Stereoscopic Visual Impairment in Vascular Dementia

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Binocular depth perception is mediated by neural pathways that involve thalamic nuclei, the posterior parietal lobe and adjacent gyri, and white matter projections connecting these regions. Vascular dementia results from a variety of pathologic syndromes that can affect these areas, and in the current study is shown to produce associated impairment of stereoacuity or complete stereoblindness. Stereoacuity was relatively more impaired by right than left hemisphere pathology, by cortical than subcortical vascular processes, and by lesions that involved the parietal lobe. The extent of impairment was related to dementia severity as reflected by measures of intelligence, memory, and visual–spatial function. Stereoacuity was unimpaired in age-matched patients with depressive disorders. © 2000 National Academy of Neuropsychology. Published by Elsevier Science Ltd

Neural mechanisms subserving binocular depth perception (stereopsis) have evolved in many species, including the barn owl (Pettigrew & Konishi, 1976), toad (Collett, 1977), and praying mantis (Rossel, 1983). Other evolutionary strategies for making depth judgements also exist. For example, the chameleon depends primarily upon the degree of accommodation or curvature of the lens necessary to focus an image to estimate distance (Harkness, 1977). Visual cues that require only one eye can aid in making depth judgements. However, the importance of stereopsis for adaptive functioning is reflected in the view among zoologists that binocularity evolved specifically for this purpose. This hypothesis is supported by observations that one-eyed dragonfly larvae, tiger beetles, praying mantids, and water scorpions fail to capture prey (Rossel, 1983).

In humans and other mammals, axons arising from the homonymous hemiretinas of each eye synapse on neurons in the lateral geniculate nucleus of the contralateral thalamus that are sensitive to binocular disparity. These neurons have separate receptive fields for each eye, and their firing pattern depends on excitatory and inhibitory interactions produced by the horizontal disparity between the two views of the same object. Binocular cells sensitive to specific disparities in area 18 of the occipital lobes receive this thalamic input. Different cells are optimally stimulated by objects at different distances. These project to adjacent (ipsilateral) regions of the posterior parietal and infe-
ior temporal lobes, which appear to be the cortical areas responsible for the perception of depth (Julesz, 1971; Ptito & Zatorre, 1988; Ptito, et al., 1993; Ptito, Zatorre, Larson, & Tosoni, 1991b; Poggio & Poggio, 1984; Tyler, 1990). Tachistoscopic, evoked potential, and position emission tomography studies in normal individuals show that both left and right cortical regions are capable of stereopsis (Breitmeyer, Julesz, & Kropfl, 1975; Lehmann & Julesz, 1978; Richards, 1970), although there appears to be relative right hemisphere dominance for the function (Durnford & Kimura, 1971; Grabowska, 1983; Ptito et al., 1993). A second neural pathway exists for the stereoscopic processing of stimuli that fall beyond or in front of the fixation point in central vision. The resulting monocular images fall on heteronymous nasal or temporal hemiretina and therefore project to separate hemispheres. Binocular integration is made possible by neural transmission between homologous parietal–occipital regions through the corpus callosum (Blakemore, 1969, 1970; Lassonde, Sauerwein, & Lepore, 1995; Mitchell & Blakemore, 1970).

Neuropathological processes that involve structures in either functional system impair stereopsis. Degenerative changes in the posterior parietal and inferior temporal cortex caused by Alzheimer’s disease are associated with reductions in stereoacuity (Mittenberg, Malloy, Petrick, & Knee, 1994) that are related to regional decreases in parietal–occipital glucose metabolism (Kiyosawa, Bosley, & Chawluk, 1989). Mental retardation is often associated with progressive neuropathological changes similar in type and topographical distribution to those of Alzheimer’s disease (Mann, Tucker, & Yates, 1987; Wisniewski, Wisniewski, & Wen, 1985) and produces similar deficits in depth perception (Fox & Oross, 1988; Garcia, Cleland, Rago, Wayne, & Swartz, 1974; Webb, Kline, & Anderson, 1973). Head trauma causes impairment in stereoacuity that is related to focal hemorrhagic parietal lesions or diffuse axonal injury (Miller et al., 1999). In cases of diffuse axonal injury there is disproportionately severe damage to the corpus callosum (Adams, Mitchell, Graham, & Doyle, 1977; Levin, Benton, & Grossman, 1982; Strich, 1956). Impaired stereopsis has also been demonstrated in patients with callosal agenesis and following surgical transection of the corpus callosum (Jeeves, 1979, 1991; Lassonde et al., 1995; Mitchell & Blakemore, 1970). Unilateral temporal (but not frontal) lobectomies reduce stereoacuity, with relatively more pronounced effects caused by right-sided excisions (Ptito & Zatorre, 1988; Ptito et al., 1991a, 1991b). Studies of the effects of focal tumors and cerebrovascular disorders demonstrate that pathology of the posterior parietal lobe and extending into surrounding temporal and occipital gyri cause astereopsis. Right hemisphere lesions have generally been shown to cause greater impairment (Benton & Hecaen, 1970; Carmon & Bechtoldt, 1969; Danta, Hilton, & O’Boyle, 1978; Hamsher, 1978; Lehmann & Walchli, 1975; Ross, 1983).

Given evidence of impairment caused by cerebrovascular disorders and knowledge of the neural systems responsible for stereopsis, reduced stereoacuity would be expected in cases of vascular dementia due to cortical or subcortical etiologies. Relatively greater impairment would be predicted in cases with right hemisphere pathology. The current report provides evidence that supports these inferences.

**METHOD**

Patients who satisfied National Institute of Neurological Disorders and Stroke–Association Internationale pour la Recherche et l’Enseignement en Neurosciences diagnostic criteria for probable vascular dementia (Roman, Tatemichi, & Erkinjuntti, 1993) were selected for study from the populations of several rehabilitation hospitals. Medical charts were reviewed to identify patients who had been diagnosed as having cerebrovas-
cular disease by a neurologist. Computerized tomography (CT) or magnetic resonance imaging (MRI) results were examined to select patients with evidence of lateralized right hemisphere \((n = 25)\) or left hemisphere \((n = 25)\) pathology. These groups were constituted so as to be closely matched on the distribution of lesion locations. An equal number of patients in each group had cortical \((ns = 15)\) or subcortical \((ns = 10)\) pathology. Cortical lesions in each group were equally distributed in the frontal \((ns = 7)\), temporal \((ns = 5)\), and occipital lobes \((ns = 2)\). Parietal lesions were present in 11 right and 13 left hemisphere cases. Lesion location totals reflect the presence of more than one visualized abnormality in many cases, including infarcts, hemorrhages, and white matter abnormalities. MRI appears to be more sensitive than CT for defining ischemic lesions, although CT is superior for detecting hemorrhage (Cummings & Mahler, 1991). It is therefore possible that some additional abnormalities were present that may not have been detected by one of these imaging techniques.

Groups were matched for age and educational level (Table 1), and did not differ significantly in gender composition (left hemisphere, 48% male; right hemisphere, 44% male). Patients were excluded if there was a history of previous psychiatric illness or neurologic disorder other than cerebrovascular disease, or a history of ophthalmic disorder that might impair stereoacuity. Patients were also excluded if they did not demonstrate adequate visual acuity to perceive the test stimulus, or auditory comprehension adequate for completion of the test protocol. Auditory comprehension was considered sufficient if the patient performed at the recommended cutoff (13 points) or better on the Commands subtest of the Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1983). Patients completed the Vocabulary and Block Design subtests from the Wechsler Adult Intelligence Scale–Revised (Wechsler, 1981), which yielded a prorated Full Scale IQ (Sattler, 1988). Patients also completed the Logical Memory and Visual Reproduction immediate and 30-minute delayed recall subtests of the Wechsler Memory Scale–Revised, reported in age-corrected standard scores (Wechsler, 1987). Judgement of Line Orientation (Benton, Hamsher, Varney, & Spreen, 1983) was administered to 38 patients. Vascular dementia patients demonstrated significant impairment of memory and intellectual functions (Table 1), and had limitations in activities of daily living necessitating either inpatient or outpatient care.

**TABLE 1**

Demographic Characteristics and Test Scores for Vascular Dementia and Depressive Disorders Control Groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Depression ((n = 25))</th>
<th>Left Hemisphere ((n = 25))</th>
<th>Right Hemisphere ((n = 25))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>74.96 (4.61)</td>
<td>73.76 (14.57)</td>
<td>75.00 (10.58)</td>
</tr>
<tr>
<td>Education</td>
<td>12.56 (1.19)</td>
<td>12.12 (1.90)</td>
<td>12.64 (3.52)</td>
</tr>
<tr>
<td>Full Scale IQ**</td>
<td>100.80 (9.23)</td>
<td>84.04 (8.64)</td>
<td>87.44 (16.52)</td>
</tr>
<tr>
<td>Vocabulary*</td>
<td>11.44 (2.50)</td>
<td>8.76 (2.55)</td>
<td>9.72 (3.55)</td>
</tr>
<tr>
<td>Block Design**</td>
<td>8.80 (2.38)</td>
<td>5.88 (2.47)</td>
<td>6.00 (3.29)</td>
</tr>
<tr>
<td>Logical Memory 1</td>
<td>94.76 (17.83)</td>
<td>95.32 (20.00)</td>
<td>95.76 (17.99)</td>
</tr>
<tr>
<td>Logical Memory 2</td>
<td>97.72 (14.63)</td>
<td>96.84 (17.36)</td>
<td>100.24 (16.49)</td>
</tr>
<tr>
<td>Visual Reproduction 1**</td>
<td>99.20 (14.38)</td>
<td>75.88 (14.88)</td>
<td>79.44 (19.87)</td>
</tr>
<tr>
<td>Visual Reproduction 2**</td>
<td>97.80 (11.83)</td>
<td>79.96 (12.95)</td>
<td>81.44 (16.71)</td>
</tr>
<tr>
<td>Stereopsis Correct**</td>
<td>4.84 (.94)</td>
<td>2.96 (2.17)</td>
<td>1.80 (2.06)</td>
</tr>
</tbody>
</table>

*\(p < .01\). **\(p < .001\).
The control group consisted of 25 patients being treated for depressive disorders on either an inpatient or outpatient basis, who were selected to match vascular dementia groups on age and educational level (Table 1). Patients had DSM-4 (American Psychiatric Association, 1994) diagnoses of recurrent major depressive disorder (n = 8), major depressive episode (n = 6), dysthmic disorder (n = 2), bipolar 1 disorder with major depressive episode (n = 1), adjustment disorder with depressed mood (n = 7) or depressive disorder not otherwise specified (n = 1) as assessed by structured clinical interview (Spitzer, Williams, Gibbon, & First, 1990). Scores on the Beck Depression Inventory (Beck, 1987) indicated symptoms that were typically of moderate severity (M = 21.52, SD = 5.56). Patients had received medical examinations to rule out neurologic disease or systemic illnesses that would effect neurologic function, and antidepressant medication was prescribed for 9 patients at the time of study. The group completed cognitive tests identical to those administered to the vascular dementia groups.

Stereoacuity was assessed in all groups using test SO-004 (Stereo Optical Company, Inc., undated). The test consists of six items of graded difficulty, measuring sensitivity to binocular disparities of 591 to 32 seconds of arc. Each item contains a row of six consecutively numbered 0.25-inch diameter circles. One of these targets appears to stand out from the background when viewed through polarized glasses if stereopsis is adequate at the given disparity level. Total scores of zero to six correct are therefore possible. The stereotest was held approximately 14 inches in front of the subject, who was asked to identify the numbers displayed in the first row. Subjects with visual acuity or naming difficulties that interfered with performance were thereby excluded. Subjects were then asked to identify the circle in each item that appeared to be raised from the page.

RESULTS

Groups did not differ significantly in age, F(2, 72) = .11, p = .90, or years of education, F(2, 72) = .34, p = .72 (see Table 1). There was also no significant difference among distributions of premorbid occupational status, $\chi^2 (10, 75) = 10.66, p = .39$, as categorized by Wechsler (1981).

Patients with lateralized cerebrovascular disease differed from depressed controls on the measure of stereoacuity, F(2, 72) = 17.93, p < .001. Planned comparisons with Fischer’s Least Significant Difference tests (ps < .05) demonstrated that stereopsis was significantly reduced in both left and right hemisphere groups compared to controls, and was significantly more impaired by right than left hemisphere pathology. Similar comparisons showed that both cortical and subcortical lesions caused impaired stereoacuity regardless of lateralization, F(2, 72) = 19.23, p = .001. Least significant difference tests also showed that cortical lesions were associated with significantly (p < .05) more impairment.

Since more than one visualized abnormality was present in many cases, the effect of lesion localization was examined by comparing residualized mean Stereotest Scores (Table 2). This procedure amounts to an analysis of covariance in that it statistically separates the effects of vascular disease at various locations. Comparisons showed that parietal, t(47) = 3.44, p < .001, and subcortical lesions, t(43) = 3.13, p = .002, reduced stereoacuity, but no significant reductions were associated with frontal, t(37) = 1.10, p = .14, or temporal lobe pathology, t(33) = .75, p = .23. The nonsignificant effect of occipital lobe lesions, t(27) = 1.65, p = .06, is likely to be a result of the small number of subjects with disease in this area. The combined affect of multiple lesions is reflected by re-
duction in stereoaucity beyond that associated with any one localized lesion in patients with cortical vascular disease as a group (Table 2).

Relationships between stereoaucity and measures of cognitive function in the vascular dementia patients are shown in Table 3. Reduced stereoaucity predicted decline in both visually and verbally mediated measures. Analyses indicate significant associations between impairment of depth perception and severity of dementia as reflected in intelligence, memory, and visual–spatial perception.

DISCUSSION

A body of research demonstrates that binocular depth perception involves the thalamus, the posterior parietal lobe and surrounding gyri, and white matter projections connecting these regions. Vascular dementia can result from a variety of pathologic syndromes that effect these areas. The large vessel disorders are primarily cortical in their effect (Roman et al., 1993). Subcortical vascular dementias (lacunar state, Binswanger’s disease, thalamic dementia) are associated with neuropathological changes in the thalamus and hemispheric white matter (Cummings & Mahler, 1991; Stuss & Cummings, 1990). Cortical vascular dementias due to cerebrovascular disease involving the middle or posterior cerebral artery territories are associated with neuropathological changes in the thalamus, parietal, temporal, and occipital lobes (Roman et al., 1993). Current results indicate that both cortical and subcortical vascular dementia cause impairment of binocular depth perception. Cortical lesions appear to have a greater effect. Results demonstrate good agreement with previous research in a variety of other pathological groups and in normal samples that suggests relatively greater importance of the right hemisphere and of the parietal lobe in the perception of depth (Benton & Hecaen, 1970; Carmon & Bechtoldt, 1969; Danta et al., 1978; Durnford & Kimura, 1971; Grabowska, 1983; Hamsher, 1978; Lehmann & Walchli, 1975; Miller et al., 1999; Ptito et al., 1993; Ross, 1983).

Depressive symptoms frequently accompany both cortical and subcortical cerebrovascular disease (Cummings, Miller, Hill, & Neshkes, 1987; Sultzer, Levin, Mahler, High, & Cummings, 1993; Robinson, Starr, Kubos, Rao, & Price, 1983; Sarkstein, Robinson, & Price, 1987), and the presence of depression supports the diagnosis of vascular dementia (Roman et al., 1993). Primary depressive disorders are also associated with complaints of cognitive impairment (American Psychiatric Association, 1994), and depression can cause cognitive impairment in older individuals (Cassens, Wolfe, & Zola,

| TABLE 2 |
| Stereotest Scores in Patient Groups |
| (maximum = 6) |

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Mean</th>
<th>(SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortical lesions*</td>
<td>30</td>
<td>1.83</td>
<td>(2.02)</td>
</tr>
<tr>
<td>Parietal lesion*</td>
<td>24</td>
<td>3.12</td>
<td>(2.27)</td>
</tr>
<tr>
<td>Frontal lesion</td>
<td>14</td>
<td>4.12</td>
<td>(2.35)</td>
</tr>
<tr>
<td>Temporal lesion</td>
<td>10</td>
<td>4.30</td>
<td>(2.20)</td>
</tr>
<tr>
<td>Occipital lesion</td>
<td>4</td>
<td>2.29</td>
<td>(3.07)</td>
</tr>
<tr>
<td>Subcortical lesions*</td>
<td>20</td>
<td>3.20</td>
<td>(2.19)</td>
</tr>
<tr>
<td>Depressed controls</td>
<td>25</td>
<td>4.84</td>
<td>(.94)</td>
</tr>
</tbody>
</table>

*p < .01 compared to depressed controls.
Stereoaucity screening may be clinically useful in the differential diagnosis of vascular dementia and primary depressive disorders. Examination of the distribution of scores showed that 68% of patients with cerebrovascular disease demonstrated clinically significant impairment (as characterized by performances more than 2 standard deviations below the control group mean), and 32% were stereoblind. The incidence of deficient stereoaucity was dependent on the location of vascular disease (Table 4), and predicted the extent of subsequent cognitive impairment (Table 3). Bilateral infarcts and white matter abnormalities are common in patients with cerebrovascular dementia (Cummings & Mahler, 1991). Because patients with bilateral pathology were excluded from the current study, the prevalence of impaired stereoaucity in this population may be underestimated by these results.

None of the depressed patients were stereoblind, and 92% were unimpaired. The distribution of stereoscopic perception thresholds in the depressed group was essentially identical to previously observed performances in elderly patients with major depression (Mittenberg et al., 1994), younger patients with orthopedic injuries (Miller et al., 1999), an elderly normative sample (Mittenberg et al., 1994), and a younger normative sample (Coutant & Westheimer, 1993). Stereoscopic thresholds of 3 items or fewer correct (equivalent to 72 seconds of arc or greater) were identified as indicative of significant abnormality in each of these studies. The consensus of evidence suggests that depth perception is minimally effected by age and unrelated to educational level (Mittenberg et

<table>
<thead>
<tr>
<th>Variable</th>
<th>$r$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full Scale IQ</td>
<td>.37**</td>
</tr>
<tr>
<td>Vocabulary</td>
<td>.25</td>
</tr>
<tr>
<td>Block Design</td>
<td>.33**</td>
</tr>
<tr>
<td>Logical Memory 1</td>
<td>.45***</td>
</tr>
<tr>
<td>Logical Memory 2</td>
<td>.37**</td>
</tr>
<tr>
<td>Visual Reproduction 1</td>
<td>.39**</td>
</tr>
<tr>
<td>Visual Reproduction 2</td>
<td>.28</td>
</tr>
<tr>
<td>Judgement of Line Orientation</td>
<td>.64***</td>
</tr>
</tbody>
</table>

*p < .05. **p < .01. ***p < .001.

TABLE 4
Incidence of Deficient Stereoaucity in Patient Groups (%)

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Stereopsis</th>
<th>Astereopsis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortical</td>
<td>30</td>
<td>80</td>
<td>40</td>
</tr>
<tr>
<td>Subcortical</td>
<td>20</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Right hemisphere</td>
<td>25</td>
<td>76</td>
<td>44</td>
</tr>
<tr>
<td>Left hemisphere</td>
<td>25</td>
<td>60</td>
<td>20</td>
</tr>
<tr>
<td>Depressive disorders</td>
<td>25</td>
<td>8</td>
<td>0</td>
</tr>
</tbody>
</table>
al., 1994; Yekta, Pickwell, & Jenkins, 1989), and no significant relationships between these factors and stereoacuity were observed in the current sample. Decreases in stereoscopic perception thresholds have been reported to occur after 60 years of age in groups that were not screened for cognitive decline (Brown, Yap, & Fan, 1993; Greene & Maddon, 1987). Impaired depth perception may well reflect the presence of vascular or Alzheimer’s dementia in this age range. Examination of stereoacuity requires minimal patient effort and time (1–2 minutes). Impairment may effect the patient’s daily functioning, possibly by contributing to the risk of falling or being involved in a traffic accident.

REFERENCES


Stereo Optical Company, Inc. Stereo Reindeer SO-004. Chicago, IL.


