Contralesional directional hypermetria associated with line bisection-specific ipsilesional neglect

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Abstract

Patients with contralesional neglect from right hemisphere injuries often fail to be aware of or respond to visual stimuli in the left hemispace. In contrast, other patients with right hemisphere damage rarely demonstrate behavior consistent with task-specific ipsilesional neglect (IN). We performed a series of experiments in a patient with IN on a line bisection task after a right frontal infarct. When asked to perform horizontal limb movements without visual feedback, the patient showed a leftward directional hypermetria. Similar performance was also observed during a representational production of a given distance without sensory input. These results suggest that IN is induced by a directional hypermetria resulting from disruption of the motor-intentional system. © 2001 Elsevier Science Ltd. All rights reserved.

Keywords: Neglect; Ipsilesional; Directional hypermetria

1. Introduction

Unilateral spatial neglect refers to a patient’s failure to be aware of or respond to stimuli presented in a portion of space [18]. Right hemisphere injury often causes contralesional neglect (CN) or left hemispatial neglect in which patients misbisection lines to the right of the true center, fail to cross out targets from the left side of a stimulus array, and draw or copy only right-sided features of a figure. In contrast to CN, recent studies also describe patients with task performance consistent with ipsilesional neglect (IN).

Various manifestations of IN have been described. Kwon and Heilman reported a patient who misbisectioned lines to the left of the true midpoint shortly after a right frontal stroke [22]. Since the patient showed erroneous saccades toward the contralesional visual stimuli on an antisaccade task, a phenomenon termed ‘visual grasp’ [5], the authors speculated that IN resulted from release of ‘approach’ behavior toward stimuli in contralesional space. Several recent studies replicated and extended these previous findings. Kim et al. [21] found that IN most frequently occurred after damage to the frontal or subcortical brain regions and was detectable primarily with line bisection tasks. Halligan and Marshall attributed IN observed on line bisection to “reciprocal inhibition and excitation between anterior and posterior regions within the right hemisphere” [14]. Another investigation reported that the direction of bisection error varied as a function of line length [1]. Their patient bisected typically sized lines (14–20 cm) to the left of the midpoint, consistent with IN, but misbisectioned extra long lines (23 and 30 cm) near the center or to the right, suggesting that leftward bisection error may result from the phenomenon known as the ‘cross-over’ effect [6]. Robertson et al. observed 17 patients with IN showing a tendency to make more omissions on the ipsilesional side on one or more tests in the course of recovery [29]. The authors speculated that learned compensations toward the left, coupled with reduction in global attentional capacity, resulted in rightward inattention.
In distinction to IN defined strictly in terms of spatial bias reversed along the horizontal axis, other investigators have described patients with signs of severe CN in combination with subtle evidence of inattention for the ipsilateral stimuli [2,11,30]. Heilman and Van Den Abell demonstrated that whereas the left hemisphere deploys attention primarily to stimuli in right space, the right hemisphere deploys attention to stimuli from both sides of space [17]. Weintraub and Mesulam interpreted their findings of mild ipsilateral inattention in the presence of severe contralateral inattention in terms of right hemisphere dominance for directed attention [30]. Other authors who obtained similar findings attributed them to diffuse and nonspecific reduction of global attentional resources [11].

We recently encountered a patient with right frontal lobe infarction who showed IN. He misbisected lines to the left of the true midpoint on both visual and tactile line bisection tasks, consistent with IN, yet retained performance compatible with CN in other tests of neglect. He also showed visual grasp on an antisaccade task. Empirical results in our patient showed that there was a directional preponderance in horizontal limb movements in the absence of visual feedback. Therefore, we performed a series of experiments to explore the nature of this direction bias.

2. Case report

A 68 year-old right-handed man was admitted because of left hemiplegia starting the day before admission. His past medical history was remarkable for hypertension, diabetes mellitus, and right lateral medullary infarction 4 years prior to admission. On examination, he was alert and fully oriented, scoring 30/30 on the Korean version of the Mini-Mental State Examination [20]. Visual fields were intact to confrontation testing; eyes moved through a full range but he showed a rightward gaze preference. Despite left hemiplegia, he was unaware of weakness unless examiners held the plegic arm in front of him and asked him to move it. The patient reported that tactile sense on the left body was reduced by 80% compared to the right. Brain magnetic resonance (MR) imaging revealed an infarct involving the right frontal and subcortical areas as well as a small satellite lesion in the right inferior temporal lobe. The MR angiogram demonstrated occlusion of the proximal right internal carotid artery (Fig. 1).

2.1. Neglect tests

Clinical assessment of neglect was derived from performance on the following tests: ten trials of line bisection using 242 and 1.2 mm thick lines, the line cancellation task [2], the Star Cancellation task [13], copying the Two Daisy figure [24] and the Ogden picture [27], representational rendering of a clock face, and extinction to double simultaneous visual or tactile stimuli.

Table 1 provides the patient’s performance across serial examinations. Two days after injury, he performed normally on line bisection but showed left-sided extinction to simultaneous stimulation in tactile and visual modalities. Performance on Star Cancellation and copying the Two Daisy figure was also consistent with CN. On repeated examination, performance on all tests besides line bisection remained unchanged. On line bisection tasks, however, he began making mean leftward deviations greater than 10 mm (Fig. 2). The patient’s bisection error was also analyzed as a function of trial number as described by Kwon and Heilman [22]. As depicted in Fig. 3, the amplitude of leftward deviation tended to increase with each trial.

2.2. Visual grasp

To test for a visual grasp, the patient and examiner faced each other with their midsagittal plane aligned.

Fig. 1. (A, B) Fluid attenuated inversion recovery (FLAIR) MR imaging shows an infarction involving the right frontal and subcortical areas. (C) MR angiography demonstrates the occlusion of the right internal carotid artery.
Fig. 2. An illustration of neglect tests performed at 42 days after onset. The patient shows ipsilesional neglect on line bisection (A) but shows contralesional neglect on the Star Cancellation task (B) and the Two Daisy figure copy (C).

The patient maintained fixation on the examiner’s nose while the examiner held the patient’s right and left hands in each respective hemispace. The examiner’s hands were approximately 500 mm apart at a distance of approximately 450 mm from the patient’s eyes. On each trial, the examiner repetitively moved the first two digits of one hand in a ‘pinching’ motion that opposed the thumb and forefinger. The patient was instructed to shift his gaze away from the moving fingers and refixate the stationary hand as soon as he detected motion. The task consisted of two blocks of 20 individual trials in which equal numbers of right and left finger motions were presented in a random sequence. Eye movements were recorded with Ag–AgCl electrodes placed at the left and right external canthi, above and below the right

Fig. 3. Repeated line bisection up to 50 trials at 42 days after onset. Millimeter deviations from the actual midpoint; (+) and (−) mm denote rightward and leftward deviation, respectively. Note that repeated trials increased the severity of leftward deviation.
eye, and referenced to an electrode at the Cz site. To
determine stimulus onset, electrodes were also placed at
the tip of the examiner’s thumb and forefingers bilater-
ally such that opposition produced a stimulus artifact.
Potentials were amplified and recorded on a digital
polygraph (Vanguard systems, USA) with a bandwidth
of 1–9 Hz.

At four days after injury, the patient made erroneous
saccades toward the moving fingers (i.e. visual grasp)
on 60% of right-sided movements compared to 20% of
left-sided movements. On repeat testing, his perfor-
ance reversed such that, by 35 days after injury, he
committed errors on 30% of right-sided stimuli com-
pared to 60% of left-sided stimuli.

2.3. Tactile bisection

We wanted to learn if contralesional bias (IN) ob-
served on the visually guided line bisection task also
affected performance on a tactile bisection task. The
patient and six controls (four men and two women aged
62.8 ± 7.1 years) were blindfolded and seated in a chair
in front of a table. Plastic dowels of two lengths (20 and
40 cm) were placed on the table parallel to the subject’s
coronal plane. The rods’ midpoint was aligned with
three different points with respect to the subject: mid-
sagittal, or 20 cm to the left or right of the midsagittal
point. The examiner placed the subject’s right index
finger at the center of the dowel and instructed the
subject to palpate the stimulus from end to end and
then stop at the point he/she estimated to be the
midpoint. There was no limit on the duration of tactile
inspection before making a judgment. The subject com-
pleted ten trials per spatial condition (midline, left, and
right) and at each stimulus length (20 or 40 cm). The
order of the 60 unblocked trials was randomized.

Table 2 presents the results of the patient and six
controls. The patient misbisected rods to the left of the
true midpoint: the patient’s mean errors across spatial
conditions for both 20 and 40 cm rods were beyond the
95% confidence interval obtained from the six control
subjects. The bisection error varied as a function of
location. Specifically, with the 20 cm rod, the patient
was accurate in the left space but made leftward error
in the center and the right space. With the 40 cm rod,
the patient was accurate in the left and center space but
made leftward deviations only in the right space.
Hence, IN impacts perception of somatesthetic modalit-
ies and, like previous reports of CN [16], bisection
error varies according to the space of task performance.

3. Experiments: general description

A series of experiments was performed from 42 to 54
days after injury. Neurologic examination at this time
did not differ from the initial assessment except for
improvement of left limb power to grade 2 and resolu-
tion of anosognosia. All experiments were carried out
using the subject’s right hand with his eyes closed. The
same tasks were administered to six right-handed per-
sons without neurologic illness who ranged in age from
55 to 69 years (mean 60.8 years).

4. Experiment 1: directional preponderance in
horizontal limb movements

The patient demonstrated both a left visual grasp and
leftward bias (IN) on both visual and tactile bisection
tasks. The first experiment considered whether there is a
directional preponderance in horizontal limb move-
ments in the absence of visual feedback.

4.1. Methods

Subjects were blindfolded and seated in a chair in
front of a table. They were requested to reproduce
distances perceived via tactile and proprioceptive
modalities. The stimuli consisted of plastic rods of three
different lengths (10, 20, and 30 cm). Rods were located
on the table approximately 40 cm from the subjects’
midsternum and parallel to their coronal plane. The
rods’ midline was aligned with the subjects’ midsagittal

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>20 cm</th>
<th>40 cm</th>
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<tbody>
<tr>
<td></td>
<td>Left space</td>
<td>Center</td>
</tr>
<tr>
<td>Patient</td>
<td>1.5 (2.7)</td>
<td>−2.6 b (2.5)</td>
</tr>
<tr>
<td>Control (n = 6)</td>
<td>−0.29 (1.01)</td>
<td>0.03 (0.74)</td>
</tr>
</tbody>
</table>

* Values are means (S.D.).

b Mean values which are out of 95% of confidence interval.
plane. The examiner placed each subject’s index finger on the center of each rod and instructed him to make a tactile estimation of the length by palpating the rod from end to end. There was no limit on the duration of tactile inspection. Immediately after palpation, the subjects were asked to reproduce the length by extending their finger leftward or rightward along the edge of a 1 m ruler positioned in front of the dowels. The starting point for length reproduction coincided with the subject’s midsagittal plane. All subjects completed 10 trials per movement direction (rightward versus leftward) and at each stimulus length (10, 20, or 30 cm). The order of the 60 unblocked trials was randomized. Subjects were given no information regarding either the rod lengths or the accuracy of their estimations.

4.2. Results

Table 3 presents the mean distance of movement reproduction in each condition. At all stimulus lengths, controls showed no difference between leftward and rightward movements. In contrast, the patient made larger movements to the left (i.e. the directional error) compared to rightward estimates for the 20 cm ($P < 0.05$) and 30 cm ($P < 0.01$) rods. The same directional tendency was observed with the 10 cm rod but the difference between the rightward and leftward movements did not reach statistical significance.

5. Experiment 2: is leftward hypermetria directional or spatial?

In Experiment 1, the patient showed hypermetria when moving leftward from the center of the body. However, the design limited leftward movements only to the body-centered left hemispace while rightward movements occurred only in the right hemispace. As a consequence, the results of Experiment 1 do not allow precise determination of whether the leftward bias is directional (leftward movements independent of the hemispace of action) or hemispatial (movements that occur in the left body-centered hemispace independent of the direction of movement) or a combination of hemispace and direction. We tested whether hypermetria occurs when movements are made in the body-centered left hemispace (spatial hypermetria) or if hypermetria depends on the movement direction regardless of space (directional hypermetria).

Table 3
The leftward and rightward movement reproduction (cm) after tactile input of 10, 20, and 30 cm (Experiment 1) a

<table>
<thead>
<tr>
<th>Direction</th>
<th>10 cm</th>
<th>20 cm</th>
<th>30 cm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Leftward</td>
<td>Rightward</td>
<td>Leftward</td>
</tr>
<tr>
<td>Patient</td>
<td>16.3 (4.2)</td>
<td>15.0 (3.8) b</td>
<td>27.2 (5.8)</td>
</tr>
<tr>
<td>Controls (n = 6)</td>
<td>10.4 (1.1)</td>
<td>10.2 (1.3) b</td>
<td>19.1 (1.7)</td>
</tr>
</tbody>
</table>

a Values are means (S.D.).
b NS = non-significant at $P = 0.05$ (Wilcoxon signed ranks test).
c $P < 0.05$.
d $P < 0.01$.

Table 4
The distance of rightward and leftward movements (cm) in left or right space with tactile input of 10 and 20 cm (Experiment 2) a

<table>
<thead>
<tr>
<th>Direction</th>
<th>10 cm</th>
<th>20 cm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Left space b</td>
<td>Right space c</td>
</tr>
<tr>
<td>Patient</td>
<td>13.1 (2.2)</td>
<td>12.7 (2.7)</td>
</tr>
<tr>
<td>Controls (n = 6)</td>
<td>10.5 (2.6)</td>
<td>11.2 (2.6)</td>
</tr>
</tbody>
</table>

a Values are means (S.D.).
b Movements were made along the edge of a ruler either leftward or rightward, starting at 20 cm to the left of the midsagittal plane.
c Movements were made along the edge of a ruler either leftward or rightward, starting at 20 cm to the right of the midsagittal plane.
5.1. Methods

The task was similar to that of Experiment 1 with the following modifications. First, movements were reproduced starting either 20 cm to the left or right of the subject’s midsagittal plane. Subjects made estimates moving either left or right from each starting point. Second, 30 cm rods were not used because 50 cm from the subject’s body center (centripetal movement of 30 cm starting 20 cm from the midsagittal point) could be beyond the subjects’ maximal reach. Within each spatial condition (left or right hemispace), subjects performed 20 trials per rod length and ten trials in each direction of movement. The order of the 80 unblocked trials was randomized.

5.2. Results

The distance of reproduced movements in each condition is presented in Table 4. A two-way ANOVA using hemispace of action (left and right) and movement direction (leftward and rightward) as within-subjects factors was performed for each stimulus length. The results of the control subjects’ performance showed no significant main effects or interactions at either stimulus length. The patient’s performance was more complex. For 10 cm rods, the patient produced longer distances moving leftward (13.8 ± 2.7 cm) compared to moving rightward (12.0 ± 2.8 cm). The ANOVA revealed a significant main effect for movement direction ($F_{(1,36)} = 4.41, P < 0.05$) but not for hemispace. The interaction of hemispace and movement direction was not significant. For 20 cm rods, there were significant main effects for both factors (hemispace $F_{(1,36)} = 58.81, P < 0.05$; movement direction $F_{(1,36)} = 19.16, P < 0.001$). The patient moved greater distances when moving leftward (22.8 ± 3.3 cm) than rightward (18.8 ± 3.0 cm). The patient also moved longer distances in the right hemispace (22.0 ± 3.7 cm) than the left hemispace (19.6 ± 3.4 cm). The interaction of hemispace and movement direction was not significant.

5.3. Discussion

If the hypermetria we observed in Experiment 1 depends only on hemispace of action, the patient should have leftward and rightward hypermetria only in the left hemispace. The results of Experiment 2 show that leftward hypermetria occurred regardless of the hemispace in which movement transpired. However, with the 20 cm stimuli, the patient produced greater leftward movements in the right than in the left hemispace. These results might be an artifact of the experimental procedure, wherein the potential range of exploration of a subject’s right hand is greater in the right than in the left hemispace. Therefore, the patient’s leftward bias would be most likely due to a directional rather than a spatial hypermetria.

6. Experiment 3: representational production of distances

In reproducing tactually perceived distances, hypermetria could result from either augmentation of perception (afferent distortion), augmentation of motor-intentional processes (efferent error), or both. Experiment 1 found that the same tactile input resulted in hypermetria in one direction but not in the other, suggesting disordered output systems as the basis for hypermetria. To further examine this conjecture, we asked the patient and control subjects to move their finger a given number of centimeters without either tactile or visual information about stimulus size.

6.1. Methods

Subjects were instructed to produce limb displacements of a given distance (10, 20, or 30 cm) by moving their right index finger along the edge of a ruler. The ruler had no palpable marks that could be counted as a surrogate for distance estimation. The estimations were made in either left or right directions and starting from the subject’s midsagittal point. The subjects performed 10 trials in each movement direction (leftward or rightward) and at each stimulus length (10, 20, or 30 cm). The order of the 60 unblocked trials was randomized.

6.2. Results

In the control group, representational length estimates approximated the actual distance for all segment sizes. In addition, the distance of leftward movements did not differ significantly from rightward movements. In the patient, however, representational production of distance generally resulted in overestimation of length (i.e. hypermetric movement) in both directions. At all three distances, hypermetria was significantly greater for leftward movements (Table 5). Exaggeration of movement was greater than when the patient produced movements after tactile input (e.g. Experiments 1 and 2).

6.3. Discussion

The patient made larger leftward movements even without sensory information about the stimulus dimensions. This result, coupled with observations from Experiments 1 and 2, suggests that directional hypermetria originates mainly from disruption of representations or computations of motor-output processes rather than
abnormal function in the sensory-perceptual component of the tasks.

7. Experiment 4: is leftward directional hypermetria due to decreased awareness in leftward movement?

The first three experiments lead to the preliminary conclusion that leftward hypermetria results from defects within the motor-output system. However, an alternative account has not been excluded. When walking, if a normal subject becomes distracted, he/she may become unaware of the distance traversed and walk too far. To perform an accurate movement once the movement is initiated and to know when to stop, a movement may require awareness of the distance traveled and the limb’s action in space. Therefore, if our patient has decreased awareness or reduced apprehension of the limbs or the distance traveled when moving his limb leftward, then directional hypermetria might result. The next experiment considered whether the patient lacked attention to or under-perceived his own leftward movements.

7.1. Methods

The blindfolded subject sat in a chair before a table. Tactile stimuli consisted of plastic rods used in Experiment 1. Each rod was placed parallel to the subject’s coronal plane with its center aligned with the subject’s midsagittal plane. The examiner placed the subject’s right index finger on either end of the rod and instructed him/her to advance the finger to the opposite end. The examiner then asked the subject to estimate the stimulus length. If our patient was unaware of the distance traveled when moving a fixed distance, he should underestimate this distance. Subjects performed 20 trials for each rod size (10, 20, or 30 cm), with ten advancing to the left and ten to the right. The order of the 60 unblocked trials was randomized.

7.2. Results and discussion

At all stimulus lengths, the patient systematically underestimated the length of the rods by approximately 50%. However, there was no significant difference between the accuracy of leftward versus rightward estimates (Table 6). Control subjects’ performances were more accurate than the patient’s performance. Control subjects’ estimates also did not significantly differ with regard to direction. Whereas underestimation of distance by the patient suggests that he may have been less aware of distance traveled than the controls, the absence of directional asymmetries is inconsistent with the hypothesis of inattention to or hypoperception of distance as an explanation for the patient’s leftward hypermetria.

8. Experiment 5: does leftward hypermetria increase on repeated trials?

As described in Section 2, the severity of IN (leftward bias) on line bisection increased with repeated trials. The next experiment was attempted to learn if this increasing error was also related to the patient’s leftward hypermetria.

8.1. Methods

The experiment followed the same general method described for Experiment 3 but the subject was asked to produce the same distance repeatedly. Specifically, blindfolded subjects were asked to produce limb displacements of a given distance (10, 20, or 30 cm) by moving their right index finger along a ruler’s edge. The estimations were made in either left or right directions and starting from the subject’s midsagittal point. After each trial, the examiner put the subject’s index finger back at the starting point. Subjects performed 30 repeated trials in each movement direction (leftward or
rightward) and at each stimulus length (10, 20, or 30 cm).

8.2. Results

The patient tended to increase the amplitude of reproduced distance across repeated trials. The augmented output was observed for all line lengths and for both rightward and leftward movements. However, the enhancement was greater for leftward movements than for rightward movements (Fig. 4). Rightward reproduction of a 30 cm target distance started at 38 cm and reached 43 cm on the last trial. For leftward reproductions using the same target, the first trial was 36.5 cm but augmented steadily across trials, ending up with 59 cm on the final trial. For rightward reproduction of 20 cm segments, the patient started at 22 cm and reached 30 cm at the end of a block of trials, while leftward reproductions started with 27.5 cm and eventually reached 61 cm. Comparable errors were also made for the 10 cm target length.

Augmentation of hypermetria with repeated trials was quantified by fitting the distance estimates into a linear time trend using least squares regression. For leftward productions, the slope of the regression lines were 0.74 cm/trial for 10 cm targets, 0.77 cm/trial for 20 cm and 0.84 cm/trial for 30 cm. The respective slopes for rightward movements were 0.22, 0.15 and 0.27 cm/trial. The difference of slopes between leftward and rightward movement across trials was significant for all line lengths (10 cm, \( t_{(56)} = 13.68 \); 20 cm, \( t_{(56)} = 6.25 \); 30 cm, \( t_{(56)} = 6.96 \); all \( P < 0.05 \)). In contrast, control subjects showed stable distance reproductions for both leftward (mean slope: 10 cm, \(-0.008 \) cm/trial; 20 cm, \(-0.06 \) cm/trial; 30 cm, \(-0.02 \) cm/trial) or rightward movements (mean slope: 10 cm, \(-0.008 \) cm/trial; 20 cm, \(-0.10 \) cm/trial; 30 cm, \(-0.002 \) cm/trial).

9. General discussion

Our patient showed IN on line bisection tasks while simultaneously showing CN on other tasks such as target cancellation and figure copying. Several experiments were designed to help specify the features of our patient’s spatially biased behavior. When asked to reproduce the distance obtained through tactile exploration in the midline peripersonal space (Experiment 1), the patient made hypermetric leftward movements but eumetric rightward movements. Hypermetria was independent of spatial location of the exploring limb (Experiment 2). Rather, abnormal movement occurred when executed toward the left, a finding consistent with directional rather than spatial hypermetria.

Theories that account for CN following unilateral brain damage have parsed deficits into component processes. For example, CN has been explained in terms of defective motor exploration toward or in the contralateral hemispace (i.e. the motor-intentional hypothesis) [3,19]. Motor-intentional disorders caused by unilateral hemispheric injury include akinesia, hypokinesia, hypometria, and motor impersistence [15]. Directional hypokinesia refers to delayed initiation of movements toward contralesional space regardless of the hemispace where movements are performed while spatial hypokinesia refers to a delay in initiating movements limited to the contralesional hemispace. Patients with brain injury can also make smaller movement amplitudes in the contralesional hemispace (spatial hypometria) or toward the contralesional side regardless of the space where movement occurs (directional hypometria).

Unlike motor-intentional disorders associated with CN, our patient with IN demonstrated a leftward directional hypermetria. The present findings constrain the possible neuropsychological basis of this phenomenon. First, the behavior cannot be attributed to defective perceptual processes since identical input resulted in hypermetric output confined to movement in the leftward direction. Other evidence against a perceptual deficit comes from Experiment 4 wherein the patient did not show a significant difference between distance estimations obtained through leftward or rightward tactile inspection of the stimulus. Therefore, we found no indication that hypoperception or lack of awareness of his own leftward movements contributed to hypermetria. Second, a distorted mental representation is

<table>
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<th>Direction</th>
<th>10 cm</th>
<th>20 cm</th>
<th>30 cm</th>
</tr>
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<tr>
<td>Leftward</td>
<td>Rightward</td>
<td>Leftward</td>
<td>Rightward</td>
</tr>
<tr>
<td>Patient</td>
<td>5.0 (0.0)</td>
<td>5.5 (1.6) b</td>
<td>10.5 (1.6)</td>
</tr>
<tr>
<td>Controls (n = 6)</td>
<td>9.0 (1.6)</td>
<td>9.0 (1.7) b</td>
<td>18.5 (5.9)</td>
</tr>
</tbody>
</table>

* Values are means (S.D.).

b NS = non-significant at \( P = 0.05 \) (Wilcoxon signed ranks test).
Fig. 4. Repeated production of a given distance 30 times. The patient tended to increase the amplitude of reproduced distance across trials in 10 (A), 20 (B), and 30 (C) cm conditions and in both rightward (●) and leftward (■) movement conditions. However, the enhancement was greater in leftward than in rightward movements.

also not a plausible account for hypermetria. If the patient’s mental representation of the left hemispace was pathologically expanded, this might be expected to manifest itself in tasks such as figure copying, with left-sided enlargement of his drawings. In fact, the opposite behavior was observed with CN on figure reproduction. Furthermore, an expanded mental representation might be expected to produce spatial hypermetria rather than directional hypermetria, we observed. Third, IN may be related to so-called ‘cross-over effect’. Halligan and Marshall demonstrated that patients with CN on long lines might show IN on short lines [14]. Although the mechanism of this cross-over effect has not been fully elucidated, IN and the cross-over effect may be the same phenomenon. Adair et al. provided evidence to support the hypothesis that IN is related to an exaggerated cross-over point, not a reversal of spatial bias [1]. In this study we had our patient bisect rods that were 20 and 40 cm in length and found that leftward deviations were greater for the longer
rods. If IN was an exaggerated form of cross-over, however, we would have expected the opposite results. Hence, we suspect that the patient’s propensity to make longer leftward movements can be explained as dysfunction at the level of motor-intentional computations and processes. This conclusion agrees with Halligan and Marshall [14], who attribute their patient’s leftward line bisection bias to premotor deficits rather than defects of perceptual/attentional operations.

Recent studies in individuals with CN suggest that the leftward hypermetria found in our IN patient may be a general manifestation of the neglect syndrome. Bisiach et al. asked ten patients with CN to set the right and left endpoints of a virtual horizontal line when shown only its midpoint [4]. Compared to a group with right brain injury but no neglect, the patients with CN significantly over-extended the left endpoint, a ‘paradoxical’ bias for which the authors offered no convincing explanation. They interpreted the results in terms of an abnormal logarithmic spatial representation with ipsilesional compression and contralesional expansion.

According to Denny-Brown and Chambers [10], the frontal lobes mediate ‘avoidance’ behavior whereas the parietal lobes mediate ‘approach’ behavior. Their model further postulates that anterior brain regions dynamically inhibit the activity in the posterior cortex (and vice versa) to the end of selecting novel stimuli. Thus, frontal lobe damage may cause a release of parietal lobe driven approach behavior such as a grasp reflex from the contralesional hand [7,9], a visual grasp in anti-saccade tasks [12,28], and possibly magnetic apraxia or utilization behavior [8,9,23]. Denny-Brown and Chambers’ hypothesis needs to be evaluated further because very little evidence exists about whether parietal lobe injury produces avoidance behaviors. However, contralesional directional hypermetria observed in our patient might represent another form of approach behavior that follows unilateral frontal injury. This interpretation concurs with previous studies that proposed that IN results from disinhibition of approach behavior toward contralesional stimuli after unilateral frontal injury [22]. Support for this postulate also comes from the observation that our patient, like those of Butter et al. [5] and Kwon and Heilman [22], has a ‘visual grasp’. Although the underlying mechanism for the visual grasp may be multifactorial, the visual grasp, like the manual grasp, may result partly from disinhibition of complex motor routines or reflexes that subserve approach behavior.

Kwon and Heilman found that repeated trials increased the severity of both CN and IN [22]. In our patient, repeated productions also aggravated the severity of both leftward and rightward hypermetria (Experiment 5). The mechanism behind worsening hypermetria remains to be elucidated. Kwon and Heilman [22] proposed that right frontal injury impaired habituation to the left side of the line, leading to leftward bias as the intact left hemisphere habituated to the right side of the line over repeated trials. Alternatively, the mental representation for leftward movements might have expanded somehow or inattention to his own leftward movements might have worsened across multiple trials. However, since verbal reports of rod lengths via unidirectional (i.e. leftward or rightward) palpation did not differ as a function of movement direction (Experiment 4), we tentatively subscribe to the hypothesis that the directional hypermetria we observed might result from lack of habituation to contralesional stimuli.

According to previous studies, IN usually develops during the recovery stage after right hemisphere injury [22,25]. In our patient, CN on initial line bisections switched to IN within 19 days after onset and visual grasp for left-sided stimuli developed within 35 days after onset. The precise time of onset of leftward hypermetria in our patient is not known because additional experiments were conducted starting 40 days after injury. While the evolution of IN behavior will require further investigation, we speculate that frontal lobe injury may produce two opposing forces along the horizontal axis. One is attentional/intentional bias toward the right hemispace and the other is parietal lobe mediated approach behavior toward the left hemispace. In the early post-stroke period, rightward attentional/intentional bias may predominate over the leftward approach tendency, resulting in CN. As has been suggested by Butter et al. [5], the rightward attentional/intentional bias may also mask the release of visually guided saccades for left-sided stimuli, thereby accounting for our patient’s stronger visual grasp for right-sided than left-sided stimuli in the early stage. On the contrary, during the recovery period, as the attentional/intentional bias abates, the approach tendency toward the left is disinhibited from the rightward attentional/intentional bias, resulting in stronger visual grasp for left-sided than right-sided stimuli, leftward hypermetria, or IN on the line bisection test (explanation for the line bisection specific nature of IN as in our patient has been given previously [26]). Admittedly, our explanation is parsimonious because it is based on the assumption that after frontal lobe injury, the parietal lobe mediated approach behavior should be weaker but more persistent than the attentional/intentional bias, which needs to be confirmed. Only a diaschisis effect on the ipsilateral parietal lobe after frontal lobe injury or early recovery of attentional bias due to widely distributed attentional system may, however, in part support our assumption.

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References