Many patients who have suffered right hemisphere stroke seem initially unresponsive to voices and environmental noises that emanate from their left (Heilman and Valenstein, 1972). The question of whether more subtle deficits might persist post-acute, and whether, like visuospatial neglect, they really are more common after right than left hemisphere damage, has been comparatively little studied. Until fairly recently, auditory neglect was the poor relation of visual neglect.

In part, this is because there are no auditory analogues of simple search tasks such as target cancellation, or of drawing or copying. Auditory bisection performance, where the subject is required to point to a position in space midway between two sound sources, has been reported (Ruff et al., 1981): patients with right parietal lesions were less accurate than those with any other lesion site in terms of absolute error, but did not show a specific bias to the right. In terms of testing at the bedside, many examiners look for auditory extinction in a rough and ready fashion by clicking their fingers or jangling keys simultaneously on either side of the patient’s head. The auditory subjective straight ahead (SSA) can also be tested without elaborate apparatus. When more sophisticated methods are used (e.g. presentation over headphones of stimuli with interaural and monaural cues to sound location in virtual space), the SSA is frequently found to show a large ipsilesional deviation in patients with left visual neglect after right parietal damage. It is important to note that this deviation to the right can occur in patients with normal or near normal pure tone hearing in both ears (Kerkhoff et al., 1999; Tanaka et al., 1999).

Studies of sound localization over a wider spatial range have typically employed either loudspeakers arranged in a semicircle in front of the patient, or have simulated sounds coming from these locales by manipulating over headphones the interaural lag between sound reaching the left and right ears and the interaural intensity difference. Investigations using the latter, more reliable technique, have shown that maximal impairment for sound localization is seen in patients with right parietal damage and consequent unilateral visual neglect (Tanaka et al., 1999). Responses are often shifted in the direction of the ipsilesional right side across the entire half field; sounds coming from the far left may be reported as originating in right space (alloacousis). In a recent study, Pavani and colleagues obviated the potentially confounding factor of responding by manual pointing or moving a lever: same/different judgements for pairs of sequentially presented sounds coming from the same or different locales were studied (Pavani et al., 2001). Patients with visuospatial neglect after right hemisphere lesions (anterior or posterior) were severely impaired in comparison with right brain-damaged controls without visual neglect. The impairment was maximal when the sounds originated in left contralesional hemispace.

In some studies, left auditory neglect in patients with associated left visual neglect can be more severe when concomitant visual information is available. For example, left auditory neglect can be ameliorated by blindfolding the patient even in conditions when, with the eyes open, the loudspeakers themselves are shielded from the observer by a screen (Ladavas and Pavani, 1998). By contrast, a patient with biparietal damage showed that visual localization deficits could be reduced by simultaneous presentation of an unattended auditory stimulus that acted as an external reference point (Phan et al., 2000).

The issue is thus raised of whether visual neglect and auditory neglect are necessarily associated. That this is not so has been demonstrated by De Renzi and colleagues (De Renzi et al., 1989). Auditory neglect was tested by requiring patients to detect interruptions in an otherwise continuous pure tone. Patients with right hemisphere damage (parietal or thalamic) were more impaired on this task, with both monaural and binaural presentation, than were patients with left hemisphere damage. Furthermore, in the patients with right hemisphere lesions, failure to detect the interruption of the signal was most frequent when the gap occurred on the left ear. Crucially, some patients with left visual neglect did not show auditory neglect, and some patients with left auditory neglect did not show visual neglect. Dissociations within the auditory neglect syndrome are convincingly shown by Bellmann and colleagues in the present issue (Bellman et al., 2001). Patients with unilateral right-hemisphere lesions were selected on the basis of showing left-ear extinction on a dichotic listening task: one word was presented to the left ear while a different word was simultaneously presented to the right ear. In the experiment proper, extinction was tested
with diotic stimuli: with this technique, both ears received both words but the manipulation of interaural time differences made one word appear to originate from the left while the other seems to originate from the right. Left ear extinction with diotic listening was found in some patients without major deficits in sound localization; in other patients, a severe ipsilesional bias in sound localization could occur without left diotic extinction. The former deficit was associated with basal ganglia lesions, the latter deficit with frontotemporal parietal lesions (Bellmann et al., 2001). These findings hence distinguish between an impairment in the allocation of auditory spatial attention (diotic extinction), and a distortion of auditory spatial representations (rightward bias in sound localization).

Although patients with either visual or auditory neglect often have large lesions, functional neuroimaging in normal volunteers has confirmed the crucial role of parietal cortex in audio-spatial tasks. The detection of sound movement (produced by changes in the delay and amplitude of sound at the ears) has been shown, with both functional MRI (fMRI) and PET to involve the differential activation of right parietal cortex (Griffiths et al., 1998). Likewise, sound localization has been shown, with PET, to implicate the superior parietal lobule, middle temporal and lateral prefrontal cortices (Bushara et al., 1999). This study investigated visual and auditory spatial localization in the same healthy subjects, finding evidence for both multimodal and modality-specific regions. The former discovery may help explain clinical dissociations between visual and auditory neglect. Weeks and colleagues have shown (with PET) that sound localization when compared with a non-spatial auditory discrimination task selectively activates the right inferior parietal lobule (Weeks et al., 1999).

Most interestingly, Kaiser and colleagues, using wholehead magnetoencephalography (MEG), have shown that supratemporal and right posterior parietotemporal areas respond to both ipsilateral and contralateral sound shifts from the midline, whereas the equivalent left hemisphere regions respond only to contralateral shifts (Kaiser et al., 2000). As the authors state, these findings ‘help to explain why unilateral auditory neglect is found much more frequently for stimuli in the left hemifield after right parietal lesions than vice versa.’ One might also note the congruence with studies of the visual system which have likewise suggested that the right hemisphere is responsive to visual events in either hemifield, whereas the left hemisphere responds preferentially to stimuli in right hemispace (for discussion, see Halligan and Marshall, 1994).

In summary, more attention is at last being paid to the audio-spatial system in both clinical practice and basic science. But one desideratum that urgently requires more work is the development of audio-spatial tests that do not require complex equipment and an anechoic chamber, but can reliably and validly be used at the bedside.

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References