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Running Head: PRISM ADAPTATION AND COVERT ATTENTION

Prism adaptation reduces the disengage deficit in right brain damage patients.

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This work was supported by a Heart and Stroke Foundation (HSF) of Canada Masters award and a Natural Sciences and Engineering Research Council (NSERC) of Canada Ph.D. award to C.S. and HSF Grant-in-Aid and NSERC Discovery Grants (#261628-03) and an NSERC Canada Research Chair (Tier II) awarded to J.D.

Abstract

Recent research has shown that prism adaptation alleviates some of the symptoms of neglect. Although prism adaptation can aid patients with neglect, the mechanisms underlying these benefits remain largely unknown. One way in which prisms may work is by altering attentional orienting mechanisms known to be impaired in neglect. To investigate this hypothesis we tested four right brain damaged patients (two with neglect) on a reflexive covert attention task before and after rightward prism adaptation and compared them to a group of healthy controls who underwent sham prism adaptation. Results demonstrated that rightward prism adaptation reduced both the rightward attentional bias, and the disengage deficit in patients with right brain damage irrespective of the presence of neglect.

Key Words: right hemisphere, parietal lobe, neglect, prism adaptation, covert attention, visuomotor adaptation

Introduction

Neglect is a common disorder resulting from right parietal lesions, in which patients fail to attend to stimuli or events on the left side [1]. Neglect is a debilitating disorder and its presence post stroke has been identified as a predictor of poor functional recovery [2]. As such, a great deal of research has focused on ways to rehabilitate neglect.

One technique for rehabilitating neglect is prism adaptation (PA) [3]. The patient is asked to make a series of pointing movements while wearing prismatic lenses that shift perception 10° to the right. Following PA, patients demonstrate remarkable improvements on clinical tests such as line bisection and figure copying [3, for a review see 4]. Subsequent studies have shown that PA improves postural imbalance, tactile perception, and mental imagery [5-8]. Finally, a recent study by Frassinetti and colleagues [9] found the effects of PA to be long-lasting.

To further investigate the mechanisms underlying PA researchers have begun to examine its effects in healthy individuals. Leftward PA in healthy individuals creates ‘neglect-like’ symptoms in line bisection and postural control [10,11]. That is, following leftward PA, healthy individuals bisect lines further to the right and shift their centre of mass further to the right. Although PA can reduce some symptoms of neglect and create neglect-like phenomenon in healthy individuals, the cognitive mechanisms underlying PA remain unknown.

One symptom of neglect is impaired visual attention such that patients are unable to attend to the left while at the same time demonstrating biases toward attending to the right [12]. Prism adaptation may alleviate some symptoms of neglect by modulating the ability to allocate and reallocate visual attention. One paradigm widely used to examine the effects of brain lesions on reflexive and voluntary visual attention is the covert orienting of visual attention task (COVAT)[13]. In the COVAT, participants fixate centrally while attending to peripheral

locations. In the reflexive COVAT non-informative (i.e. 50% valid), abrupt onset peripheral cues are used to automatically attract attention to one location or another. In contrast, the voluntary COVAT typically utilizes an informative (e.g. 80% valid), symbolic cue (e.g., a centrally presented arrow symbol pointing in one direction or another) to direct consciously controlled strategic orienting [13]. In a single trial a cue will direct the participant's attention to one location. On valid trials, targets appear in the location previously cued. On invalid trials, targets appear in the opposite location resulting in a response time (RT) advantage (i.e., faster RT's) for validly cued versus invalidly cued targets. This RT advantage is typically represented as a cue-effect size (CES) by subtracting the RTs to validly cued targets from the RTs to invalidly cued targets (positive CESs are indicative of RT advantages for valid targets).

Posner and colleagues [14] found that for both reflexive and voluntary COVATs, left and right parietal patients displayed abnormally long RTs on invalidly cued trials when the cue directed attention towards the ipsilesional field but the target appeared contralesionally. Interestingly, both groups of patients demonstrated equally fast RTs for ipsilesional and contralesional validly cued targets, indicating that parietal patients were impaired at 'disengaging' attention from an ipsilesional cue in order to reorient towards contralesional space. The magnitude of this "disengage deficit" was later found to be correlated with the severity of neglect [15, for a review see 16]. In addition, subsequent studies demonstrated that patients with neglect were more impaired at reflexive orienting [16,17] which has led some researchers to suggest that neglect is primarily a deficit involving reflexive attention [12].

We recently demonstrated that PA influenced performance in both reflexive and voluntary COVATs (described above) in healthy individuals [18]. Most relevant to the current study was the observation that for reflexive covert attention leftward PA enabled participants to

reorient attention more quickly from a right visual field cue towards a left visual field target (and vice versa for rightward PA). For both left and right PA, the effects were limited to the shortest (50ms) stimulus onset asynchrony (SOA) and to participants with a large CES prior to PA. These effects were similar (with opposite effects on RT) to the effects observed by Posner and colleagues [14] in patients with parietal lesions. We suggested that PA influences visual attention and furthermore, this may represent one of the mechanisms by which PA exerts its beneficial effects in neglect patients.

The purpose of the current investigation was to examine whether rightward PA would reduce the disengage deficit in patients with right brain damage (RBD). To do this we tested four patients with RBD (2 with neglect) on a reflexive COVAT paradigm before and after PA and compared their performance to a group of healthy controls (N=26) who performed the same task before and after sham (i.e., no shift) PA. Our hypothesis was that prior to PA, RBD patients would demonstrate a much larger cost for reorienting attention leftwards compared to rightwards (i.e., a disengage deficit). Following rightward PA we predicted that the disengage deficit would be reduced and fall closer to the mean and standard deviation of controls.

Methods

Participants

The 26 controls (8 male, 4 left handed, mean age= 20.07 yrs, SD=2.37) were undergraduate students from the University of Waterloo who participated in the study for payment. Clinical information for the 4 patients, including lesion location information, is presented in Table 1. Anatomical scans of each patient's lesion are presented in Figure 1. Patients were tested for clinical signs of neglect using a combination of line bisection, star

cancellation, and figure copying (Table 1). A significant rightward bias in line bisection was defined as >5% of the total line length. To do this, the patient's bisection mark was measured in terms of the deviation away from true centre (measured in millimeters; leftward deviations scored as negative, rightward deviations scored as positive). This deviation score was then converted to a percentage of the total line length. Impaired cancellation performance was defined as >10% omissions of left targets. Figure copying was scored based on a visual inspection of the patient's performance. Patients were considered to have neglect if they were impaired in at least two of the three tests. Two RBD patients did not present with neglect on any of the tests used. All participants provided informed consent prior to participating in the experiment and the experimental protocol was approved by the Office of Research at the University of Waterloo and the Tri-Hospital Research Ethics Board of Kitchener-Waterloo in accordance with the Helsinki declaration.

-- insert Table 1 about here --

-- insert Figure 1 about here --

Apparatus and Procedure

For the COVAT we used non-informative (i.e., 50% valid) abrupt onset peripheral cues. Target locations were indicated by green circles subtending 2° and presented 12° to the left and right of fixation. A cue consisted of the brightening of one target location. Targets consisted of filled red circles presented within the location marker. Reaction times were measured by an external button press attached to an IBM compatible Pentium IV computer with a 19 inch CRT monitor.

Participants maintained fixation throughout the COVAT. Patient fixation was observed directly by the experimenter. Each trial began with a fixation cross, after a variable time period

one of the target locations was brightened. This cue remained present until the participant responded. After an SOA of either 50 or 150ms the target appeared at either the cued (valid) or uncued (invalid) location and remained on the screen until the participant responded or 3000ms elapsed. Non-cued trials where targets appeared without a preceding cue were used to measure simple RTs in the absence of cues.

The PA procedure used was adapted from Rossetti and colleagues [3]. Prior to PA, participants sat with their head in a chin rest and made five pointing movements to a subjective position straight-ahead of their body's midline with their eyes closed. The experimenter recorded the endpoints of these movements which were used to calculate each participant's pre-PA notion of straight-ahead. Patients then wore wedge base prismatic lenses (Optique Peter, France) which shifted visual perception 10° to the right. Controls wore sham prisms which induced no visual shift in order to take into account practice effects when comparing the performance of the controls to the patients. All participants used their right hand to point during PA. While wearing prisms they were asked to point to targets to the left and right of an objective straight-ahead position once every 2-3 seconds for a period of 5 minutes. Immediately following PA participants closed their eyes and pointed five more times to 'straight-ahead'. The endpoints of these pointing movements were recorded by the experimenter in order to determine the degree of adaptation to the prisms.

Data Analysis

Mean RTs were calculated for each trial type for each participant. For controls RTs were discarded if they were $<150\text{ms}$ or more than 2 standard deviations above the participant's overall mean. For patients, RTs were discarded if they were $<150\text{ms}$ or $>1000\text{ms}$. In order to examine the effects of rightward PA on the disengage deficit we calculated CESs for leftward and

rightward shifts of attention for each participant at each SOA. For leftward shifts we subtracted RTs to validly cued right targets from the RTs to invalidly cued left targets. For rightward shifts we subtracted the RTs to validly cued left targets from the RTs to invalidly cued right targets (Figure 3). For both left and right shift CES calculations the initial component of each trial is identical – a shift of attention to a cue in the left or right visual field. The only difference is the cost to reorient attention in the opposite direction to detect invalidly cued targets [18]. Prism adaptation pointing data were analyzed by calculating the mean deviation from centre for each participant prior to and immediately following PA. These data were converted into degrees of visual angle with leftward errors coded as negative and rightward errors coded as positive.

Results

Pointing data pre- and post-PA for the controls and patients is presented in Figure 2. Cue-effect size data pre- and post-PA for the patients are plotted in relation to the mean and standard deviation of controls in Figure 3.

--- insert Figure 2 about here ---

Controls

Controls showed no shift in pointing data between the pre (.08) and post (-.68) pointing sessions ($t(24)=1.2$, $p=.23$) as was expected given that the controls underwent sham PA. CES data for controls was analyzed using a 3-way ANOVA with prisms (pre vs. post), attention shift (left vs. right), and SOA (50ms, 150ms) as within subject factors. There was a main effect of prisms indicating that overall CES in the controls was slightly reduced (24ms pre vs. 17ms post) following sham PA ($F(1,25)=4.5$, $p=.04$). There was also a main effect of SOA with CES at the

50ms SOA (29ms) being larger than CES at the 150ms SOA (12ms; $F(1,25)=35.6$, $p<.0001$). No other main effects or interactions were significant.

Patients

We examined each patient's pointing data separately using t-tests with a Bonferonni correction ($p=.012$). In all but one patient (RBD1) there was a significant leftward after-effect in straight-ahead pointing post-PA (Figure 2).

Prior to PA all patients demonstrated a disengage deficit such that leftward shifts of attention at both SOAs were outside the range of controls (Figure 3). Based on previous findings [18] our specific hypothesis was that the disengage deficit (i.e., leftward shifts) in patients with RBD would be significantly reduced following rightward PA. To test this we compared CESs pre- and post-PA for leftward shifts of attention at the 50 and 150ms SOAs and found that the disengage deficit was significantly reduced at the 50ms SOA post-PA (93ms pre vs. 39ms post; $t(3)=2.88$, $p=.032$). There was also a trend towards a reduced disengage deficit at the 150ms SOA (89ms pre vs. 55ms post; $t(3)=1.29$, $p=.14$). In addition, analysis of the non-cued trials indicated that prior to PA, patients were slower for left non-cued targets compared to right non-cued targets (502ms left vs. 431ms right; $t(3)=2.33$, $p=.05$), a difference that was reduced following PA (450ms left vs. 434ms right; $t(3)=.964$, $p=.41$).

--- insert Figure 3 about here ---

Discussion

Previous research demonstrated that rightward PA is an effective way of alleviating some symptoms of neglect [3,4]. Although PA alters the performance of neglect patients, the mechanisms underlying these effects are unknown. Results from the current study clearly indicate that rightward PA substantially reduced the disengage deficit in all four patients with

RBD. Indeed, post-PA at the 50ms SOA the disengage deficit for 3 out of 4 patients was reduced to be within the range of controls. Thus rightward PA influences leftward attentional reorienting in RBD patients by enabling faster reorienting away from right field cues to detect left (contralesional) field targets. These results are similar to results in healthy individuals in that PA facilitated reorienting at the earliest (50ms) SOA [18]. It should also be noted that prior to PA, left non-cued targets were detected more slowly than right non-cued targets by all patients, a difference that was also reduced post-PA.

Although the current results are clear cut, there are still some outstanding questions to be addressed. One patient (RBD1) demonstrated a significantly reduced disengage deficit following PA, however he failed to demonstrate a significant leftward after-effect in pointing. Although seemingly contradictory, Pisella and colleagues [19] reported one patient who showed almost no pointing after-effect but demonstrated large and long-lasting changes in line bisection. Both their patient and RBD1 in our study have restricted subcortical lesions involving the caudate. This suggests the caudate may not be important for modulating orienting behaviour post-PA, but may be vital in generating the pointing after-effect, possibly through connections with the cerebellum [20].

Another discrepancy in the current study is that one patient's (RBD2) disengage deficit did not decrease as substantially as it did for the other patients. There are several potential reasons for this. First, the patient's disengage deficit was by far the largest of the four patients pre-PA. This means that the patient would have had to improve much more than the others in order to fall within the range of controls post-PA¹. Another explanation is related to the fact that the patient's lesion extended into occipital cortex. A recent study by Serino and colleagues [21]

¹ Note that the patient's disengage deficit was still reduced by 32ms post PA

found that lesions extending into occipital cortex were related to poor recovery from neglect post-PA.

Finally, it is interesting to note that PA reduced the disengage deficit in all four RBD patients regardless of whether or not they demonstrated signs of neglect. This adds to the mounting body of evidence which suggests that the neglect syndrome is much more than simply a disorder of spatial attention. That is, recent research has demonstrated that patients with neglect are also impaired in tasks which require sustained (non spatial) attention, spatial working memory, and temporal perception [1,22,23]. It is the confluence of these disorders – of which biases in spatial attention represent an important component – that leads to the presence of neglect. The suggestion here is that without the other impairments – sustained attention, spatial working memory, temporal perception deficits – a bias in spatial attention alone will not be enough to produce the full neglect syndrome. In addition, just as a disorder of spatial attention alone is not sufficient to cause neglect, reducing deficits in spatial attention may not be sufficient to completely rehabilitate neglect [24]. Another important insight from this result is the suggestion that PA may also be useful in rehabilitating attentional and spatial processing abnormalities present in patients with RBD without clinical signs of neglect.

Conclusion

The present findings show that PA reduces the rightward attentional bias and speeds attentional reorienting away from ipsilesional stimuli in patients with RBD with or without neglect. These findings add to the accumulating body of evidence suggesting that PA influences attentional processes perhaps via activation of regions in the right cerebellum and left temporal-parietal cortex which have been shown to correlate with the recovery from neglect post-PA [25].

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Table 1. Clinical details of patients.

patient	age	sex	time post stroke (months)	lesion	cancellation		figure copy	LB
					L	R		
NP1	55	M	22	F,T,P	66.67	33.33	+	9.11
NP2	55	M	11	F*,T,P	14.8	7.4	+	5.9
RBD1	71	M	12	Cau**	0	0	-	1.13
RBD2	80	M	8	M,P,O	0	0	-	-1.6

P=parietal; T=Temporal; F=frontal; O=occipital; Cau=Caudate nucleus; M=motor cortex.

*indicates that NP2 had a frontal craniotomy; **indicates that RBD1 also had a small lesion in the left thalamus; LB=line bisection task. Performance on line bisection was calculated as the amount of deviation from true centre and then converted to a percentage of the total line length (rightward deviations are scored as positive). Cancellation performance represents the percentage of omitted left (L) and right (R) sided targets. Neglect on a figure copying task is indicated by the + symbol (absence of neglect on figure copying is indicated by -).

Figure Captions**Figure 1.**

Figure 1 depicts the CT anatomical scans for each of the right brain damaged patients. The scans are presented in radiological convention with left and right reversed. Only the slices relevant to identifying each patient's lesion are presented.

Figure 2.

Figure 1 depicts the mean (\pm SE) pointing data (in degrees of visual angle) for the controls and each patient prior to (open bars), and following (grey bars) prism adaptation.

Figure 3.

Figure 3 depicts the mean cue-effect size (CES) data in milliseconds for the four patients and the mean and standard deviation from the 26 controls as a function of attentional shift (left, right) and stimulus onset asynchrony (SOA). The top panel depicts the pre prism adaptation data, and the middle panel depicts the post prism adaptation data. The bottom panel represents a schematic of how the CESs for left and right attentional shifts were calculated (see Methods). The solid lines represent where the person is fixating, the dotted lines represent the cued location where covert attention is allocated.

Figure 1

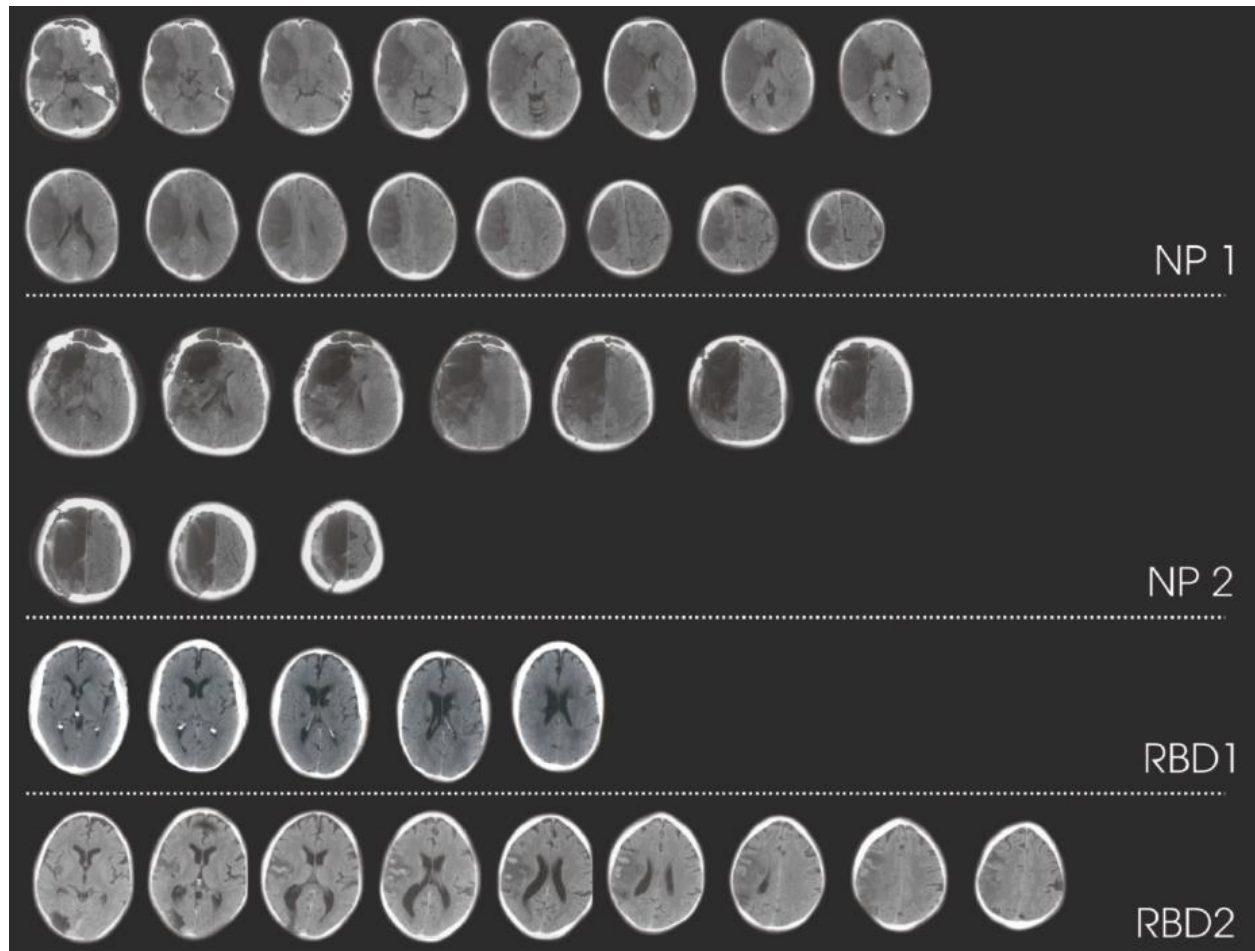


Figure 2.

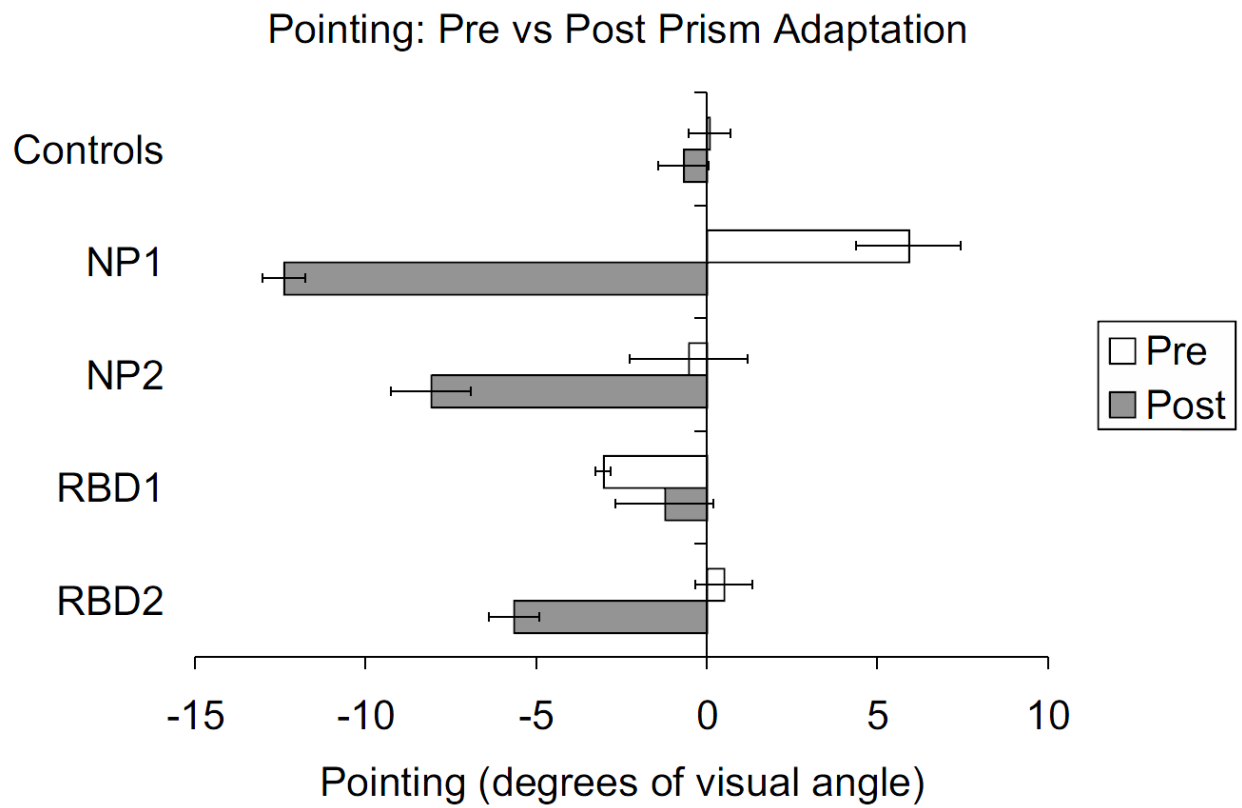


Figure 3.

