Line Bisection Error and Its Anatomic Correlate

Bernhard Baier, MD, PhD; Notger Mueller, MD; Marcel Fechir, MD; Marianne Dieterich, MD

Background and Purpose—Patients with chronic visual field defects typically show a contralateral line bisection error (LBE). However, in the acute phase of the disease, it has been suggested that the LBE points to the ipsilesional side. The aims of the current study were first, to test whether specific lesions are associated with LBE, and second, to determine whether there is a difference in the LBE between the acute and the chronic phase.

Methods—Twenty-two patients with visual field defects due to stroke without neglect were tested for line bisection and for the anatomic lesion site by voxelwise lesion-behavior mapping analysis.

Results—Patients with visual field defects in the acute and chronic phases differed in neither the direction nor the extent of their LBE. An association between lesions of the lingual gyrus, the cuneus, and the extent of contralateral LBE was found.

Conclusions—Present data support the view that a contralatal LBE is due rather to lesions of the lingual gyrus and the cuneus and not to an adaptive attentional mechanism over time. (Stroke. 2010;41:1561-1563.)

Key Words: stroke ■ line bisection error ■ visual field defect ■ lingual gyrus ■ cuneus

Traditionally, it has been assumed that line bisection errors (LBEs) directed to the ipsilesional side are associated with brain lesions leading to hemispatial neglect, whereas patients with lesions inducing visual field defects (VFDs) would tend to have LBEs that point to the contralateral side. However, a recent study found that acute hemianopic stroke patients showed ipsilesional LBEs rather than contralateral LBEs. Thus, it was suggested that the contralateral LBEs observed in chronic VFD patients reflect an attention- (and eye movement) related overcompensation of the initial deficit. Furthermore, there is some uncertainty about which brain areas are exactly associated with LBEs, with occipitotemporal extrastriate areas being the most recently postulated candidates. The aims of this study were 2-fold. First, we wanted to clarify whether the direction of the LBE indeed changes from the acute to the chronic phase of stroke resulting in VFDs. Second, by using modern voxelwise lesion-behavior mapping (VLBM) analyses, we wanted to reassess which anatomic regions induce LBEs in patients with VFDs.

Subjects and Methods

We investigated 22 stroke patients with lesions along the visual pathways, all of whom had VFDs (mean±SD age, 62±15 years). Eight patients were considered to be in the acute phase after stroke onset; that is, their testing time since the appearance of the lesion was <3 weeks (mean±SD, 0.8±0.3 week). Fourteen patients were in the chronic phase (mean±SD, 56±29.6 weeks). Thirteen patients (59%) had hemianopia and 9 patients (41%) presented with quadranopia (see the Table). There was no difference in the type of VFD (ie, hemianopia or quadranopia) between patients tested in the acute and chronic phases (chi^2 test=.806). The patients were all right-handed and had a corrected visual acuity of 0.7 or better. In addition, we tested an age-matched control group of 21 healthy individuals (mean±SD age, 58±11 years; t test P=0.349). A line bisection task with sheets of white paper that were centered directly in front of each patient was assigned. The patients were instructed to bisect six 20-cm-long horizontal black lines (2 mm thick) into 2 parts of equal length by marking the subjective midpoint of each line with a fine pencil and using their right hand. Viewing distance was ~40 cm. LBEs were measured, with approximation to the nearest millimeter, and expressed as a percentage of the total line length. VFDs were assessed with a standardized neurologic confrontation technique: The patients were asked to signal as soon as she/he perceived the examiner’s waving fingers move inward from beyond the boundaries of each visual field quadrant. In addition, the patients were requested to count stationary fingers presented sequentially in each visual field quadrant ~15° eccentric to fixation. Hemineglect was assessed as described previously with Bell’s test.

For all patients, magnetic resonance imaging (MRI) scans were performed. The T2-weighted, fluid-attenuated inversion-recovery sequence was acquired with 38 axial slices with an interislice gap of 3.3 mm. Diffusion-weighted imaging was performed with an interislice gap of 3.5 mm. The median time between lesion appearance and MRI was 6 days (range, 1 to 14 days). We used diffusion-weighted imaging within the first 48 hours after stroke and fluid-attenuated inversion-recovery sequences when imaging was conducted 48 hours or later. We combined all patient data by “flipping” the lesions of left brain–damaged subjects to the right side. With the use of MRICro software, lesions were mapped onto slices of a T1-weighted template MRI scan from the Montreal Neurological Institute.

Both the scan and lesion shape were mapped into stereotaxic space by using the normalization algorithm provided by SPM8 (available at http://www.fil.ion.ucl.ac.uk/spm/software/spm8). Montreal Neurological Institute coordinates were assigned to cortical structures by...
using a Montreal Neurological Institute space utility software tool (available at www.ihb.spb.ru/~pet_lab/MSU/MSUMain.html). The number of voxels did not differ between patients tested in the acute and chronic phase \((t\text{ test } P=0.088)\).

A statistical VLBM analysis was performed with the nonparametric Brunner-Munzel test by treating the mean LBE as the dependent, continuously measured variable.\(^8,9\) To prevent a rise in the probability of a familywise error, we computed a false discovery rate correction in both cases.

### Results

Although the affected regions were considered to be associated with hemineglect, none of the patients showed signs of neglect. The mean contralesional line bisection deviation for all 22 patients was 4.3% (SD, 2.2%). The 8 acute stroke patients showed a mean contralesional line bisection deviation of 4.4% (SD, 2.3%), whereas the 14 patients tested in the chronic phase had a mean line bisection deviation of 4.1% (SD, 2.2%). There was no difference in the LBE between patients tested in the chronic and acute phases \((t\text{ test } P=0.778; \text{ Figure 1})\). All but 1 patient with a right-sided lesion had a contralesional LBE. The 13 patients with right-sided lesions performed similarly to the 9 patients with left-sided lesions, with a contralesional LBE of 4.2% (SD, 2.0%) versus 4.4% (SD, 2.5%), respectively. In contrast, the control subjects showed only a slight mean LBE to the left of 0.1% (SD, 0.1%). Thus, there was a difference between control subjects and patients in the acute as well as the chronic phase \((1\text{-way ANOVA } P<0.01; \text{ Bonferroni corrected } P<0.01)\).

![Figure 1. Box-and-whisker plot of line bisection performance in control subjects and stroke patients (bars indicates SD).](http://stroke.ahajournals.org/)

### Table. Clinical Data for 22 Patients With Lesions Along Visual Pathways, All Showing VFDs

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Lesion Side</th>
<th>Side of VFD</th>
<th>Type of VFD</th>
<th>No. of Omissions of Bells Within the 3 Contralesional Columns Compared With the 3 on the Ipsilesional Side*</th>
<th>Lesion Size, No. of Voxels</th>
<th>Lesion Location</th>
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\(*\text{Hemispatial neglect was assessed with the Bell’s test.}^6\) This test consists of 7 columns, each containing 5 targets (bells) and 40 distractors. Three of the 7 columns (15 targets) are on the left side of the DIN A4 sheet, 1 is in the middle, and 3 are on the right. The patient is asked to cross out all of the bells; the maximum score was 30. Omission of >5 bells within the 3 contralesional columns compared with the 3 on the ipsilesional side was considered indicative of hemispatial neglect; “minus” indicates more right omissions than left; “plus” indicates more left omissions than right.
Anatomic analysis revealed that the locations of maximum overlap were the lingual gyrus and the cuneus (Figure 2A). Figure 2B shows the VLBM analysis (false discovery rate–corrected level of $P \leq 0.05$; Brunner-Munzel test), which indicates that lesions of the cuneus ($x=9$, $y=-80$, $z=10$) and lesions of the lingual gyrus ($x=7$, $y=-75$, $z=7$) were associated with larger contralesional LBEs.

**Discussion**

The present data indicate first, that patients with VFDs in the acute and chronic phases differed with regard to neither the direction nor the extent of their LBE. Second, this study shows an association between lesions of the lingual gyrus and cuneus and the extent of contralesional LBEs.

These results are interesting, because recent data have demonstrated that stroke patients with VFDs in the acute phase showed ipsilesional rather than contralesional LBEs.\(^5\) This observation has allowed some authors to speculate that the contralesional LBE in the chronic state may result from a slow, strategic, attentional adaption process.\(^5,10\) However, our data are contrary to this assumption and support the view that a contralesional LBE is an immediate emerging deficit.\(^1,4\) Therefore, a contralesional LBE is a diagnostic sign in patients with acute and chronic VFDs.

Furthermore, our data support previous reports that found an association between relative lesion frequency in extrastriate areas and LBEs.\(^4\) In the present study, lesions in these areas induced a contralesional LBE even in the acute phase after stroke. This suggests that this deficit is causally related to these areas and is neither the consequence of a later adaptive attentional mechanism to compensate for the VFD nor a direct consequence of the VFD.\(^3\) Thus, an LBE seems to present a visuospatial disorder leading to a contralesional shift of the visuospatial midline.

Despite the fact that lesion mapping plots were thoroughly examined for possible brain edema, we cannot entirely exclude a later change in lesion volume, although it has been recently shown that lesion volumes calculated from baseline images give a very good estimate of final lesion size in ischemic stroke patients.\(^11\)

**Disclosures**

None.

**References**

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