shift and the hand used for pointing might be addressed by measuring the postural after-effect induced by prism adaptation when the left hand is used.[1]

Conclusion

Our study has shown that after-effects of a visuo-manual adaptation to a leftward lateral shift of the visual field could be extended to the postural control in normal subjects. These results support the idea that postural control in humans depends on both sensory inputs and an inner postural body scheme. The plasticity of inter-sensory and sensorimotor co-ordination involved during prism adaptation may thus affect higher-level representations of extrapersonal and internal body space.

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References

Baizer JS, Kralj-Hans I, Glickstein M (1999) Cerebellar lesions and prism adaptation in macaque monkeys. J Neurophysiol 81:1960–1965

Benton A, Hannay HJ, Varney NR (1975) Visual perception of line direction in patients with unilateral brain disease. Neurology 25:907–910

Berberovic N, Mattingley JB (2002) Effects of prismatic adaptation on judgements of spatial extent in peripersonal and extrapersonal space. Neuropsychologia (in press)

Berlucchi G, Aglioti S (1997) The body in the brain: neural bases of corporeal awarness. Trends Neurosci 20: 560–564

Bisiach E, Bulgarelli C, Sterzi R, Vallar G (1983) Line bisection and cognitive plasticity of unilateral neglect of space. Brain Cogn 2:32–38

Bohannon RW, Smith MB, Larkin PA (1986) Relationship between independent sitting balance and side of hemiparesis. Phys Ther 66:944–945

Brandt T, Dieterich M, Danek A (1994) Vestibular cortex lesions affect the perception of verticality. Ann Neurol 35:403–412

Bremmer F, Schlack A, Duhamel JR, Graf W, Fink GR (2001) Space coding in primate posterior parietal cortex. Neuroimage 14:S46–S51

Brugger P, Kollias SS, Muri RM, Crelier G, Hepp-Reymond MC, Regard M (2000) Beyond re-membering: phantom sensations of congenitally absent limbs. Proc Natl Acad Sci USA 97:6167–6172

Choe CS, Welch RB (1974) Variables affecting the intermanual transfer and decay of prism adaptation. J Exp Psychol 102:1076–1084

Clower D, Hoffman JM, Votaw JR, Faber TL, Woods RP, Alexander GE (1996) Role of posterior parietal cortex in the recalibration of visually guided reaching. Nature 383:618–621

Clower DM, Robert AW, Lynch JC, Strick PL (2001) The inferior parietal lobule is the target of output from the superior colliculus, hippocampus and cerebellum. J Neurosci 21:6283–6291

Colent C, Pisella L, Bernieri C, Rode G, Rossetti Y (2000) Cognitive bias induced by visuo-motor adaptation to prisms: a simulation of unilateral neglect in normal individuals? Neuroreport 11:1899–1902

Elliot D, Roy EA (1981) Interlimb transfer after adaptation to visual displacement: patterns predicted from the functional closeness of limb neural control centers. Perception 10:383–389

Farné A, Rossetti Y, Toniolo S, Làdavas E (2002) Ameliorating neglect with prism adaptation: visuo-manual and visuo-verbal measures. Neuropsychologia 40:718–729

Grüsser OJ, Pause M, Schreiter U (1990a) Localization and responses of neurons in the parieto-insular vestibular cortex of awake monkeys. J Physiol 430:537–557

Grüsser OJ, Pause M, Schreiter U (1990b) Vestibular neurons in the parieto-insular cortex of monkeys: visual and neck receptor. J Physiol 430:559–583

Gurfinkel VS, Levick YS (1991) Perceptual and automatic. aspects of the postural body scheme In Paillard J (ed) Brain in space. Oxford University Press, Oxford, pp 147–162

Halligan PW (1995) Drawing attention to neglect. The Psychologist, June 1995, pp 257–264

Halligan PW, Cockburn JM (1993) Cognitive sequelae of stroke; visuo-spatial and memory disorders. Crit Rev PhysRehabil Med 5:57–81

Heilman KM, Valenstein E (1979) Mechanisms underlying hemispatial neglect. Ann Neurol 5:166–170

Hyvarinen J (1982) Posterior parietal lobe of the primate brain. Physiol Rev 62:1060–129

Jeannerod M, Rossetti Y (1993) Visuo-motor coordination as a dissociable function: experimental and clinical evidence. In: Kennard C (ed) Visual perceptual defects. Baillère's clinical neurology, international practice and research. Ballière Tibdall, London, pp 439–460

Kerkhoff G (1999) Multimodal spatial orientation deficits in left-sided visual neglect. Neuropsychologia 37:1387–1407
Kitazawa S, Kimura T, Uka T (1997) Prism adaptation of reaching movements: specificity for the velocity of reaching. J Neurosci 17:1481–1492
Kolb FP, Lachauer S, Diener HC, Timmann D (2001) Changes in conditioned postural responses. Comparison between cerebellar patients and healthy subjects. Acta Physiol Pharmacol Bulg 26:143–146
Kornheiser AS (1976) Adaptation to laterally displaced vision: a review. Psychol Bull 83:783–816
Kurata K, Hoshi E (1999) Reacquisition deficits in prism adaptation after muscimol microinjection into the ventral premotor cortex in monkeys. J Neurophysiol 81:1927–1938
Lackner JR (1973) The role of posture in adaptation to visual rearrangement. Neuropsychologia 11:33–44
Lackner JR (1976) Influence of abnormal postural and sensory conditions on human sensorimotor localization. Environ Biol Med 2:137–177
Luauté J, Michel C, Rode G, Boisson D, Halligan PW, Costes N, Lavenne F, Rossetti Y (2002) Cerebral plasticity induced by prismatic adaptation in neglect patients. A PET study. In: Proceedings of Third World Congress in Neurological Rehabilitation, Venice, Italy, 3–6 April 2002
Martin TA, Keating JG, Goodkin HP, Bastian AJ, Thach WT (1996a) Throwing while looking through prisms. I. Focal olivocerebellar lesions impair adaptation. Brain 119:1183–1198
Martin TA, Keating JG, Goodkin HP, Bastian AJ, Thach WT (1996b) Throwing while looking through prisms. II. Specificity and storage of multiple gaze-throw calibrations. Brain 119:1199–1211
Massion J, Viallet F (1990) Posture, coordination, movement. Rev Neurol (Paris) 146:536–542
McIntosh RM, Rossetti Y, Milner AD (2002) Prism adaptation improves chronic visual and haptic neglect. Cortex 38:309–320
Melzack R (1990) Phantom limb and the concept of a neuromatrix. Trends Neurosci 13:88–93
Mergner T, Nasios G, Maurer C, Becker W (2001) Visual object localisation in space. Interaction of retinal, eye position, vestibular and neck proprioceptive information. Exp Brain Res 141:33–51
Michel C, Pisella L, Halligan PW, Luauté J, Rode G, Boisson D, Rossetti Y (2002) Simulating unilateral neglect in normals using prism adaptation: implications for theory. Neuropsychologia DOI 10.1016/s0028-3932(02)00135-5 (in press)
Paulus WM, Straube A, Brandt T (1984) Visual stabilisation of posture. Physiological stimulus characteristics and clinical aspects. Brain 107:1143–1163
Pérennou DA, Amlard B, Laassel EM, Pélissier J (1997) Hemispheric asymmetry in the visual contribution to postural control in healthy adults. Neuroreport 8:3137–3141
Pérennou DA, Leblond C, Amblard B, Micallef JP, Rouget E, Pélissier J (2000) The polymodal sensory cortex is crucial for controlling lateral postural stability: evidence from stroke patients. Brain Res Bull 53:359–365
Pérennou DA, Leblond C, Amblard B, Micallef JP, Hérisson C, Pélissier J (2001) Transcutaneous electric nerve stimulation reduces neglect-related postural instability after stroke. Arch Phys Med Rehabil 82:440–448
Pisella L, Rode G, Farné A, Boisson D, Rossetti Y (2002) Dissociated long lasting improvements of straight-ahead pointing and line bisection tasks in two hemineglect patients. Neuropsychologia 40:327–334
Redding GM, Wallace B (1988) Components of prism adaptation in terminal and concurrent exposure: Organization of the eye-hand coordination loop. Percept Psychophys 44:59–68
Redding GM, Wallace B (1992) Adaptative eye-hand coordination: implications of prism adaptation for perceptual-motor organization. In: Proteau L, Elliot D (eds) Vision and motor control. Elsevier, Amsterdam, pp 105–127
Redding GM, Wallace B (1997a) Prism adaptation during target pointing from visible and non visible starting locations. J Mot Behav 29:119–130
Redding GM, Wallace B (1997b) Part II: Prism adaptation. Paradigm and generalizations (Chapter 4). In: Redding GM, Wallace B (eds) Adaptive spatial alignment. Lawrence Erlbaum Associates, Mahwah N. J. pp 53–65
Richard C, Honoré J, Rousseaux M (2000) Is there a distortion of the body projection in extracorporeal space in neglect patients? Neuroreport 11:3047–3051
Rode G, Tiliket C, Boisson D (1997) Predominance of postural imbalance in left hemiparetic patients. Scand J Rehabil Med 29:11–16
Rode G, Tiliket C, Charlopain P, Boisson D (1998) Postural asymmetry reduction by vestibular caloric stimulation in left hemiparetic patients. Scand J Rehabil Med 30:9–14
Rode G, Rossetti Y, Li L, Boisson D (1999) Improvement of mental imagery after prism exposure in neglect: a case study. Behav Neurol 11:1–8
Rode G, Rossetti Y, Boisson D (2001) Prism adaptation improves representational neglect. Neuropsychologia 39:1250–1254
Rode G, Pisella L, Rossetti Y, Farné A, Boisson D (2003). Bottom-up transfer of plasticity to the recovery: visuo-motor adaptation and spatial neglect. In: Pelisson D, Prablanc C, Rossetti Y (eds) Progress in Brain Research series: Neural control of space coding and action production. Elsevier, Amsterdam, (in press)
Rossetti Y, Rode G (2002) Reducing spatial neglect by visual and other sensory manipulations: non-cognitive (physiological) routes to the rehabilitation of a cognitive disorder. In: Karnath HO, Milner AD, Vallar G (eds) The cognitive and neuronal bases of spatial neglect. Oxford University Press, Oxford (in press)
Rossetti Y, Rode G, Pisella L, Farné A, Li L, Boisson D, Perenin MT (1998) Prism adaptation to a rightward optical deviation rehabilitates left hemispatial neglect. Nature 395:166–169
Rossetti Y, Rode G, Pisella L, Farné A, Li L, Boisson D (1999) Sensori-motor plasticity and cognition: prism adaptation can affect various levels of space representation. In: Grealy M Thomson JA (eds) Studies in perception and action. Erlbaum, New York, pp 265–269
Schmahmann JD (1998) Dysmetria of thought: clinical consequences of cerebellar dysfunction on cognition and affect. Trends Cogn Sci 2: 362–371
Sekiyama K, Myauchi S, Imaruoka T, Egusas H, Tashiro T (2000) Body image as a visuomotor transformation device revealed in adaptation to reversed vision. Nature 407:374–377
Sugita Y (1996) Global plasticity in adult visual cortex following reversal of visual input. Nature 380:523–526
Taira M, Kawashima R, Inoue K, Fukuda H (1998) A PET study of axis orientation discrimination. Neuroreport 9:283–288
Taub ET, Goldberg IA (1973) Prism adaptation: control of intermanual transfer by distribution of practice. Science 180:755–757
Thach WT, Goodkin HP, Keating JG (1992) The cerebellum and the adaptive coordination of movement. Ann Rev Neurosci 1992:403–442
Tilikete C, Rode G, Rossetti Y, Pichon J, Ling L, Boisson D (2001) Prism adaptation to rightward optical deviation improves postural imbalance in left hemiparetic patients. Curr Biol 11:1–20
Timmann D, Horak FB (2001) Perturbed step initiation in cerebellar subjects: 2. Modification of anticipatory postural adjustments. Exp Brain Res 141:110–120
Weiner MJ, Hallett M, Funkenstein HH (1983) Adaptation to lateral displacement of vision with patients with lesions of the central nervous system. Neurology 33:766–772
Welch RB (1986) Adaptation of space perception. In: Boff KR, Kaufman L, Thomas JP (eds) Handbook of perception and human performance, vol. 1. Sensory processes and perception. Wiley, New York, pp 24.1–24.45