Cortical areas mediating stereopsis in the human brain: a PET study

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Using PET, we investigated the neural substrates of stereodepth perception in humans. The presentation of Julesz-type random-dot stereograms (RDS) produced significant rCBF elevations in Brodmann areas (BA) 18, 19 and 7, all in the right hemisphere. Activation foci were also found in both middle temporal areas (MT). These results demonstrate that, as in primates, cortical area MT and extrastriate areas are central to stereovision and that a network of predominant right hemispheric regions is recruited to meet visuo-spatial processing demands associated with horizontal binocular disparity inputs. *NeuroReport* 13:895–898 © 2002 Lippincott Williams & Wilkins.

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INTRODUCTION

Stereopsis is traditionally defined as the perception of depth based on small positional differences between images formed on the two retinae (binocular horizontal disparity) [1]. Single unit recordings (for review see [2]) carried out in a variety of non-human primates implicate visual cortical areas beyond striate cortex (V1 or BA 17) in stereopsis. Compared to V1, more cells in extrastriate regions V2, V3 and V5 (middle temporal cortex (MT)) are tuned to retinal disparity [3,4]. Moreover, striate cells appear to be equally sensitive to both anti-correlated and correlated random-dot stereograms (RDS) [5], suggesting that their activity cannot totally account for the perception of stereopsis and that further processing of their signals is necessary for perception of depth [6]. The MST (middle superior temporal area) and MT therefore appear to be likely candidates in the processing of stereodepth since they contain an abundance of disparity-sensitive neurons [7] and electrical stimulation of clusters of disparity-selective neurons can bias the behavioral perceptual judgement of depth [8]. In monkeys, it has been demonstrated that 53% of neurons located in the lateral bank of the intraparietal sulcus (area CIP, adjacent to area V3A), showed selectivity to binocular disparity cues in RDS without perspective cues [9]. This selectivity for RDS may be a result of higher-order processing of binocular disparity beyond the prestriate cortices. With the evidence from animal studies, it is reasonable to assume that higher-order structures in the visual system process stereocopic cues more efficiently.

Despite the advent of neuroimaging techniques, little is known about the neural substrates of stereopsis in humans. The few studies that have been carried out hardly corroborate the non-human primate work, and are often in opposition. In particular, the importance of striate activation and activation of area MT in processing stereoscopic cues are in dispute. Some studies find activations at the earliest Brodmann visual areas (BA 17) [10–13] while others do not [14,15]. MT activation in stereopsis is also unclear. Consistent activation of area MT has been found only for motion stereopsis [13], but the relative contributions of motion and stereodepth are ambiguous. Parietal activations (BA 7 or 40) were also observed in some studies [11–14], suggesting that the superior parietal lobule could be a higher center for stereopsis.

To date, the neuroimaging work done on stereopsis has been unable to clearly establish the neural components associated with stereodepth perception. In the present study we examined the changes in regional blood flow (rCBF) associated with stereodepth perception using horizontal disparity cues. We report that, as in monkeys, both the middle temporal area (MT) and extrastriate cortices are involved in stereo-processing.

MATERIALS AND METHODS

Subjects: Fifteen volunteers (age 21–31 years) participated in the study. All had normal stereopsis, as determined psychophysically while the subject was lying in the PET scanner prior to the actual recording. All participants gave written informed consent in accordance with the guidelines approved by the Montreal Neurological Institute Ethics committee.

PET imaging: The PET scan session consisted of two conditions, disparity and form, repeated three times at random. Thus, the data set consisted of six scans per subject for a total of 90 CBF volumes. Subjects wore a green filter over the left eye and a red one over the right during the scans. Prior to the experimentation, a fine needle-catheter was inserted into the bracchial vein for administration of a small amount of a short-lived radioactive substance (12 injections of 10 mCi $H_2{}^{15}O$). All scans were carried out 10 s after the subject viewed the stimuli on the television screen (Multisync XP 21 inch, resolution 832 × 624) placed 57 cm from the eyes. The subjects were asked to concentrate on the resulting percept, since they would subsequently be asked to describe what they saw. Images of each subject's brain were collected with high-resolution MRI scans. Individual MRI scans were spatially normalized to a standard stereotactic space [16] and co-registered with the PET images. PET images were reconstructed as 128 × 128 matrices of 2 × 2 × 2 mm pixels using an 18 mm Hanning filter to overcome residual anatomic variability persistence after stereotaxic standardization, normalized for rCBF for all intracerebral voxels. Individual MRI images were subjected to the same averaging procedure. MRI and PET images were merged to allow direct localization of *t*-statistic peaks.

Stimuli: The two stimuli were created with equal transparency, same dot size and density and same average luminance. The surround square measured 27.16 cm (280 pixels) and the central square 19.4 cm (200 pixels; pixel size: 0.097 cm). At a distance of 57 cm ($1^\circ = 1$ cm), the visual angles subtended by the stimuli were 27.16° and 19.4°, respectively, in all conditions.

The disparity-based stimulus (RDS) consisted of a central square composed of correlated dots (50% black and 50% yellow) having a horizontal disparity (23.37 min of arc) and surrounded by random dots. The colored filters made the square appear colorless and floating out in space (crossed stereopsis; Fig. 1b, left). The form stimulus was also a square made out of random-dots with zero disparity, which resulted in a visible central square. All subjects reported the perception of the square without depth.

Statistical analysis: The presence of a significant focal change was tested by a method based on the 3D Gaussian random-field theory [17]. Values equal to or exceeding a criterion of $t = 3.5$ were considered significant ($p < 0.0004$, 2-tailed, uncorrected).

RESULTS

To highlight the brain areas showing a change in rCBF when pure binocular disparity was isolated, we performed a voxel by voxel subtraction of disparity-based stereogram minus form. Activated brain regions, Talairach coordinates and *t* values are reported in Table 1. The corresponding PET images superimposed on 3D slices of anatomical MR images are illustrated in Fig. 1. Significant rCBF increases were found in the right occipital lobe (BA 18 and 19), the right superior parietal lobule (BA 7) and in the middle temporal lobe (area MT) in both hemispheres, with right-sided predominance. This right hemispheric dominance is clearly shown on the PET images in Fig. 1.

Table 1. Brain areas showing significant activations in the experimental conditions.

Brain areas	H	X	Y	Z	*t*
BA 18	R	15	−68	−6	3.5
BA 19	R	39	−78	15	4.7
MT	R	39	−62	−4	3.9
	L	−50	−59	0	3.6
BA 7 (SPL)	R	35	−52	60	4.1

MT, middle temporal area; SPL; superior parietal lobule. Coordinates: x: left/right; y: anterior/posterior; z: inferior/superior. *H*: hemisphere; L: left; R: right; *t*: statistical value

DISCUSSION

Our results are in agreement with numerous studies carried out in monkeys, which have shown that the perception of depth based on spatial disparity cues lies outside striate cortex. In animals, disparity sensitive neurons are distributed at several levels along the dorsal visual pathway including V2, V3 and V5 (MT). In contrast to recent brain imaging studies on stereopsis [13], we suggest that area MT is important in static depth perception in humans. Lesion studies seem to support our results since temporal lobe lesions lead to an impairment in stereopsis [18]. This deficit in stereopsis was reported for right as well as for left temporal lobectomy and was more marked following right-sided excisions, which is coherent with our observation of bilateral MT activation and predominant recruitment of right hemisphere structures [10,13,14]. It is well established from monkey anatomical studies that the middle temporal lobe is part of the dorsal visual pathway and receives visual information from posterior visual areas such as BA 18 and 19 en route to the parietal lobe [19]. Our results indicate that this hierarchical organisation of the visual pathway in the processing of visual information is also valid in humans.

Since the superior parietal cortex acts as the output of the dorsal pathway of visual information to construct the object location [19], it is not surprising that in our study this cortical area was strongly activated by our depth-producing stimuli. In monkeys, neurons in the caudal part of the lateral bank of the intraparietal sulcus respond to binocular disparity, 25% of them being predominantly selective for RDS and 28% showing selectivity for RDS and solid figure stereograms [9]. Moreover, our study support the previous brain imaging findings [14] underlying the important contribution of the superior parietal lobe in stereopsis. Gulyas and Roland [11,12] and Nagahama *et al*. [13] also reported parietal activation (BA 7 and 40) in a static depth perception task. Patients with parietal lobe lesions show severe impairments in stereopsis [20], adding support to the pivotal role played by this cortical area in stereoperception. The importance of the parietal lobe in various aspects of visual perception such as motion-in-depth [13,21], optic flow (for review see [22]), biological motion [23], and perception of space [24] has been well documented in recent years. This area of integration of various perceptual and other sensory modalities is perfectly suited to guide movements on the basis of visual information, which appears now to be another function of the dorsal stream (the 'how' [19] and the 'where' [25] systems).

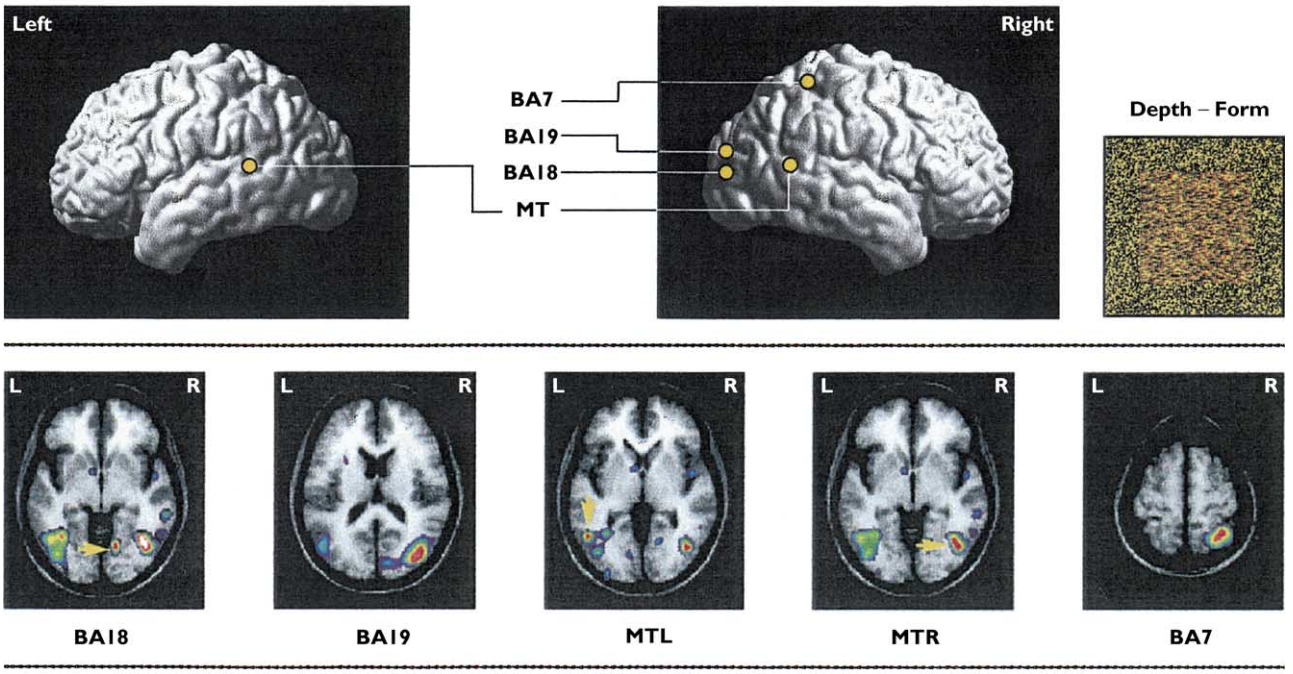

Fig. 1. Activation sites for the disparity-based condition. In the upper portion of the figure, the coordinates (derived from the sections in the lower part) of activated cortical regions are placed on 3D cerebral hemispheres and evidence the activation of the dorsal visual pathway in stereopsis. Note the bilateral activation of area MT. Lower portion: the disparity-based stimulus is on the left (the stereo effect can been seen by wearing red-green filters) and the sections showing the significant rCBF elevation sites (yellow arrows) are depicted on the right. The left side of the brain is on the left. MTL: left area MT; MTR: right area MT.

However, we failed to show a significant activation of area V1 (BA 17) in the present experiment. This is consistent with the results obtained in some studies [14,15,26] but at odds with others [10–13]. Contradictory findings regarding the implication of BA 17 in stereopsis might be related to the experimental paradigm and stimulus parameters used in the different investgations. RDS are considered second-order stimuli since the shape in the stereogram is not defined by luminance [27]. Studies have shown that second-order stimuli are processed primarily by higher order visual cortical areas [27]. In some experiments that have demonstrated the activation of BA 17, first-order stimuli (solid figure stereograms) were used [14]. Others used different control conditions than our own to isolate depth processing. For example, in the study by Ptito *et al*. [10], the influence of contour was subtracted from a solid rectangle defined by stereopsis. This assumes that the contour is sufficient to account for the entire object. In our case, the control stimulus included the entire surface, which is analogous to the solid depth object, therefore allowing the isolation of pure depth. In the former experiment, the object surface was not subtracted. This may explain the differences between studies in terms of V1 activation. As stated previously, our data confirm both recent brain imaging studies [14,15,26] and investigations in monkeys showing the implication of structures forming the dorsal visual pathway in stereoscopic processing [4].

CONCLUSION

These results clearly establish that in normal human subjects depth perception based on positional disparity is processed outside BA 17, in so-called higher order areas located in the occipital (BA 18 and 19) and superior parietal lobes (BA 7). Moreover, our results support the idea that, as previously suggested by studies in monkeys and humans, area MT is implicated in the processing of static binocular disparities. The present experiment also leads to the conclusion that the right hemisphere may be dominant for static stereopsis [10,13] as well as for motion-in-depth [21].

REFERENCES

1. Howard IP and Rogers BJ. *Binocular Vision and Stereopsis*. Oxford: Oxford University Press; 1995.
2. Gonzalez F and Perez R. *Prog Neurobiol* **55**, 191–224 (1998).
3. Poggio GF, Gonzalez F and Krause F. *J Neurosci* **8**, 4531–4550 (1988).
4. DeAngelis GD, Cuming BG and Newsome WT. *Nature* **394**, 677–680 (1998).
5. Cumming BG and Parker AJ. *Nature* **389**, 280–283 (1997).
6. Cumming BG and Parker AJ. *J Neurosci* **19**, 5602–5618 (1999).
7. Roy JP, Komatsu H and Wurtz RH. *J Neurosci* **12**, 2478–2492 (1992).
8. De Angelis GC, Cumming BG and Newsome WT. *Nature* **13**, 677–680 (1998).
9. Taira M, Tsutsui K-I, Jiang M *et al*. *J Neurophysiol* **83**, 3140–3146 (2000).
10. Ptito A, Zatorre R, Petrides M *et al*. *Neuroreport* **4**, 1155–1158 (1993).
11. Gulyas B and Roland P. *Proc Natl Acad Sci USA* **91**, 1239–1243 (1994).

12. Gulyas B and Roland P. *Eur J Neurosci* **6**, 1811–1828 (1994).
13. Nagahama Y, Takayama Y, Fukuyama H *et al*. *Neuroreport* **7**, 1717–1721 (1996).
14. Nishida Y, Hayashi O, Iwami T *et al*. *Neuroreport* **12**, 2259–2263 (2001).
15. Mendola JD, Dale AM, Fischl B *et al*. *J Neurosci* **19**, 8560–8572 (1999).
16. Talairach J and Tournoux P. *Co-Planar Stereotaxic Atlas of the Human Brain*. New York: Thieme; 1988.
17. Worsley K, Evans AC, Marrett S *et al*. *J Cerebr Blood Flow Metab* **12**, 900–918 (1992).
18. Ptito A, Zatorre RJ, Larson WL *et al*. *Brain* **114**, 1323–1333 (1991).
19. Milner AD and Goodale MA. *The Visual Brain in Action*. Oxford: Oxford University Press; 1995.
20. Rothstein TB and Sacks JG. *Am J Ophthalmol* **73**, 281–284 (1972).
21. Ptito M, Kupers R, Faubert J and Gjedde A. *NeuroImage* **14**, 1409–1415 (2001).
22. Bremmer F, Duhamel JR, Ben Hamed *et al*. Stages of self-motion processing in primate posterior parietal cortex. In: Lappe M (ed). *Neuronal Processing of Optic Flow. International Review of Neurobiology*, vol. 44. San Diego: Academic Press; 2000, pp. 173–198.
23. Bonda E, Petrides M, Ostry D and Evans A. *J Neurosci* **16**, 3737–3744 (1996).
24. Cabeza R and Nyberg L. *J Cogn Neurosci* **12**, 1–47 (2000).
25. Ungerleider LG and Haxby JV. *Curr Opin Neurobiol* **4**, 157–165 (1994).
26. Savoy RL, Tootell RBH, O'Craven KM and Reppas JR. *Human Brain Mapp (Suppl.)* **1**, 57 (1995).
27. Baker CL Jr and Mareschal L. *Prog Brain Res* **1**, 171–191 (2001).

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