

was no detailed evaluation for coagulation function, except routine complete blood count and PT/PTT, which had normal results. Patients with sickle cell anemia are at higher risk of hemorrhagic stroke and the potential role of sickle cell disease in Case 2 cannot be evaluated in this study.

Although larger studies are needed to confirm these findings, a few comments can be offered from the findings of this series. SAH should be included into the spectrum of neurologic manifestations of severe PIH. Headache may not be severe in these patients, and occurrence of SAH in patients with PIH appears to carry relatively a benign prognosis.

Acknowledgment

The author thanks Drs. S. Chaturvedi and J. Whitty for their help.

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Ipsilateral neglect versus hemianopic compensation

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Abstract—Hemianopic patients may make line bisection errors in the direction of contralesional space, which could represent functional compensation or might be due to ipsilateral neglect. The authors report a patient with stroke with left homonymous hemianopsia and contralesional (leftward) bias on the line bisection task while upright, lying on his left side, and sitting with head bent 90 degrees leftward. Because hemianopic compensation should map retinotopically and the patient consistently erred body-leftward, the authors conclude that his contralesional errors are consistent with ipsilateral neglect.

NEUROLOGY 2003;61:120–123

Hemianopic patients may make contralesional line bisection (LB) errors.^{1,2} A compensatory contralesional bias may require awareness; active contralesional eye or head turning, or both; and preferential contralesional hemispacial attention. However, nonhemianopic patients may also err contralesionally³ (ipsilateral neglect [IpN]). Hemianopia could result from posterior cerebral artery (PCA) infarction, affecting geniculocalcarine pathways, calcarine cortex, and potentially the thalamus. Because IpN may follow thalamic infarction,⁴ contralesional LB bias after PCA stroke could represent either a compensatory bias or IpN.

We examined a 50-year-old patient with right temporal-occipital and ventrolateral thalamic infarction and right caudate hemorrhage (figure 1). He had

left-sided homonymous hemianopia; face, arm, and leg (4+/5) hemiparesis and mild incoordination; and hemihypesthesia. Echocardiogram showed a small patent foramen ovale. The patient received clopidogrel 75 mg daily.

The patient demonstrated left spatial neglect (day 3 poststroke, left-sided line cancellation omissions, left auditory extinction to bilateral stimulation, drawing copy omitted left-sided details). He erred 15 mm *leftward*, however, bisecting a 21-cm line. Whereas hemianopic visual defects map retinotopically, IpN might respect environment, head, or body-centered coordinates.⁵ By dissociating these reference frames, we wished to learn whether compensatory bias or IpN better explained the patient's errors.

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Supported by the Department of Medicine, PA State College of Medicine, the National Institute of Neurological Disorders and Stroke, National Institutes of Health (A.M.B.), the Penn State General Clinical Research Center (A.M.B.), and the Department of Veteran Affairs (K.M.H.).

Received July 24, 2002. Accepted in final form February 21, 2003.

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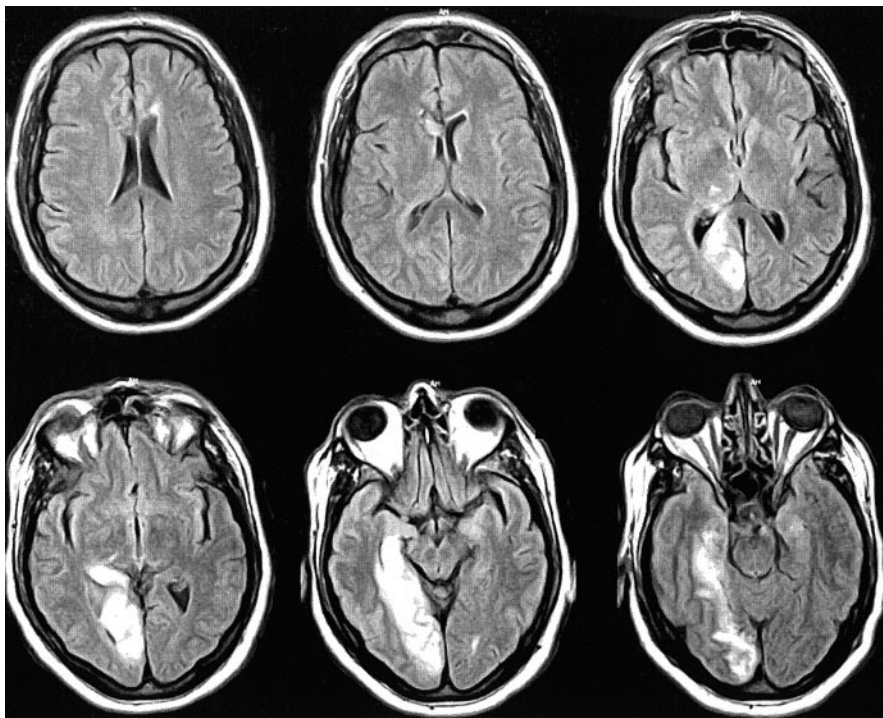


Figure 1. Fluid-attenuated inversion recovery brain MRI 1 day poststroke reveals right medial temporal-occipital cortical infarction (left side of image) as well as a small hyperdensity in the right ventral lateral thalamus (a probable lacunar stroke) and hyperdensity consistent with hemorrhage at the head of the right caudate nucleus.

Methods/procedures. Whereas compensatory bias should map retinotopically, IpN might map environmentally. Body position changes could dissociate these error types. If the patient had compensatory bias, and bisected horizontal lines presented at the intersection of his transverse and coronal planes (body-perpendicular) while lying on his left side, this position, which rotates the axis of his hemianopia, should induce leftward errors (environmentally downward, left of body midsagittal plane). Environment-centered IpN, however, should not induce systematic environment-downward (body-leftward) LB errors while left lying. Compensatory bias should not induce systematic errors while left lying and bisecting body-parallel lines, but environment-centered IpN would induce environment-leftward errors.

If IpN is body-centered, however, left lying may fail to distinguish compensatory from pathologic bias, because leftward (environmentally downward) errors may represent body-centered IpN. If the patient sat upright, head tilted 90° leftward (head-left), and bisected body-perpendicular lines, body-centered IpN should induce leftward errors. Compensatory bias, however, should change with head-left. As the axis of the hemianopia rotates 90 degrees, the axis of compensatory LB errors should change from environment-horizontal to environmentally vertical.

To test these alternative predictions, the patient bisected 80 lines in three positions over two sessions. On day 1 (26 days poststroke), he bisected 40 24-cm lines while sitting upright (10 body-parallel/environmental-vertical; 10 body-perpendicular/environmental-horizontal) and left lying (10 each body-parallel, body-perpendicular). On day 2 (27 days poststroke), the patient bisected 40 22-cm body-parallel and body-perpendicular lines with head upright (same body/head position as sitting upright) and head left (20 each, figure 2). The examiner helped the patient maintain head-left position. Error (mm) was coded on body coordinates: body-upward, -rightward, and -distal errors coded positive, body-downward, -leftward, and -proximal errors coded negative.

Analysis. We compared mean LB errors to zero for all conditions (one-sample *t*-tests). We performed four planned comparisons between mean errors (paired-sample *t*-tests): 1) body-parallel errors, sitting vs left-lying positions, 2) body-perpendicular errors, same positions, 3) body-parallel errors, head-straight vs head-left positions, 4) body-perpendicular errors, same positions.

Results. Figure 2 shows mean errors, each condition. Sitting LB error was environmentally, body-centered, and

retinotopically leftward (mean -15.4 mm, SD 13.35; $p = 0.005$) and upward (mean $+15.70$ mm, SD 8.02; $p < 0.001$).

Left lying, LB error was environmentally downward (body-centered and retinotopically leftward; mean -16.8 mm, SD 16.6; $p = 0.011$) and leftward (mean $+29.1$ mm, SD 8.12; $p < 0.001$). Mean retinotopically and body-centered leftward error magnitude was unchanged between positions ($p = 0.850$, NS), suggesting that leftward bias while sitting was body- or retinotopically centered. However, retinotopic and body-centered upward LB errors increased with left lying (mean $+29.1$ mm, SD 8.12; $p = 0.001$), suggesting a secondary environmental-left bias augmented retinotopic or body-centered bias in this position.

Day 1 testing results were consistent with either retinotopic compensatory bias or body-centered IpN. On day 2, we attempted to distinguish these possibilities by changing head position. With head upright (sitting), the patient again erred environmentally, body-centered and retinotopically leftward (mean -13.5 mm, SD 15.01; $p = 0.019$), but vertical errors did not reach significance (mean $+4.4$ mm, SD 7.52; $p = 0.097$). With head left, the patient made no consistent retinotopic-leftward (body- and environment-downward) errors (mean $+8.0$ mm, SD 19.3; $p = 0.22$). Additionally, retina-leftward errors were smaller with head left than head upright ($p = 0.028$). With head left, the patient again erred body- and environmentally leftward (mean -13.8 mm, SD 9.72; $p = 0.002$). These results are consistent with body-centered IpN.

The patient may have had secondary environment-centered leftward bias. We compared a personal (retinotopic, body-centered) up bias in the sitting and left-lying positions on day 1. Personal (retinotopic, body-centered) up bias increased while left lying compared with sitting upright ($p = 0.001$). This suggests that, while left lying, su-

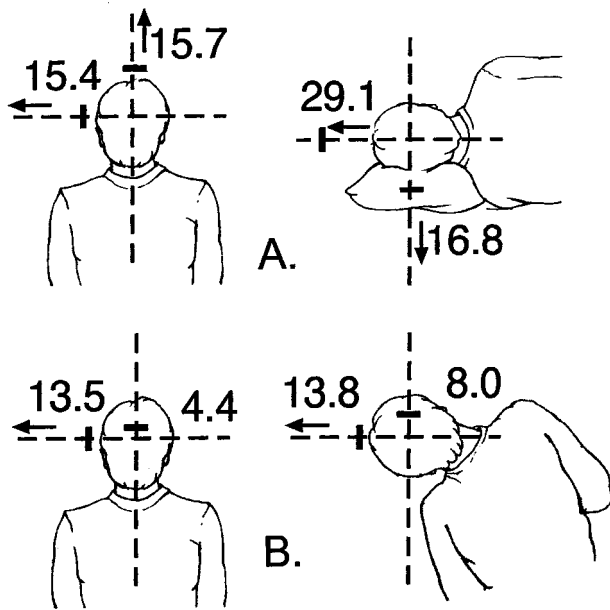


Figure 2. Artist's depiction of the current experiment and results. Axes are presented in the environmental frame; numbers indicate millimeters of error. The patient was tested in the sitting and left-lying positions (A) and in the head-straight (same as sitting) and head-left positions (B). He bisected lines either parallel or perpendicular, in the coronal plane, to his body axis in each of these positions. Mean bisection error (mm) and direction of error on environment-centered coordinates are labeled for each testing position (errors are not depicted exactly to scale). Sitting upright (sitting and head straight), he erred environment- and body-centered leftward and upward (although up bias was not significant in B). Lying on his left side, he erred environment-centered leftward and downward (A). Sitting upright with head left, he erred environment- and body-centered leftward (B). In this position, environment- and body-centered up bias did not reach significance. See text for detailed presentation of results.

perimposed environmental-left bias might increase a retinotopic or body-centered bias.

After examining these initial results, we observed that although body-leftward bias appeared stable over days 1 to 2, body-upward bias may have decreased. We performed a paired-samples *t*-test comparing environment- (retina-, body-) upward bias in sitting and head-upright (same as sitting) positions on testing day 1 vs day 2. Environment-, retina-, and body-upward bias decreased in the sitting position on day 2 vs day 1 ($p = 0.011$).

Discussion. While sitting upright, our hemianopic patient made environmental, body-centered, or retinotopic leftward LB errors. While left lying, however, he made body- and retinotopic-leftward errors—consistent with personal, not environmental, coordinates. Although retinotopic compensatory bias could produce these results, left lying also rotates the body: he could have had body-centered IpN. Comparing LB errors with head straight to those made with head/eyes tilted leftward 90 degrees (head left) revealed LB errors consistent with a body-centered,

not retinotopic, bias. Hemianopic compensation should be retinotopic, and body-centered leftward LB errors suggest IpN. Our patient, like others previously reported, demonstrated contralesional bias on LB and ipsilesional bias on other spatial tasks. This dissociation may reflect differential activation of competing visual-spatial systems⁶ or other factors.

Our patient also made retinotopic or body-centered upward errors, which increased in the left-lying position. This implies that a secondary environment-centered leftward bias may have increased a body-centered or retinotopic upward bias while left lying. Although an environment-left bias might account for part of the patient's leftward bias when sitting, it cannot account for environment-downward or body-centered leftward errors while left lying. Bisecting body-parallel lines while *right*-lying could have confirmed a secondary environmental-leftward bias. Unfortunately, this procedure was not performed.

Although our patient had comparable retinotopic, body-centered, and environmental leftward bias while sitting upright over the testing period, on day 2 he demonstrated reduced retinotopic (body-centered, environmental) upward bias while sitting upright. A reduced vertical bias may represent random variability in retinotopic, body-centered, and environmental upward bias magnitude, spontaneous recovery, or other effects, but we cannot distinguish between these possibilities. A variable retinotopic, body- and environmental-upward bias, however, cannot explain the body-centered leftward bias with head left on day 2.

Previously reported patients with IpN had predominantly frontal lesions.^{3,4} Our patient's caudate hemorrhage could cause contralesional approach behavior,⁷ but thalamic nuclei project to the frontal lobes and the basal ganglia. The patient's thalamic damage may have helped induce IpN. Other patients with PCA stroke reported to have a compensatory bias may also have thalamic injury: these patients might also have IpN. Future studies of a compensatory hemianopic bias should include careful assessment for IpN.

Spatial neglect may significantly affect functional status.⁸ Behavioral treatments and medications might improve function,⁹ but patients diagnosed with compensatory bias may not receive treatments. Clinicians should assess compensatory contralesional bias to learn if it is environment-, body-, or retina-centered. Contralesional bias in a nonretinotopic reference frame suggests neglect, and may respond to either standard or targeted¹⁰ therapeutic interventions.

Acknowledgment

The authors thank the patient for volunteering his time for this project and Kerri Hansell and Leon Dore for help summarizing and coding experimental data.

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New onset of body dysmorphic disorder following frontotemporal lesion

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Abstract—The etiology and pathophysiology of body dysmorphic disorder (BDD) have not been delineated. The authors report a 24-year-old man who developed BDD at age 21 after an inflammatory brain process. Neuroimaging studies showed new atrophy in the frontotemporal region. The authors review cases from the literature with similar clinical features and neuroimaging findings as well as discuss the possible correlation between the neuroanatomic lesion and the clinical presentation of BDD in the patient.

NEUROLOGY 2003;61:123–125

Body dysmorphic disorder (BDD) is a disabling disorder marked by preoccupation with a nonexistent or slight defect in appearance that causes clinically significant distress or impairment in functioning.¹ Facial concerns are most common, particularly concerns with the skin, hair, or nose, but any body area can be the focus of preoccupation.² Insight is often poor and approximately half of the patients are delusional.² Activities of daily living are affected, as most patients perform time-consuming associated behaviors such as mirror checking, skin picking, excessive grooming, and camouflaging.^{1,2} Owing to the similar clinical presentation between BDD and obsessive compulsive disorder (OCD), BDD is widely considered to be an OCD spectrum disorder.² To our knowledge, there have been no neuroimaging studies connecting BDD to a specific neuroanatomic lesion or region.

Case report. We evaluated a 24-year-old man with childhood onset insulin-dependent diabetes mellitus who developed BDD at age 21 after a brain injury. An inflammatory process was presumed based on blood tests, lumbar puncture, and neuroimaging studies. The patient was treated prophylactically with ceftriaxone and acyclovir owing to the possible diagnosis of an infectious process; however, the final diagnosis was never determined as all tests were negative for bacterial, fungal, and viral infection. The first head CT with contrast done in the emergency room showed no significant pathology. A brain MRI with contrast 2 days later demonstrated mild gyral

swelling with increased signal in the left sylvian gyri, suggesting an inflammatory process with mass effect on the left. In the acute recovery period, the patient had an expressive aphasia, memory deficits with recent memory more severely affected, and new onset of a focal complex seizure disorder. He improved over a period of 6 weeks and was discharged home. The patient and his mother reported that soon after returning home from the initial hospitalization, he became preoccupied with the appearance of his nose, believing it was too wide, despite the lack of an objective defect in his appearance. He had severe ideas of reference, thinking that others talked about him and avoided him because of his “disgusting appearance.” As a result, he became socially isolated and homebound. He spent hours in front of the mirror checking his nose and repetitively asked family members for reassurance about his appearance. He began saving money for cosmetic surgery and had poor insight into his condition. The patient had no therapeutic response to a trial of paroxetine at 40 mg/day for 2 months, and continued to insist on the need for cosmetic surgery.

A brain MRI 6 weeks after the initial event demonstrated mild atrophy in the left frontotemporal region compared to the right. Two years later, a brain MRI confirmed left frontotemporal region atrophy (figures 1 and 2).

Discussion. Little is known about the etiology and neurobiology of BDD. Two neuropsychological studies found that subjects with BDD had impaired verbal and nonverbal memory, suggesting frontostriatal system dysfunction.³ Two cases of BDD after medical illnesses with an inflammatory component were recently reported.⁴ No neurologic deficit was identified, and it was hypothesized that cytokines may play a role in the

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Received October 16, 2002. Accepted in final form March 17, 2003.

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Neurology 2003;61;120-123
DOI 10.1212/01.WNL.0000072331.56477.12

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