Astereopsis Caused by Traumatic Brain Injury

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Impaired depth perception (astereopsis) has been observed in a variety of cerebral pathologies affecting the posterior parietal lobe. In the current study of 93 consecutive head trauma admissions, 24% had complete astereopsis and 41% performed more than 2 SDs below the orthopedic control group mean. Degree of impairment was related to Glasgow Coma Scale score, length of posttraumatic amnesia, reduced visuospatial and memory abilities, and the presence of intracranial pathology of the parietal lobes. Impairment was also related to trauma severity in patients without any visualized intracranial pathology, presumably due to diffuse axonal shearing. Clinically meaningful impairment was observed in 25% of this group; 10% had complete astereopsis. Stereoacuity screening requires 1 to 2 minutes. Undetected astereopsis may increase risk for subsequent motor vehicle accidents or falls. © 1999 National Academy of Neuropsychology. Published by Elsevier Science Ltd.

With the invention of the stereoscope by Wheatstone (1838), binocular depth perception (stereopsis) was discovered to depend on the disparity between views that a three dimensional object affords to each eye. This binocular disparity can be perceived as a horizontal shift in the visual field of one eye compared to the other when each eye is closed in alternation. The two images are fused in the cerebral cortex and experienced as a single three dimensional representation under normal circumstances (Poggio & Poggio, 1984). Some patients with astereopsis complain of difficulty perceiving distance, perspective, depth, or thickness. Automobiles can appear to be on a collision course with each other or pedestrians, and stairs may appear to be relatively flat (Riddoch & Humphreys, 1989). However, the majority of these patients lack awareness of any impairment.
The absence of subjective symptoms may be a function of several factors including anosognosia, reliance on familiarity with the environment, dependence on monocular depth cues such as motion parallax and linear perspective, or the use of binocular convergence and accommodation. Stereopsis is more accurate than these other distance cues and does not depend on previous experience in the environment. It evolved in mammals with frontally positioned eyes at the expense of a more lateral eye position, which affords a wider visual field that includes the back of the head (Julesz, 1971; M. Ptito, Leport, & Guillemot, 1991). Stereopsis permits the recognition of objects that would otherwise be hidden by their perceptual similarity to the background. For example, predators are able to identify prey in spite of their protective camouflage by using this ability, and stereoscopic aerial photographs can be used to disembed camouflaged targets (Julesz, 1986).

Depth perception is innate (Atkinson & Braddick, 1976; Bower, 1971; Fox, Aslin, Shea, & Dumais, 1980), but development of the ability can be arrested by oculomotor disorders. Adult levels of stereoacuity are normally attained by 7 to 9 years of age (Amigo, 1973; Cooper, Feldman, & Medlin, 1979; Romano, Romano, & Puklin, 1976) and remain stable through the lifespan into the 70s (Mittenberg, Malloy, Petrick, & Knee, 1994; Yekta, Pickwell, & Jenkins, 1989). Approximately 3% of the population is stereoblind primarily as a result of strabismus, an ophthalmologic condition characterized by misalignment of the eyes (Cooper, 1979; Julesz, 1986; Richards, 1970). Impaired stereoacuity has also been observed in patients with a variety of cortical pathologies including Alzheimer’s disease (Mittenberg et al., 1994), tumors and cerebrovascular disorders (Benton & Hecaen, 1970; Danta et al., 1978; Hamsher, 1978; Lehmann & Walsh, 1975; Ross, 1983), and following surgical excisions for the treatment of intractable epilepsy (A. Ptito & Zatorre, 1988; A. Ptito, Zatorre, Larson, & Tosoni, 1991). The localization of pathological processes in these studies suggests that unilateral or bilateral cortical lesions involving the posterior parietal lobe and adjacent gyri reduce stereoacuity. The incidence of astereopsis following traumatic brain injury has not been systematically examined. Impairment may occur due to focal cortical lesions, but also as a result of diffuse axonal injury that may involve the corpus callosum, optic chiasm, or cranial nerves subserving binocular vision and fusion. The present study was designed to determine the frequency of impaired stereoacuity after head trauma and to examine the nature of the pathology associated with this impairment.

**METHOD**

The sample consisted of 93 consecutive hospital admissions for treatment of head trauma due to motor vehicle accidents (56%), falls (14%), blows with blunt objects (24%), or other mechanisms (6%). On admission to the emergency department, 79% of the injuries were characterized by a Glasgow Coma Scale (GCS) of 13 to 15, 13% by a GCS of 9 to 12, and 8% by a GCS of 8 or less. Length of posttraumatic amnesia (PTA) measured by serial daily administration of the Galveston Orientation and Amnesia Test (Levin, O’Donnell, & Grossman, 1979) averaged 3.37 days \( SD = 7.34 \), with a range of 0 to 49 days. Computerized tomography (CT) results were reviewed to determine the location of any abnormality. Evidence of intracranial abnormality was present in 44 cases, and 25 patients had sustained skull fractures. The group averaged 11.77 years of education \( SD = 2.85 \) and had a mean age of 33.05 years \( SD = 13.69 \). A subset of this group \( n = 57 \) 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), the Rey Complex Figure and the Rey Auditory Verbal Learning Test (Lezak, 1995)

A control group was constituted from 30 consecutive hospital admissions for treatment of orthopedic injuries not involving the head and characterized by normal neurological status. The group had an average age of 37.63 years ($SD = 17.56$) and a mean educational level of 11.50 years ($SD = 3.09$). Head trauma and control groups did not differ significantly in age, $t(121) = 1.48, p = .14$, or educational level, $t(121) = .45, p = .66$.

Stereoacuity was assessed using Stereo Optical Company Test 004 (Stereo Optical Company, Inc., n.d.) following resolution of any PTA. The test consists of six items of graded difficulty, measuring sensitivity to binocular disparities of 591 to 32 seconds of arc. Each item consists of a row of six sequentially numbered .25” diameter circles. One of these targets appears to be raised from the background when viewed through polarized glasses if stereopsis is adequate at the given disparity level. Total scores of 0 to 6 correct are therefore possible. The stereotest was held approximately 14” in front of the patient, who was asked to read the numbers displayed in the first row. Individuals with visual acuity difficulties that interfered with performance were thereby excluded. Patients were then asked to identify the circle in each item that appeared to be raised from the page.

**RESULTS**

Stereopsis was significantly, $t(121) = 3.54, p = .001$, impaired by traumatic brain injury ($M = 3.85, SD = 2.2$) relative to orthopedic controls ($M = 5.33, SD = 1.09$). Degree of impairment was dependent upon the severity of trauma as characterized by GCS, $r(91) = .37, p < .001$, and length of PTA, $r(91) = -.37, p < .001$, in the head-injured group. Examination of the distribution of scores showed that 24% of the sample demonstrated stereoblindness. Clinically significant impairment of stereoacuity was present in 41% of the head trauma group, as characterized by performance more than 2 $SD$s below the orthopedic control group mean. No controls had astereopsis, but two had impaired stereoacuity.

Head trauma patients with skull fractures ($M = 2.44, SD = 2.27$) showed significantly, $t(91) = 4.03, p < .001$, lower stereopsis scores than those without fractures ($M = 4.37, SD = 1.95$). Patients with intracranial pathology on CT had significantly, $t(91) = 3.65, p < .001$, reduced stereoacuity ($M = 3.02, SD = 2.30$) compared to head-injured patients with normal CTs ($M = 4.59, SD = 1.85$). There was no significant difference between those with unilateral versus bilateral lesions, $F(1, 38) = .61, p = .72$.

Because many patients presented with CT-visualized abnormalities (primarily hematoma and/or hemorrhage) at more than one location, the effect of lesion localization was analyzed by simultaneous multiple regression of all localizations on the stereopsis score. Dummy coded variables representing each lesion location (parietal, occipital, frontal, and temporal) were created by assigning each head trauma patient a value of 1 when an abnormality was present and zero when no abnormality was imaged at a given localization. The resulting four location variables were entered in a regression equation to predict stereoacuity score. The overall prediction equation was significant, $R(4, 88) = .49, p < .001$, indicating that intracranial pathology was associated with reduced perception of depth.

Residualized means (Table 1) were then generated using the regression equation to predict the stereoacuity scores of patients in each localization group after controlling for variance due to coincident abnormalities in any other location. This procedure is essen-
tially an analysis of covariance in that it statistically removes the effects of any damage in other areas from the scores of those with injuries at the site of interest. The statistical significance of reductions in stereoacuity associated with each focal lesion was evaluated by a test of the corresponding partial regression coefficient (beta weight). Patients with parietal lesions evidenced significantly, $t(88) = 2.86, p = .005$, reduced stereopsis compared to head trauma patients without intracranial abnormalities on CT. Damage in other regions was not associated with any significant reduction beyond that attributable to diffuse injury. The effect of parietal lesions remained significant after controlling for injury severity by removing variance due to GCS, $R(1,40) = .40, p < .001$, or both PTA and GCS, $R(1,89) = .38, p < .001$.

However, impaired stereoacuity was not entirely attributable to localized cortical pathology. Depth perception was also significantly reduced, $t(77) = 1.99, p = .05$, in head trauma patients without intracranial abnormalities ($M = 4.59, SD = 1.85$) compared to orthopedic controls ($M = 5.33, SD = 1.09$). Degree of impairment was related to GCS, $r(47) = .27, p = .03$, and length of PTA, $r(47) = -.34, p = .009$, in brain-injured patients with normal CTs. Astereopsis was present in 10% of this group ($n = 49$), and 25% demonstrated clinically meaningful impairment by performances more than 2 $SD$s below the orthopedic control group mean.

The relationships between stereoacuity and cognitive test performance in the head trauma group are shown in Table 2. Reductions in stereoacuity were associated with decreased visuospatial ability and reduced visual and verbal memory efficiency.

### TABLE 1
Stereotest Scores in Patient Groups
(Maximum Score = 6)

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parietal lesion</td>
<td>19</td>
<td>2.78</td>
<td>2.04</td>
</tr>
<tr>
<td>Occipital lesion</td>
<td>8</td>
<td>3.41</td>
<td>2.18</td>
</tr>
<tr>
<td>Frontal lesion</td>
<td>32</td>
<td>3.80</td>
<td>1.90</td>
</tr>
<tr>
<td>Temporal lesion</td>
<td>15</td>
<td>4.63</td>
<td>2.27</td>
</tr>
<tr>
<td>No lesion</td>
<td>49</td>
<td>4.59</td>
<td>1.85</td>
</tr>
<tr>
<td>Orthopedic control</td>
<td>30</td>
<td>5.33</td>
<td>1.09</td>
</tr>
</tbody>
</table>

$^a$ $p = .01$ compared to head trauma patients without focal lesions.
$^b$ $p = .05$ compared to orthopedic controls.

### TABLE 2
Correlations Between Stereoacuity and Cognitive Measures

<table>
<thead>
<tr>
<th>Cognitive Measure</th>
<th>$r$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full Scale IQ</td>
<td>.20</td>
</tr>
<tr>
<td>Vocabulary</td>
<td>.20</td>
</tr>
<tr>
<td>Block Design</td>
<td>.21</td>
</tr>
<tr>
<td>Rey Complex Figure Copy</td>
<td>.38**</td>
</tr>
<tr>
<td>Immediate Recall</td>
<td>.37**</td>
</tr>
<tr>
<td>Delayed Recall</td>
<td>.34**</td>
</tr>
<tr>
<td>Rey Auditory-Verbal Learning Total, Trials 1–5</td>
<td>.30*</td>
</tr>
<tr>
<td>Words Learned</td>
<td>.30*</td>
</tr>
<tr>
<td>Delayed Recall</td>
<td>.32*</td>
</tr>
</tbody>
</table>

$^a p < .05$.  
$^* p < .01$.  
$^** p < .001$.  

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DISCUSSION

Head trauma appears to produce impairment or complete loss of binocular depth perception in a clinically significant proportion of patients. The extent of subsequent impairment is associated with the severity of head trauma as reflected by admission GCS, length of PTA, reduced visuospatial and memory abilities, the presence of CT-visualized abnormality, or skull fracture. At least two distinct pathophysiological mechanisms may be responsible for this.

Binocular images of an object at the fixation point in central vision fall on the homonymous hemiretinias in each eye. Each homonymous hemiretinal pair then projects to the contralateral occipital lobe via the geniculocalcarine tract. Adjacent posterior parietal cortex appears to be responsible for the perception of depth. A second neural system exists for the stereoscopic processing of stimuli positioned beyond or in front of the fixation point. The resulting images fall on heteronymous temporal or nasal hemiretina, and therefore project monocular information to separate hemispheres. Binocular integration occurs by neural transmission across the corpus callosum from homologous parietal-occipital regions (Howard & Rogers, 1995).

Focal posttraumatic damage involving the parietal lobes is associated with astereopsis, similar to the previously demonstrated effects of a variety of other cerebral pathologies involving this region. However, reductions in stereoacuity also appear to be related to injury severity in patients without focal pathology. Diffuse axonal shearing may be responsible for impaired stereopsis in these cases. Shearing injuries frequently involve the corpus callosum (Levin et al., 1989; Richardson, Springer, Varney, Struchen, & Roberts, 1994), the optic chiasm (Crompton, 1970; Heinz, Nunery, & Grossman, 1994; Russell, 1960; Savino, Glaser, & Schatz, 1980), or the cranial nerves subserving coordinated eye movement (Keane, 1993; Leport, 1995). Both the corpus callosum and optic chiasm are necessary for the integration of binocular information, and lesions of these structures are known to produce astereopsis (Blakemore, 1970; Mitchell & Blakemore, 1970; M. Ptito et al., 1991). Shearing injuries involving the oculomotor, trochlear, or abducens nerve systems can impair stereoacuity by causing binocular misalignment (Falk & Aksionoff, 1992). Fracture of the orbit may also alter eye alignment (Russell, 1960). Peripheral and central changes in systems affecting the eye musculature may result in double vision and in turn the suppression of visual input from one eye, causing loss of stereoscopic vision. Although the incidence of these changes was not evaluated in the current study, an ophthalmologic examination may be useful in the differential diagnosis of stereoblindness in clinical practice.

The prognosis for recovery of stereopsis is not known, but impairments appear to persist in patients who sustain head injuries that are severe (Schlageter, Gray, Hall, Shaw, & Sammet, 1993). Visual perception and memory difficulties may limit the extent to which familiarity with the environment can effectively substitute for intact stereoacuity. Accommodation and convergence, binocular depth cues that could partially compensate for astereopsis, are also frequently impaired by traumatic head injury (Cohen, Grosswasser, Barchadski, & Appel, 1989; Falk & Aksionoff, 1992; Harrison, 1987; Leport, 1995; Padula, Shapiro, & Jasin, 1988). The neural substrates of convergence and accommodation are not well understood, but the midbrain, frontal, and occipital lobes are thought to be involved (Leport, 1995; Spierer, Huna, Rechtman, & Lapidot, 1995).

Stereoacuity testing is brief (1–2 minutes) and is well tolerated by inpatients because of the minimal effort required. Undetected impairment of binocular depth perception may contribute to an increased risk for subsequent traumatic brain injury due to motor vehicle accidents or falls (Humphriss, 1987; McKnight, Shinar, & Hilburn, 1991; Munck-Fairwood, 1992).
REFERENCES


