Brain damage and global stereopsis

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When a single object lies in front of or beyond the plane of fixation its retinal image lies on disparate positions in the two eyes. This 'local' retinal disparity is an excellent cue to depth, and retinal disparities of a few seconds of arc are detectable by people and monkeys. However, most visual scenes produce a complex array of contours in each eye and we can detect the disparity in the arrays despite the ambiguous nature of the disparities, i.e. each contour in one eye could be related to any of several similar contours in the other eye. This ability, known as 'global' stereopsis, may be selectively impaired following brain damage in man. Global stereopsis was measured in rhesus monkeys before and after removing a different cortical visual area in different groups of animals. Only removal of the inferotemporal cortex impaired global stereopsis. The result is related to the findings with human patients and to receptive field properties of neurons in the inferotemporal cortex of monkeys.

INTRODUCTION

When something lies further or closer than the fixation point of the two eyes, its image lies on non-corresponding or disparate regions of the retinae. We can detect disparities of as little as 2–3", arc seconds at the fovea, which corresponds to perceiving that one vertical needle is about 0.5 mm closer than another at a viewing distance of 2 m. This is known as stereoacuity, the smallest local disparity we can detect. It is not in principle difficult to understand how we do this. Numerous physiological experiments on cats during the past 10 years have revealed binocular neurons that are tuned to small local disparities produced by a simple target like a line.

However, many judgments of relative distance are more complicated. We can see at a glance that a clump of trees lies in front of the forest rather than forming the background of a glade. But it is very difficult with only one eye, so presumably retinal disparity rather than secondary and monocular depth cues are used. This creates a problem. A clump of trees and its background create innumerable retinal images of stems and leaves. How does the brain know how to match a particular image in one eye with its correct partner in the other eye? There are hundreds of images, many so identical in size and shape and orientation as to provide acceptable image matches at differing disparities and therefore to indicate a variety of confusing distances.

This problem is formalized and exemplified by the random element stereogram
(Julesz 1971) in which an array of randomly generated elements, which may be
dots, lines, letters, shapes, or combinations of them, is presented to each eye
separately, usually by projecting and viewing them through polarizing filters. As
the two arrays overlap completely in the visual field only a single array is seen.
The random elements in the surround of the display are identical and in register
for each eye and we therefore see it in one plane. However, if an entire group of
elements in the centre of one array is shifted laterally there is now horizontal
disparity between each element of the central group and its corresponding element
in the other eye. What we perceive is a central figure apparently hovering in front
of the surround or lying behind it, depending on the direction of the shift. It is
like a clump of trees at a different distance from the rest of the forest.

The random element stereogram is a powerful tool in investigating stereopsis
because no cue other than retinal disparity is present. As neither eye alone sees
any figure, disparity is being used to construct the global figure in the absence of
monocular contours and monocular pattern perception. It shows that depth and
pattern can be processed separately and suggests that one of the biological roles
of detecting a global figure from multiple disparities is to see a camouflaged object
that is invisible monocularly. But the most intriguing problem with the random
element stereogram is the fact that we do see a single figure at a particular depth
(Julesz 1971; Bishop & Henry 1971). Like the trees in the forest, each element
seen by the left eye could be matched with not one but every one of a large number
of dots in the other eye, and there are therefore many possible disparities or depth
planes in a random element stereogram. What the brain seems to do is select that
particular disparity where every image in one eye has a matching image in the other
at that disparity, and to ignore all other less common disparities at which a pro­
portion of the elements happen to match. This mechanism, which is physiologically
mysterious, although Nelson (1975) has proposed a solution based on excitation
and inhibition between columns of neurons tuned to different disparities, is called
global stereopsis by Julesz (1971).

Is the mechanism by which we achieve global stereopsis resident in a different
part of the brain from that where local disparities are initially coded? If so, it
should be possible to disturb global stereopsis without necessarily impairing local
stereopsis and stereoacuity. We present evidence that local and global stereopsis
can be dissociated by brain damage. Of course, we are not the first to have this
idea. Examination of human patients with brain damage has shown that global
stereopsis, measured with random letter stereograms, may be very impaired even
though stereoacuity is within the normal range (see Hamsher (1978a) for review).
The site of the critical brain damage is not known but the temporal lobes are
implicated. Other brain lesions involving the parietal lobes may impair stereo­
acuity, but unfortunately global stereopsis was not examined (Rothstein &
Sacks 1972).

A quite different reason for suspecting that the temporal lobes are involved in
global stereopsis arises from the experiments of Gross and his colleagues (Gross
et al. 1969, 1972; Gross 1973; Rocha-Miranda et al. 1975; Gross & Mishkin 1977). They have shown that neurons in a region extending caudally from the inferior occipital sulcus to near the temporal pole rostrally (see figure 1) have huge visual receptive fields that always include the fovea and often cross the vertical retinal meridian. The ipsilateral components of these fields are much greater than can be accounted for by the interhemispheric connections of the striate cortex and visual areas II and III of the occipital lobe, as these connections join only the representations of the vertical meridian (Zeki 1974, and this symposium). The median size of these fields is about 20° across, and some are as large as 70°. In terms of receptive field size, only they have the necessary property for coding multiple identical disparities over a large area of the visual field. Although their response to disparity has not been investigated, many of the neurons have very complex trigger features of the kind that would be provided by a multi-contoured visual object. Furthermore, they obtain their input from striate cortex via several synapses involving intervening visual areas where local disparity is coded (Hubel & Wiesel 1970; Fischer & Poggio, this symposium). If these temporal areas receive information about local disparities from the occipital lobe and use it to create global stereopsis, it should be possible to impair global stereopsis selectively by removing parts of the inferotemporal neocortex.

AN EXPERIMENT

We trained rhesus monkeys (Macaca mulatta) to look at two random dot stereograms in anaglyph format. Each stereogram consisted of an array of red and green dots. When examined with a red filter in front of one eye and a green filter in front of the other, each eye saw only one set of dots. In the centre of the array one set of dots was shifted laterally, leading to the perception of a central figure (a square) in a different depth plane from the surround. By taking two identical stereograms and inverting one of them, which reversed the direction of the horizontal shift, we had two stimuli in which the central figure lay in front of the surround in one and behind it in the other. Each monkey was trained to look at the stereograms and respond consistently either to the one with a global figure in front of the surround or to the other. Details of the testing procedure and of the numerous controls which demonstrate that the monkeys are not discriminating anything but disparity cues are given in experiment IV of Cowey et al. (1975). Briefly, the animals wore red/green goggles and sat in a restraining chair for each daily testing session of approximately 30 min. The chair was placed in a testing chamber containing an opaque screen between the animal and the stimuli. When the screen was raised, the animal saw a transparent board reclining at 25° from vertical, holding two stereograms 30 cm apart and 45 cm from the animal's eyes and beyond its reach. After 5 s the display was moved forward 15 cm and the animal allowed to displace one stereogram. Each stereogram had a foodwell beneath it and a peanut was placed in the well behind the positive stereogram. From trial to trial the positive and negative stimuli appeared randomly on the left and right and in
addition each stereogram was made positive or negative by rotating it, again randomly. All stereograms were taken from two copies of the sets provided by Julesz (1971) and are referred to by their numbers in the book.

We trained 13 rhesus monkeys to discriminate the relative depth in two stereograms to a criterion of 90 correct responses in 100 consecutive trials. Each stereogram was a 100 x 100 array of cells with a central shifted array of 40 x 40. At the 30 cm viewing distance each stereogram subtended 19° with a central square of 7.5°. Angular size of dots and disparity were approximately 12' and 50' respectively.

We then introduced a new task in which seven new pairs of stereograms were used, in random order, for 49 trials per session (series 8.1-2, Julesz 1971). The stereogram pairs differed in that the dots were systematically decorrelated across the entire array from perfect correspondence to only 40% correspondence, in steps of 10%. The effect for a human observer was to make it progressively more difficult to perceive the central square and its apparent depth and the stimuli can be used with people to assess stereopsis deficiency (Julesz 1971). Each monkey was tested until no further improvement in discrimination occurred, i.e. in 32 consecutive testing sessions totalling 1568 trials the difference in percentage correct in the first and second 16 sessions did not exceed five percentage points for any of the seven stereogram pairs. Each stereogram subtended approximately 15° with a central square of 8.5°, the size of the elements was 10', and the disparity of the correlated shifted elements relative to the surround was 20'.

We now removed the following cortical areas bilaterally by sub-pial aspiration

Figure 1. Schematic drawing of cortical lesions in the right hemisphere, with three of the sulci opened. Heavy stippling shows lateral striate ablation, and V2 lesion is indicated by black dots. Within the region shown by open circles the posterior half (pre-striate), middle half (posterior inferotemporal), or anterior half (anterior inferotemporal) was removed. l.s., lunate sulcus; i.o., inferior occipital sulcus; s.t., superior temporal sulcus; v.m. and h.m., representations of vertical and horizontal retinal meridia respectively in striate cortex.
under general anaesthetic. (1) In two monkeys the lateral striate cortex corresponding to the central 5° of vision (Daniel & Whitteridge 1961) was removed. This operation produces a central field defect and impairs visual acuity (Weiskrantz & Cowey 1963; Cowey 1967). It also deprives secondary visual areas of their major input from that part of striate cortex concerned with central vision (Zeki 1974). The extent of the lesion is shown by stippling in figure 1. (2) In four monkeys we

removed that part of visual area 2 (Zeki 1974) corresponding to the central 2–3° of vision. This region adjoins V1 and occupies the caudal bank of the lunate sulcus and the superior bank of the inferior occipital sulcus, together with cortex on the lateral surface. Neurons tuned to local disparity occur in V2 in both cat and monkey (Barlow et al. 1967; Hubel & Wiesel 1970; Fischer & Poggio, this symposium). (3) In two monkeys we removed the posterior half of the region shown by open circles in figure 1. We shall call this a prestriate lesion as it coincides with the foveal prestriate lesion studied by Cowey & Gross (1970). Many of the neurons with large receptive fields crossing the mid-line lie in this region. (4) In two monkeys we removed the central portion of this region, from the inferior occipital sulcus caudally to the middle of the inferior temporal cortex rostrally. (5) Finally, in three monkeys we removed the anterior half of this region. With the exception of

Figure 2. (a)–(e) Percentage correct responses pre-operatively (●) and post-operatively (○) with varying binocular correlation for the five groups of monkeys. (f) Percentage correct responses with easy and difficult stereograms in all of which binocular correlation was 1.0. See table 1 for details of these stereograms. a.i.t., anterior inferotemporal; p.i.t., posterior inferotemporal; p.s., prestriate.
the two animals in group (1), which are still being tested, all of the other lesions have been verified histologically and full details will be published separately.

The results are shown by the open circles in figure 2a–e. Neither the lateral striate lesions (V1) nor the central V2 lesions impaired global stereopsis. The former group improved slightly after operation, indicating that even our strict criterion of stability had not ensured that all animals had reached the limits of depth discrimination before operation. By contrast, all three groups with temporal lobe damage were impaired, and there is an indication that the more posterior the lesion the greater the impairment. Although figure 2 shows only mean results, no animal in groups 1 and 2 was impaired whereas every animal in groups 3–5 was impaired.

### Table 1. Details of the Ten Stereograms Used Post-Operatively

<table>
<thead>
<tr>
<th>Stereogram Pair</th>
<th>Code Number†</th>
<th>Element Size (arc min)</th>
<th>Disparity (arc min)</th>
<th>Orientation (arc min)</th>
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<tr>
<td>1</td>
<td>2.4-1</td>
<td>12</td>
<td>60</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>5.5-4</td>
<td>12</td>
<td>42</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>3.10-5</td>
<td>2–7</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>5.4-3</td>
<td>30</td>
<td>30</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>A</td>
<td>12</td>
<td>24</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>4.5-2</td>
<td>2</td>
<td>22</td>
<td>0</td>
</tr>
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<td>3.6-1</td>
<td>2</td>
<td>12</td>
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<td>8</td>
<td>5.3-1</td>
<td>18 x 3</td>
<td>20</td>
<td>± 7.5</td>
</tr>
<tr>
<td>9</td>
<td>5.3-2</td>
<td>18 x 3</td>
<td>20</td>
<td>± 15</td>
</tr>
<tr>
<td>10†</td>
<td>3.6-3</td>
<td>2</td>
<td>12</td>
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</table>

† From Julesz (1971).
‡ The elements consisted of horizontal lines with vertical breaks, and it was the minute breaks that were disparate in the eyes.

Before accepting that this experiment demonstrated an impairment of global stereopsis following temporal lobe damage we had to eliminate the following problem. When the dots in a stereogram are progressively decorrelated, human observers report that the global figure disappears first, leaving a condition where individual dots or clusters are still seen in depth. This distinction is often called surface depth as opposed to dot depth. As the monkeys with temporal lobe damage were most impaired at low binocular correlation, have we simply shown that stereoscopic perception of a global figure is normal whereas the perception of local disparities of clusters of dots in a degraded stereogram is impaired?

We therefore presented the monkeys with a task they had not encountered pre-operatively. We selected 10 new pairs of stereograms in all of which the binocular correlation was 1.0. In pairs 1–5 the elements and the disparity were large, yielding a prominent global figure and clear impression of depth to human observers. In pairs 6–10 the global perception of the central square was made progressively more difficult by reducing the size of the elements and their disparity, by misaligning their orientation in the two eyes, and finally by selecting
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a disparity consisting only of minute vertical shifts in horizontal lines. Each stereogram subtended 19° at 30 cm, with a 7.5° central square. Details are given in table 1. The 10 stereogram pairs were presented in random order in sessions of 50 trials for a total of 1200 trials. Figure 2f shows the percentage correct choices over 120 trials for each stereogram pair for the four monkeys with V2 ablation and the seven monkeys with prestriate, posterior inferotemporal, or anterior inferotemporal ablation. The scores of the latter animals were combined to form one group, as there were no differences among them. No animal in this combined group performed better than expected by guessing with the five difficult stereograms, whereas they were not significantly different from the others with the easy stereograms. Their defect is therefore not restricted to stereopsis with degraded binocular correlation and appears to involve global stereopsis. It should be stressed that although the four monkeys with V2 ablation were much better with the difficult stereograms than the other group, they may have been impaired. The absence of normal unoperated control animals with similar previous experience makes it impossible to tell.

Discussion

We must ask, ‘What has been established, what does it mean, and what remains to be done?’ It is clear that temporal lobe damage impairs global stereopsis, measured psychophysically by varying binocular correlation and calculating a threshold, or assessed more qualitatively by presenting stereograms with identical binocular correlation but varying in disparity, size, number, and orientation of the elements. This result should be contrasted with a number of others showing no change in the visual fields or in the threshold for acuity, flicker fusion, detection of a flash, and visual masking (see Gross (1973) for review). Whatever causes the defect it is not a general deterioration of visual thresholds. It is equally clear that removing those portions of V1 or V2 concerned with central vision does not impair global stereopsis per se, whereas the former lesion greatly elevates stereo-acuity thresholds in monkeys (Cowey & Wilkinson, in preparation).

These findings confirm findings on patients with brain damage (see Hamsher 1978a, b) and indicate that global stereopsis is mediated in temporal lobe areas that receive information about local disparities from the primary and secondary visual areas of the occipital lobe. But why is global stereopsis intact after the destruction of areas in V1 and V2 where local disparity is coded (Hubel & Wiesel 1970; Fischer & Poggio, this symposium)? The answer may be simple. Destruction of those parts of V1 and V2 concerned with central vision should affect only a small region of the central visual field and, provided the stimuli and their disparities are resolvable by extrafoveal regions, the global mechanism can make use of local disparity information from the remaining portions of V1 and V2. But if the size and disparity of the elements in random element stereograms are so small that they can be resolved only by the central few degrees of the retina, both local and global stereopsis should be impaired by our V1 and V2 lesions.
There is one other possible explanation of the disturbance of global stereopsis after brain damage in monkeys and people. Only in the temporal neocortex have neurons been described that have large receptive fields that extend more than 1 or 2° into the ipsilateral visual field. Removing the inferior temporal neocortex may prevent the brain from relating disparity information from both halves of the visual field: in effect the left and right halves of a stereogram whose centre is fixated may be processed separately without any possibility of relating them to form a global percept of the entire figure. Although this suggestion is compatible with the temporal lobe being critically involved in global stereopsis, there are no good grounds for rejecting the idea that many cortical visual areas may be concerned in global stereopsis and that the only unique feature of the temporal lobe is that its neurons have access to multiple disparity cues over a wider region of the visual field and from both halves of the visual field.

We can now assess what remains to be investigated to solve these problems. Tests with human patients may show whether it is specifically temporal lobe damage that impairs global stereopsis and whether patients in whom stereoacuity is impaired following parietal lobe damage are not also impaired on tests of global stereopsis with stereograms with small disparity cues. It is difficult to imagine that they will not be impaired if the global mechanism depends on input from local disparity detectors. The same problem could easily be investigated with monkeys, as can the contribution of V2 to stereoacuity. It will also be important to measure stereoacuity after temporal lobe ablation to show whether the impairment in global stereopsis is not a trivial consequence of impaired local stereopsis. The role of other cortical visual areas in local and global stereopsis remains to be examined. V3 and the region in the depths of the posterior quarter of the superior temporal sulcus (s.t.s.) both receive a projection from V1 (Zeki 1974) and cells in V3 may respond to local disparity (Hubel & Wiesel 1970) and those in s.t.s., to changes in disparity (Zeki 1974). Finally, the role of the temporal lobes in integrating global disparities between the right and left visual fields is more difficult to evaluate, but tests with normal human subjects viewing whole stereograms or only half of the same stereogram should indicate whether functionally isolating the halves by bilateral removal of temporal neocortex may explain the impairments in monkeys. If it is as easy for a normal observer to perceive the apparent depth in a half stereogram confined to one side of the vertical retinal meridian as in a whole stereogram fixated centrally, then isolating the two halves of a whole stereogram by a brain lesion should not impair global stereopsis and this explanation can be discounted. We are now testing these various ideas.
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References (Cowey & Porter)


