Chronic unilateral visuo-spatial neglect is usually ‘defined’ clinically as a condition in which patients ignore objects and events in contralesional space. As such, ‘it’ has been observed after lesion to many distinct right hemisphere loci (and much more rarely after left hemisphere lesion). After cortico-subcortical damage, left neglect can be consequent upon right posterior parietal (Vallar and Perani, 1986), right temporal (Samuelsson et al., 1997), and right frontal (e.g., Damasio et al., 1980; Husain and Kennard, 1996) lesions. Neglect has also been reported after subcortical lesions of the thalamus (e.g., Watson and Heilman, 1979; Rafal and Posner, 1987), the basal ganglia (e.g., Damasio et al., 1980; Ferro et al., 1987) and the white matter/internal capsule (e.g., Healton et al., 1982; Ferro and Kertesz, 1984). There is, nonetheless, reasonable consensus that severe visuo-spatial neglect is most typically associated with lesion of the supramarginal gyrus of the right inferior parietal lobule – Brodmann’s area 39/40 (Vallar, 2001). Consensus is, of course, no guarantee of truth and some students have even argued that the detailed manifestations of so-called visuo-spatial neglect are too variable (both within- and between-patients) to be meaningfully united into one clinical entity (Halligan and Marshall, 1992; Stone et al., 1998).

A recent report by Karnath et al. (2001) confirms that unilateral visuo-spatial neglect (in patients who meet the customary clinical features thereof) can result from lesions of right temporal cortex and from lesions of right parietal cortex (Vallar, 2001). But, more controversially, Karnath et al. then make a partially novel functional and anatomical distinction between two types of spatial neglect. In “pure” spatial neglect there is purportedly loss of “spatial awareness” contralateral to the lesion; the locus of the responsible lesion is exclusively superior temporal, without extension into occipital cortex or the optic radiations (and hence the patients do not manifest visual field deficits). By contrast, in ‘impure’ spatial neglect, there is a disorder of “directly coding space for action” (but not apparently of spatial awareness); here the lesion includes the inferior-posterior parietal cortex but also extends into regions which, when damaged, produce visual field deficits (see also Karnath, 2001, for further discussion). The functional distinction that Karnath et al. (2001) draw has some similarities with the contrast that Poppelreuter (1917) made between the roles of attentional...
orientation and visual praxis in neglect; likewise, the distinction that Heilman et al. (1993) make between attentional and intentional neglect has some resemblance to that contrast.

The basic problem with the functional distinction as deployed by Karnath and colleagues (2001) is that they provide no indication of which diagnostic tests can distinguish between a disorder of hemi-spatial awareness and a disorder of coding (left) space for action. Indeed, all four visuo-motor tests deployed in their study draw upon the ability to code space for action. They are certainly not pure tests of “orienting towards and exploring the contralesional part of space or objects”, which is how Karnath (2001) characterizes the relevant functions of right superior temporal cortex. The problem with the anatomical distinction is that there are patients who show spatial neglect after occipito-temporal lesions and accordingly have a visual field deficit (Doricchi and Angelelli, 1999), and patients with spatial neglect after parietal lesions who do not have a visual field deficit (Karnath and Fetter, 1995; Husain et al., 2001). Which functional disorder does the hypothesis of Karnath et al. predict for these patients?

Many different tests of neglect have been proposed, some of which have been claimed to be more sensitive than others (Halligan et al., 1989). Of these tests, there are some (not used in the study of Karnath et al.) that could plausibly be interpreted as assessing disorders of spatial awareness without simultaneously involving “coding space for action”. These include the landmark task whereby patients judge whether a pretransected horizontal line is correctly bisected (Harvey et al., 1995), and the chimeric objects test whereby patients attempt to detect that a picture has been constructed from two incompatible halves (Buxbaum and Coslett, 1994). Likewise, there are patients who show ‘imaginal’ neglect and cannot report from memory the left side of a scene they are fully familiar with (Beschin et al., 1997). Patients with parietal lesions have been reported to be impaired on each of these tasks (Harvey et al., 1995; Buxbaum and Coslett, 1994; Beschin et al., 1997). Likewise, studies using functional neuroimaging in healthy volunteers demonstrate that right inferior parietal cortex is implicated in the explicit spatial judgments drawn upon by the landmark task (Fink et al., 2000). Furthermore, in healthy subjects, transient disruption of right posterior parietal cortex by repetitive transcranial magnetic stimulation (rTMS) provokes impairment on the landmark task similar to that found after structural lesions to that area (Fierro et al., 2000). Finally, there are patients with right parietal damage and left neglect who are unaware of visual stimuli they are nonetheless able to process for meaning (Berti and Rizzolatti, 1992).

Contrary to the position of Karnath and colleagues, all of these findings seem to indicate that a disorder of spatial awareness (without explicit coding of space for action) can be consequent upon damage to right parietal cortex. This conclusion is not inconsistent with the possibility that lesions of right temporal cortex may also impair contralesional spatial awareness. But the findings of Karnath et al. (2001) do not address these issues due to their exclusive use of tests that measure “coding space for action.” By contrast, there are at least two studies that plainly distinguish between (relatively well-preserved) spatial awareness (as indexed by verbal report) and (impaired) coding of space for action (as indexed by cancellation). One right brain-damaged patient reported by
Bottini et al. (1992) was able to identify target letters or bells among distracters in the left and right sides of space, but failed to cross out left-sided targets using the right hand. Patient J.R., reported by Marshall and Halligan (1995), was presented with Navon figures (hierarchical drawings in which a global form such as a letter is made up of smaller local letters). J.R. could correctly name each global letter (and the local letters) but could only cancel (with the right hand) the local letters on the right of each global figure.

Karnath et al. (2001) and Karnath (2001) attempt to bolster their neuroanatomical claims about neglect in human patients by reference to phylogenetic issues. Specifically, they assert that visuo-spatial neglect in monkeys is consequent upon lesions of either left or right superior temporal cortex. Accordingly, they conjecture that the crucial “transition from monkey to human brain seems to be a restriction of a formerly bilateral function to the right side, rather than a shift from the temporal to the parietal lobe” (Karnath et al., 2001). But even here, the consensus of informed opinion suggests that frontal and parietal lesions more typically provoke acute neglect-like symptoms in monkey (e.g., Heilman et al., 1993; Rizzolatti et al., 2000; Wardak et al., in press).

Although we find the psychological, neuroanatomical and evolutionary claims made by Karnath et al. (2001) implausible, they do serve to draw attention to an important issue: Visuo-spatial neglect fractionates into a wide range of behavioural subtypes, many of which can double dissociate (Halligan and Marshall, 1992) and hence place constraints upon the claim that some tests of neglect are intrinsically more ‘sensitive’ than others (Halligan et al., 1989). It is consistent with these behavioural dissociations that visuo-spatial neglect can be associated with lesions of a wide range of cortical and subcortical structures. In these circumstances, one might expect to discover reliable mappings between specific components of neglect and specific loci of damage (Vallar, 1994). But, thus far, attempts to specify such mappings in human patients have provided a rather mixed set of results (e.g. Bottini et al., 1992; Coslett et al., 1990; Na et al., 1998; but see McGlinchey-Berroth et al., 1996; Bisiach et al., 1998; Karnath et al., 2001). A radical rethink of this topic may be necessary (Young et al., 1999). Here the combination of lesion-based neuropsychological work and functional neuroimaging may contribute significantly (Marshall and Fink, 2001; Vallar, 2000).

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