

Neuroscience

Awareness of space

Michael S. A. Graziano

Damage to particular parts of the brain can cause spatial confusion and even eliminate awareness of areas of space around the body. The brain regions responsible for spatial awareness, however, are still under debate.

Certain types of brain damage can lead people to suffer from profound spatial disorientation and an inability to use spatial information to direct their movements — symptoms first described in detail near the beginning of the last century^{1,2}. Some of the people who suffer from such damage, to the right posterior parietal lobe of the brain's cortex, show a phenomenon called hemispacial neglect³, whereby they lose their awareness of the half of the space around them that lies opposite to the brain lesion. Someone with hemispacial neglect might shave only half of his face, dress just half of his body, or, when copying a picture, draw only one side of it.

On page 950 of this issue, Karnath and colleagues⁴ revisit the topic of spatial neglect. They look at a set of patients with an

especially pure form of the deficit, who lack some of the other symptoms that can occur together with hemispacial neglect after damage to the posterior part of the brain. The results threaten to overturn the century-old view that the parietal lobe (Fig. 1) is the region of the brain associated with spatial awareness.

The hypothesis that the parietal lobe is the key brain region for processing spatial information is based almost exclusively on studies of humans who have suffered damage to this part of the brain, often as the result of a stroke. Attempts to reproduce hemispacial neglect in monkeys by surgically damaging the parietal lobe have not been successful^{5,6}. Such animals do have defects in processing somatosensory (tactile or heat) stimuli and in directing hand and eye move-

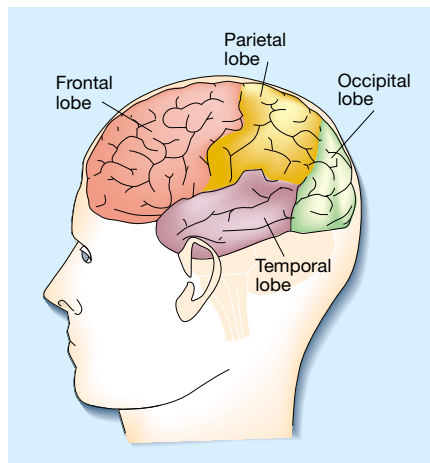


Figure 1 The human brain. Karnath and colleagues⁴ have shown that the topmost part of the temporal lobe is involved in spatial awareness.

ments in space, as do humans with parietal brain damage. But monkeys with parietal damage do not appear to develop hemispatial neglect.

One early study⁷ of the properties of single neurons in the monkey parietal lobe led to the conclusion that much of this region has motor functions — that is, it is involved in producing movements. More recent single-neuron experiments indicated that different subregions of the monkey parietal lobe may be specialized for using sensory information to guide different types of movement, such as movements of the eyes or arms, or shaping the hand for grasping^{8,9}. These subregions connect to specific parts of the motor cortex in the frontal lobe. It has been argued¹⁰ that at least some humans with parietal damage have difficulties mainly in using spatial or sensory information to guide movements, rather than in spatial awareness. So, there is quite strong evidence that the parietal lobe matches sensory input to specific patterns of motor output in both monkeys and humans. But the evidence for a role of the parietal cortex in spatial awareness is less convincing. The classical finding that parietal damage causes hemispatial neglect in humans was ripe for re-examination, and this is what Karnath *et al.*⁴ have done.

The problem is that strokes, or other types of brain damage, usually affect a large part of the brain, so the area responsible for hemispatial neglect in humans has never been known precisely. Karnath *et al.* set out to track down this region. They first selected patients who had damage to the cortex in the right posterior hemisphere. They studied four categories of patients: those with symptoms of hemispatial neglect combined with blindness in parts of the visual field (a typical combination); those with hemispatial neglect but no evidence of blindness; those with blindness but no

hemispatial neglect; and those with neither set of symptoms.

The authors found that the patients with both hemispatial neglect and blindness in parts of the visual field tended to have damage centred on the posterior parietal lobe, fitting with the past century of observations. But the lesions were large, and typically also included the superior temporal lobe. The patients with hemispatial neglect but no blindness had smaller lesions centred on the superior temporal lobe. The few patients with visual-field defects but no neglect had damage that involved the posterior parietal lobe but not the superior temporal lobe. Finally, patients with neither set of symptoms showed no tendency for either the posterior parietal lobe or the superior temporal lobe to be affected. The implication is that damage to the superior temporal lobe is responsible for hemispatial neglect, whereas parietal damage underlies the visual-field defects, perhaps because of injury to the adjacent band of nerve fibres that transmits information to the primary visual cortex. (One might also expect the patients with parietal damage to have problems in integrating sensory and motor information, but this was not tested.)

If confirmed, the finding that spatial neglect is associated with the superior temporal lobe would help to resolve the confusion in the literature. One could then view the human parietal lobe as being involved primarily in matching sensory input and motor output, as it seems to be in monkeys. Spatial awareness or spatial cognition, independent of the control of specific

movements, would depend on other brain structures. These probably include not only the superior temporal lobe but also parts of the frontal lobe, as frontal lesions in both monkeys and humans sometimes cause symptoms of spatial neglect¹¹.

Of course, the assignment of particular functions to specific brain regions can only be taken so far. Brain areas that link sensory and motor information, like those in the parietal lobe, must have some role in spatial awareness, given the apparently close link between planning to make a movement to a location in space and directing attention to that location¹². After all, researchers in the field of systems neuroscience aim not only to determine the properties of specific brain areas, but also to understand how mental functions can emerge from the interactions among different regions. ■

Michael S. A. Graziano is in the Department of Psychology, Green Hall, Princeton University, Princeton, New Jersey 08544-1010, USA.
e-mail: graziano@princeton.edu

1. Holmes, G. *Br. J. Ophthalmol.* **2**, 449–516 (1918).
2. Balint, R. *Monatsschr. Psychiatr. Neurol.* **25**, 51–81 (1909).
3. De Renzi, E. *Disorders of Space Exploration and Cognition* (Wiley, New York, 1982).
4. Karnath, H.-O., Ferber, S. & Himmelbach, M. *Nature* **411**, 950–953 (2001).
5. Ettlinger, G. & Kalsbeck, J. E. *J. Neurol. Neurosurg. Psychiatry* **25**, 256–268 (1962).
6. Lamotte, R. H. & Acuna, C. *Brain Res.* **139**, 309–326 (1978).
7. Mountcastle, V. B., Lynch, J. C., Georgopoulos, A., Sakata, H. & Acuna, C. *J. Neurophys.* **38**, 871–908 (1975).
8. Snyder, L. H., Batista, A. P. & Andersen, R. A. *Nature* **386**, 167–170 (1997).
9. Sakata, H. & Taira, M. *Curr. Opin. Neurobiol.* **4**, 847–856 (1994).
10. Goodale, M. A. *et al. Curr. Biol.* **4**, 604–610 (1994).
11. Husain, M. & Kennard, C. *J. Neurol.* **243**, 652–657 (1996).
12. Moore, T. & Fallah, M. *Proc. Natl Acad. Sci. USA* **98**, 1273–1276 (2001).