

# Cerebellar Allocentric and Action-Intentional Spatial Neglect

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**Abstract:** Contralesional hemispacial neglect most often results from lesions in the right posterior temporoparietal cortex. Less commonly, contralesional and ipsilesional neglect are caused by lesions in the frontal lobe. Although unilateral left cerebellar lesions have been reported to cause body-centered (egocentric) ipsilesional neglect, they have not been reported to cause left-side object-centered (allocentric) neglect together with a leftward action-intentional bias. We describe a patient who had these signs of neglect 7 months after a left cerebellar hemorrhage. This 61-year-old right-handed woman reported emotional lability and difficulty walking, frequently bumping into things on her left side. Neurologic examination revealed ocular dysmetria and left-side limb ataxia. Neuropsychological tests showed evidence of neglect. On a clock-drawing test, the patient accurately drew a circle but her number placement deviated to the left side. She showed the same leftward deviation when she tried to draw a circle composed of small triangles. Although her line bisection was normal, on an allocentric task of open-triangle cancellation she was most likely to neglect triangles with a left-side opening. Her performance on this task indicated left allocentric neglect. Her leftward deviation on the clock and figure drawing tasks seems to be a form of an action-intentional grasp, which may have been induced by right frontal dysfunction superimposed on a deficit of global attention.

**Key Words:** neglect, cerebellum, allocentric, intention, attention  
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Spatial neglect has been defined as the failure to report, respond to, or orient to novel or meaningful stimuli in specific portions of space when this failure is not caused by primary sensory or motor deficits (Heilman et al, 2012). Spatial neglect can affect 1 or multiple frames of

reference. If patients have egocentric (body-centered) spatial neglect, they neglect stimuli on 1 side of their midsagittal plane (Heilman and Valenstein, 1979). In contrast, if patients have allocentric (object-centered) neglect, they neglect 1 side of an object, regardless of where it is in relation to their body.

Hemispacial neglect commonly results from injury to the right cerebral hemisphere (Albert, 1973; Gainotti et al, 1972), and is usually contralesional. However, cases of ipsilesional hemispacial neglect have been reported in patients with injury to either the right (Kim et al, 1999; Kwon and Heilman, 1991; Kwon et al, 2012; Robertson et al, 1994) or the left (Cocchini et al, 2006) cerebral hemisphere. While contralesional neglect most often results from lesions of the right posterior temporoparietal lobes (Maguire and Ogden, 2002; Vallar and Perani, 1986), ipsilesional neglect is most often associated with the frontal lobes (Kim et al, 1999).

In many patients, the signs and symptoms of neglect are perceptual-attentional (affecting how the patient perceives and attends to the environment), but the features can also be action-intentional (affecting how the patient moves in the environment) (Watson et al, 1978). One important action-intentional deficit is akinesia, the loss or impairment of the ability to implement an action fully. Akinesia may be related either to a body part, as with limb akinesia, or to a portion of space, as with hemispacial or directional akinesia (Heilman et al, 2012).

Contralesional directional akinesia is either the inability to move fully in the direction away from the injured hemisphere or a motor deviation toward the ipsilesional hemispace, and the deficits can be differentiated from perceptual-attentional deficits (Coslett et al, 1990; Heilman et al, 1985; Na et al, 1998). Most patients with action-intentional neglect have contralesional akinesia with an ipsilesional action deviation, but some have ipsilesional akinesia with a contralesional action deviation (Butter et al, 1988; Choi et al, 2001; Cohen et al, 2010). Patients with action-intentional neglect tend to have frontal lesions, while those with perceptual-attentional neglect are more likely to have temporoparietal lesions (Binder et al, 1992; Bisiach et al, 1990; Coslett et al, 1990; Harvey et al, 1995; Na et al, 1998; Tegnér and Levander, 1991).

Each cerebellar hemisphere has extensive connections with the contralateral cerebral cortex, especially the prefrontal and parietal lobes (Middleton and Strick, 1994; Schmahmann and Pandya, 1997). These areas are

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not directly involved with movement, and evidence is increasing that a cerebellar insult can cause cognitive dysfunction (Bower, 1997; Bürk, 2007; Leggio et al, 2000; Molinari et al, 1997; Schmahmann, 1996; Timmann and Daum, 2007). Cases of hemispatial neglect have been associated with both left and right cerebellar injury (Aarsen et al, 2004; Hildebrandt et al, 2002; Kim et al, 2008; Silveri et al, 2001). Although left cerebellar lesions have been reported to cause egocentric ipsilesional neglect, they have not been reported to cause allocentric ipsilesional neglect and action-intentional neglect.

We describe a patient who, after suffering a left cerebellar hemorrhage, developed allocentric (object-centered) ipsilesional neglect along with a leftward ipsilesional action bias with spatial deviation.

## CASE REPORT

### History of Present Illness

A 61-year-old married right-handed woman came to our Cognitive and Memory Disorders Clinic seeking evaluation of her poor memory.

Seven months earlier, the patient had suffered a left cerebellar hemisphere intracranial hemorrhage. A computed tomographic brain scan on admission showed the large hemorrhage with mass effect (Figure 1, left panel). The lesion required surgical evacuation.

After the surgery, the patient remained hospitalized with a depressed level of consciousness for about 2 weeks. When she went home, she could not walk because of poor balance; later she became able to walk if someone helped her.

Her short-term memory was poor. It improved somewhat over the few months after discharge, but she continued to have some difficulty remembering appointments and past conversations. She was treated with memantine for her memory difficulties. It may have helped at first but did not have sustained benefit.

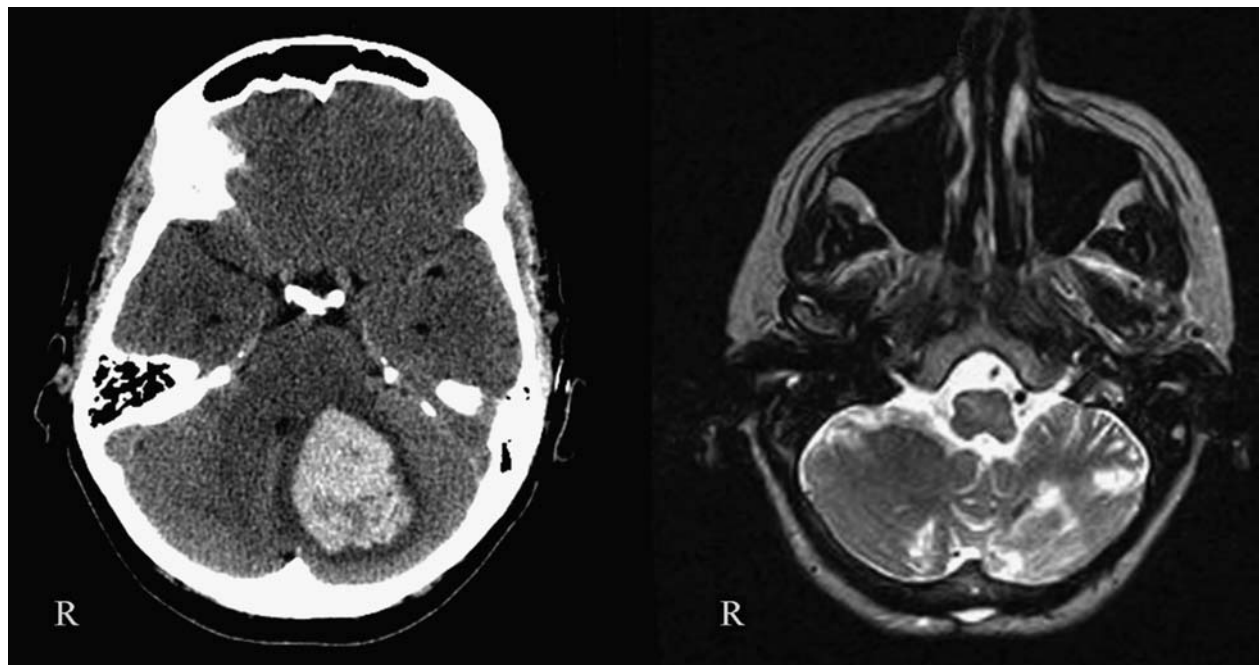
Magnetic resonance imaging 5 months after admission showed that the hemorrhage had resolved but the patient had some encephalomalacia of the left cerebellar hemisphere (Figure 1, right panel).

### Evaluation at the Clinic

#### Initial Interview

When we evaluated the patient at our clinic, she described not only problems with short-term memory since her hemorrhage, but also depressive feelings and serious emotional lability and crying spells. She became tearful several times during our interview. She also felt that she had lost her “filter” and would occasionally say inappropriate things in social settings. She reported having difficulty controlling her eyes and focusing on an object. Her husband said that when she tried to walk, not only did she have poor balance but she often bumped into things on her left side.

During college, the patient had been diagnosed with epilepsy. It began as staring spells and progressed to generalized tonic-clonic seizures. Both before and after her stroke, her antiepileptic medications were carbamazepine and lamotrigine. She had not had a seizure since her hemorrhage.



**FIGURE 1.** Left: This patient’s computed tomogram on admission shows an acute left cerebellar hemisphere hemorrhage with mass effect. Right: Five months later, T2-weighted magnetic resonance imaging shows encephalomalacia of her left cerebellar hemisphere.

She had a >10-year history of anxiety and depression, for which she took desvenlafaxine and, as needed, alprazolam. Her medical history also included restless leg syndrome, for which she took pramipexole; migraines; obstructive sleep apnea; a prosthetic mitral valve replacement; gastroesophageal reflux; cervical cancer many years earlier; and an unknown type of pituitary tumor, which had been removed 10 years earlier.

The patient had a master's level of education. She had not been able to work since her hemorrhage.

She did not smoke and rarely drank alcohol. Both of her parents had developed dementia of an unknown type in their 70s.

### Physical Examination

The patient had an unremarkable general physical examination, including ophthalmologic and cardiac examinations. Her general neurologic examination revealed significant ocular dysmetria and bilateral end-gaze nystagmus, worse when she looked to the left. She had intermittent left lower face myoclonus. Her strength was normal, but she had low tone in her left arm. Her lower extremities showed bilateral brisk deep tendon reflexes with crossed adductor responses and bilateral extensor plantar responses. She had normal coordination of her right side, but had moderate dysmetria and dysdiadokinesia of her left arm and mild dysmetria of her left leg.

### Neuropsychological Tests

During the patient's clinic evaluation, we gave her a battery of neuropsychological tests. She scored 26 of a possible 30 on the Mini-Mental State Examination (Folstein et al, 1975); she missed the day of the month, recalled only 1 of 3 objects, and could not make an accurate copy of intersecting pentagons. She scored 21 of a possible 30 on the Montreal Cognitive Assessment (Nasreddine et al, 2005); she missed the day of the month, recalled none of 5 words (although she recognized 4 of the

5), and could not copy a cube or accurately place the numbers and hands on a clock (Figure 2, left panel).

However, she performed normally on the Hopkins Verbal Learning Test–Revised (Brandt and Benedict, 2001). Her total free recall score was 21 of a possible 36: On Trial 1, she recalled 5 of the 12 words; on Trial 2, she recalled 7 of the 12; and on Trial 3, she recalled 9 of the 12. For delayed recall, she scored 8 of the 12 words. In the recognition part of the test, she recognized all 12 words in the original list, while never falsely identifying an incorrect word as being on the original list.

Her phonemic fluency was normal on the Controlled Oral Word Association Test (Benton et al, 1983). Using the letters *F-A-S*, she had a total score of 43. To evaluate her semantic fluency, we asked her to name as many animals as she could in 1 minute. She was able to name 19 animals, a normal performance (Acevedo et al, 2000). Her naming was intact, as measured by the short form of the Boston Naming Test (15/15) (Kaplan et al, 2001). She had difficulty singing “Happy Birthday” and “Silent Night,” evidence for a singing apraxia.

She performed normally on the forward and backward Digit Span Test (adapted from the Wechsler Memory Scale–Revised) (Wechsler, 1987). She had no ideomotor apraxia or deficits in emotional prosody.

Copying tasks showed that she had visuospatial dysfunction. When asked to copy the 2 intersecting pentagons for the Mini-Mental State Examination, she was able to draw 2 pentagons, but rather than intersecting they shared a single side. The intersection showed an extra pentagon shape rather than the expected diamond. For the Montreal Cognitive Assessment, she tried twice to copy the cube. The first time, she drew a square with a cross through it. The second time, she drew 2 intersecting squares, but did not connect the corners to form a cube. She did not show evidence of neglect on these copying tasks. When looking at a large triangle made of small squares, she did not show simultanagnosia.



**FIGURE 2.** After her hemorrhage, this patient's drawings show a leftward deviation. Left: Clock drawn as part of the Montreal Cognitive Assessment. Right: Her response when asked to draw a circle made up of small triangles; the dotted line denotes her approximate midsagittal plane.

We gave her several tests to assess for neglect. Neither her medical history nor our observation and examination of her indicated personal neglect. To assess for egocentric hemispatial neglect, we gave her line bisection and cancellation tasks. For the bisection task, we gave her a line 239 mm long, and she deviated 3.5 mm left of center. As her performance was within the range of normal, we did not repeat this task.

For the cancellation tasks, we first gave her a page of 55 scattered letters and asked her to cross out all the letter *As*. She successfully marked each of the 10 *As* on the page.

To assess for allocentric neglect, we gave her a page of open and closed triangles and asked her to cross out all the open triangles. The page had a total of 45 triangles: 21 on the left half of the page, 21 on the right, and 3 at the midline. Randomly scattered among the 45 triangles were 14 with openings on the right side and 14 with openings on the left side. Each opening was a break in the line on 1 side. She failed to identify 6 of the triangles with open left sides and 2 of the triangles with open right sides. Of the 6 missed left-side-open triangles, 3 were on the left half of the page and 3 were on the right half. Of the 2 missed right-side-open triangles, 1 was on the left half of the page and 1 was at the midline.

Marsh and Hillis (2008) obtained normative data for a similar open circle cancellation task from 42 hospitalized control volunteers who had no history of stroke. The mean rate of missed gaps was 0.4%, and no participant missed >1 circle with a gap (Marsh and Hillis, 2008). Our patient missed 43% of the left-side-open triangles and 14% of the right-side-open triangles. Using the Bayesian test for a deficit to compare a single case study to a normative sample, as described by Crawford and Garthwaite (2007), we found that our patient had a significant deficit based on her rates of total missed open triangles ( $P < 0.0001$ , 2-tailed), left-side-open triangles ( $P < 0.0001$ , 2-tailed), and right-side-open triangles ( $P < 0.0001$ , 2-tailed). This finding is consistent with a 2005 study in which Hillis et al defined neglect by  $\geq 10\%$  left- or right-side errors.

Marsh and Hillis (2008) further defined left- or right-side allocentric neglect as significantly more missed left- or right-side-open circles, determined statistically using  $\chi^2$  analysis ( $P < 0.05$ ). Although allocentric neglect could be determined using a single trial, the authors also allowed the data to be analyzed from 2 cancellation trials in order to identify all cases of neglect. Our patient's single open-triangle cancellation task showed a trend toward significantly more missed left- than right-side-open triangles ( $\chi_1^2 = 2.8$ ,  $P = 0.10$ ).

As part of the Montreal Cognitive Assessment, we asked her to draw a clock. She drew a circle correctly, but when she added numbers to the clock face, she put them to the left of where they should be (Figure 2, left panel). Specifically, her numbers 12, 1, 2, 3, and 4 were shifted to the left, away from the inside of the clock wall. Her number 5 was in the 6 position. Her 6, 7, 8, and 9 were shifted to the left, all partially outside the clock wall; her 8 was almost completely outside. Her 11 was close to its accurate position,

but she left little room for the 10. The point of attachment for the clock hands was supposed to be in the center of the circle, but she shifted it far to the left. We asked her to show the time as "10 past 11." She accurately drew the minute hand at the 2, but she placed the hour hand at the 9. Her placement of the hour hand would have been accurate if she had not deviated in placing the numbers.

We also asked her to draw a Navon figure (Navon, 1977). A Navon figure is a large shape made up of many copies of a small shape, eg, a big letter *H* made up of many little *As*. Navon tests are used to assess how well a person can simultaneously allocate global and focal attention. For this test, we asked the patient to draw a large circle made of small triangles. As shown in the right panel of Figure 2, she demonstrated the same pattern of leftward deviation as in her clock drawing.

Her leftward deviation on both these tasks revealed that she had an action-intentional leftward spatial bias.

## DISCUSSION

Both our patient's history of bumping into objects on her left side and our evaluation of her supported a diagnosis of spatial neglect. She had an asymmetric (left side worse than right side) allocentric hemispatial neglect on the open-triangle cancellation task and a leftward action-intentional bias on the tasks constructing the clock and Navon figure. Her ocular dysmetria and left-side ataxia were evidence of continued left cerebellar dysfunction. Given that she had no cerebral abnormalities, her neglect may be attributed to her cerebellar hemorrhage.

To our knowledge, this is the first report of allocentric neglect in a patient with a cerebellar lesion. However, cerebellar lesions have been associated with other forms of neglect.

In 2001, Silveri et al described a patient with a right cerebellar hemorrhage who had right-side ipsilesional hemispatial neglect on cancellation and copying tasks, along with right-side akinesia with a leftward action-intentional spatial bias.

Hildebrandt et al (2002) described a patient who developed neglect after suffering a ruptured basilar artery aneurysm at the exit of the right posterior inferior cerebellar artery. This rupture resulted in a lesion in the right cerebellum and brainstem. Results of a line bisection task, sentence reading, and the Attention Test Battery indicated that this patient had left-side contralesional hemispatial neglect.

Aarsen et al (2004) examined 23 children after cerebellar astrocytoma resection, and found 2 who had evidence of neglect. The first child had right upper limb neglect after resection of a tumor in the superior vermis. The second child had left upper limb neglect after resection of a tumor involving the bilateral hemispheres and midline of the cerebellum. The authors did not specify the nature of these children's neglect; it may have been personal neglect or asomatognosia.

Finally, Kim et al (2008) used line bisection, cancellation, and figure copying to assess 28 patients for neglect

after cerebellar ischemic strokes. Of the 8 patients who showed evidence of neglect, 4 had had right-side strokes and 4 had had left-side strokes. Of the 4 patients with right-side strokes, 3 had ipsilesional neglect. Of the 4 with left-side strokes, 3 had contralesional neglect. None of the patients' figure copying suggested allocentric neglect.

In 3 of these 4 reports, the authors proposed their own hypothesis for the pathophysiology of cerebellar neglect. Silveri et al (2001) thought that their patient's right-side ipsilesional neglect was a secondary defect in motor organization, related to interruption of the functional system involving the right cerebellum and contralesional frontal lobe. Hildebrandt et al (2002) suggested that their patient's left-side contralesional neglect was related to her brainstem lesion, which could cause a transient imbalance of the noradrenergic activating system and lead to hemi-inattention.

Kim et al (2008) found that in some of their patients, neglect was associated with injury to the vermis. They supposed that lesions to the vermis could lead to neglect via the vermis' connections to the superior colliculus (Niemi-Junkola and Westby, 2000), as injury to the superior colliculus has been shown to cause neglect (Dean and Redgrave, 1984). As an alternative, the authors suggested that the cerebellar strokes in their patients had led to dysfunction of the vestibular system. Because the vestibular system is part of the egocentric coordinate system essential to spatial orientation and attention, a disruption in this system could lead to a hemispacial bias.

None of these hypotheses can explain our patient's ipsilesional allocentric hemispacial neglect. Although a disruption of the vestibular egocentric coordinate system could lead to her walking deviation, such a disruption would not explain her allocentric neglect because she had neglect regardless of an object's relationship to her space. Besides, despite some initial mass effect, she did not appear to have residual injury to her brainstem or vermis, which would allow for the other proposed hypotheses of cerebellar neglect.

Similar to what Silveri et al (2001) suggested, our patient's neglect may be explained by right frontal and temporoparietal lobe dysfunction caused by the disruption of their extensive connections with the contralesional cerebellar hemisphere (Middleton and Strick, 1994; Schmahmann and Pandya, 1997). Contralesional neglect is most often associated with lesions of the right posterior temporoparietal lobes (Maguire and Ogden, 2002; Vallar and Perani, 1986).

Using functional magnetic resonance imaging, Hillis et al (2005) showed that while left egocentric neglect is most strongly associated with hypoperfusion of the right angular gyrus of the parietal lobe, left allocentric neglect is most strongly associated with hypoperfusion of the right superior temporal gyrus. Schmahmann and Pandya (1991) have shown significant connections between the cerebellum and the superior temporal polymodal cortex in the rhesus monkey. Thus, our patient's left cerebellar lesion could have caused dysfunction of her right superior temporal lobe, leading to her left-side allocentric hemispacial neglect.

Previous studies help explain why our patient showed both ipsilesional and contralesional allocentric neglect. In 1980, Heilman and Van Den Abell reported physiologic evidence in normal volunteers that the left hemisphere attends primarily to the right hemispace, but the right hemisphere attends to both the right and left hemispaces. In 1988, Weintraub and Mesulam performed a visual target cancellation task with patients who had right- or left-side hemisphere lesions and found that only the patients with right-hemisphere lesions had contralesional as well as less severe ipsilesional visual inattention. Although Weintraub and Mesulam assessed their patients only for egocentric neglect, perhaps the allocentric neglect induced by left cerebellar lesions produces a parallel asymmetric but bilateral defect of allocentric attention that has a distribution similar to Weintraub and Mesulam's egocentric neglect. Further studies are needed to test this hypothesis.

Our patient's allocentric hemispacial neglect appeared to be related to perceptual-attentional deficits, but she also had signs of an action-intentional deficit with a leftward deviation on the construction tasks. She could bisect lines accurately. She could also draw the circular outline of a clock correctly (Figure 2); but when she placed the numbers, they were deviated to the left of where they belonged, and most of the numbers on the left side extended so far to the left that they were outside the clock's circular outline. Her clock hands were also far to the left of center. Similarly, when she tried to draw a circle made of small triangles (Figure 2), she had the same leftward pattern of deviation. With her left cerebellar lesion, this leftward deviation would be considered a form of contralesional action-intentional neglect and would be ipsilesional to her presumed right cortical hemisphere dysfunction. As noted earlier, ipsilesional neglect has been reported, most often associated with frontal lesions (Kim et al, 1999).

Denny-Brown and Chambers (1958) proposed that the parietal lobes mediate approach behaviors and the frontal lobes mediate avoidance behaviors. If a frontal lobe is injured, the ipsilesional parietal lobe may become disinhibited, resulting in aberrant approach behavior. Kwon and Heilman (1991) suggested that aberrant approach behavior may lead to ipsilesional neglect caused by a contralesional perceptual-attentional or action-intentional grasp. A grasp is an ability to disengage from a stimulus. Our patient's left cerebellar lesion may have caused right frontal lobe dysfunction, and this dysfunction could have led to an action-intentional bias because of the leftward parietal-mediated approach behavior. Support for this hypothesis comes from a report by Cohen et al (2010), who described a patient with right hemisphere atrophy who had a leftward action-intentional deviation and exhibited the same clock-drawing abnormalities as our patient.

Our patient showed no left spatial bias on line bisection, but did show this bias when she placed the numbers on the clock and when she tried to draw the circle made of triangles. Our ability to reach conclusions about her line bisection is limited because we had her try

it only once. It is possible that the line bisection task, which requires mainly global attention, is much simpler than the 2 drawing tasks. Cohen et al (2010) suggested that when performing the more complex drawing tasks, a person may have to allocate both global and focal attention. For example, writing the numbers on the clock face requires focal attention at the same time that the person must allocate global attention to placing the numbers in the correct relationship to the previously drawn circle. Thus, the bias shown by both our patient and Cohen's patient may have resulted from an impairment in global attention combined with a left-side action-intentional bias.

Because the right hemisphere mediates global attention and the left hemisphere mediates focal attention (Barrett et al, 1998; Lamb et al, 1990), our patient may have had more trouble allocating global than focal attention. This may explain why she could bisect the line and draw the outer circle of the clock correctly, but when she used more focused attention to draw the numbers and hands on the clock, her right hemisphere dysfunction caused her to lose track of the global form, resulting in the left deviation. When we asked her to draw a Navon figure and she had to use her focal attention to draw the small triangles, she lost track of the global circle and again deviated to the left.

Daini et al (2008) also offer a relevant hypothesis. They had groups of patients with left or right cerebellar damage, as well as healthy controls, perform a line bisection task with and without being able to see their arm and hand. With visual feedback, all of the patients and controls had similar bisection accuracy, all 3 groups showing a slight leftward bias. Without visual feedback, the controls and the patients with right cerebellar damage showed a rightward bias, but the patients with left cerebellar damage continued to show a leftward bias. The authors concluded that the left cerebellar hemisphere may play a role in guiding movement during a bisection task in the absence of visual monitoring. When that cerebellar hemisphere is damaged, without visual feedback the patient cannot overcome the normal right hemisphere's leftward bias.

Our patient may have had inadequate visual feedback of the left hemispace because of her hemispatial neglect. Without this feedback, she may have developed the leftward bias seen in other patients with left cerebellar damage. Alternatively, if the leftward bias that Daini et al (2008) saw in their patients with left cerebellar damage was the result of right frontal lobe dysfunction leading to right parietal lobe approach behavior, this would further support our hypothesis that our patient's leftward bias was caused by an action-intentional grasp.

It is becoming increasingly clear that cerebellar dysfunction is associated with multiple forms of neglect. The signs and symptoms of cerebellar neglect may be caused by contralesional cortical dysfunction. Further research is needed to understand the disease mechanisms and to learn how these disorders can best be treated.

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