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Auditory neglect

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SUMMARY Auditory neglect was investigated in normal controls and in patients with a recent unilateral hemispheric lesion, by requiring them to detect the interruptions that occurred in one ear in a sound delivered through earphones either mono-aurally or binaurally. Control patients accurately detected interruptions. One left brain damaged (LBD) patient missed only once in the ipsilateral ear while seven of the 30 right brain damaged (RBD) patients missed more than one signal in the mono-aural test and nine patients did the same in the binaural test. Omissions were always more marked in the left ear and in the binaural test with a significant ear by test interaction. The lesion of these patients was in the parietal lobe (five patients) and the thalamus (four patients). The relation of auditory neglect to auditory extinction was investigated and found to be equivocal, in that there were seven RBD patients who showed extinction, but not neglect and, more importantly, two patients who exhibited the opposite pattern, thus challenging the view that extinction is a minor form of neglect. Also visual and auditory neglect were not consistently correlated, the former being present in nine RBD patients without auditory neglect and the latter in two RBD patients without visual neglect. The finding that in some RBD patients with auditory neglect omissions also occurred, though with less frequency, in the right ear, points to a right hemisphere participation in the deployment of attention not only to the contralateral, but also to the ipsilateral space.

Neglect phenomena following right brain damage have been extensively investigated in the visual modality, where they manifest themselves as failure to orient attention to left-sided stimuli, which may go undetected under conditions that should urge the patient to survey the whole space (for example, when required to read a sentence, which lacks meaning if only the words lying to the right are taken into account). While visual neglect is readily apparent on a series of tasks requiring the scanning of the environment, no convincing evidence of failure to respond to auditory stimuli having their source located in the left space has so far been provided. Right brain damaged patients do show errors of localisation and lateralisation of auditory stimuli, shifting them towards the right side[1 2] and sometimes answer a question addressed from the left turning their head and eyes to the right,[3] but they have never been reported to ignore a stimulus located in the left space, except in response to double simultaneous stimulation. Extinction, that is, the failure to perceive a stimulus in the contralateral ear when it is simultaneously delivered to the ipsilateral ear, has been observed in nearly 50% of unilaterally brain damaged patients in the early stage of a stroke,[4] but, contrary to what happens with visual neglect, no significant association of the deficit with right hemisphere lesions has been observed. This lack of asymmetry in auditory extinction, which has been confirmed in a recent investigation,[5] casts some doubt on the close relation of extinction to neglect. Moreover, there were patients with long lasting extinction,[4] whose injury was so located as to interrupt the central auditory pathways in their course from the medial geniculate body to the temporal cortex, thus making extinction more likely to be contingent on a sensory than an attentional impairment.

The difficulty of showing neglect in the auditory modality is probably due to the physical characteristics of acoustic stimuli and the arrangement of the neural apparatus transmitting them to the cortical centres. The sound waves emitted by a left sided acoustic source reach both ears, though for the right one with delay and decreased intensity,[6] and are thence sent to both temporal cortices, though the contralateral pathway is stronger. It follows that, when the right hemisphere is damaged, the left hemisphere remains able to perceive and attend to a sound coming from the left side, due to its connections with both

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ears, and can also lateralise it, though not accurately, by computing the time lag and the intensity difference with which the right ear has been stimulated. It would appear that a prerequisite for bringing out auditory neglect, if it indeed exists, is to confine the acoustic stimulation to the ear contralateral to lesion.

In this study we assessed the ability of brain-damaged patients with a recent hemispheric disease to monitor the interruptions that occurred at random intervals in one ear in a continuous sound delivered either unilaterally or bilaterally through earphones. We contrasted omissions in the ear contralateral and ipsilateral to the side of lesion and related them to the patients' manifestations of auditory extinction and visual neglect.

Material and methods

Subjects

Fifty five patients participated in this investigation. Ten were control (C) patients, hospitalised for diseases not involving the brain (mean age: 56·4 yr), 15 were left brain-damaged (LBD) patients (mean age: 61·2 yr) and 30 right brain-damaged (RBD) patients (mean age: 63·9 yr). Seven of the 15 LBD patients were aphasic. The side of the lesion was based on clinical evidence and confirmed by CT in all patients, except two RBD patients who had negative CT findings, though showing unequivocal signs (left hemiparesis and hemianopia, respectively) of right hemisphere damage. In four RBD patients CT also showed an old left vascular lesion that had been clinically silent and in a fifth patient two silent left hemisphere metastases in addition to a clinically evident metastasis of the right hemisphere. Since neglect was the main subject of this investigation, the RBD group selection was biased towards patients who presented clinically with signs of lateralised attentional disorders. Thus the frequency with which neglect appears in the present sample cannot be generalised to the RBD population.

Table 1 shows the clinical characteristics of brain-damaged patients.

Tests

Patients were first submitted to pure tone audiometry to rule out the presence of major peripheral deficits of hearing and to ascertain whether there was an asymmetry between the ear curves. If there was asymmetry the intensity of the test signal delivered to the impaired ear was increased so as to equate the two curves. It must be added that no patient with auditory neglect (see below) showed a difference in threshold between ears.

Auditory attention test

A sound generator delivered through headphones to the patient a continuous 3000 Hz pure tone at a level of 102 dB SPL; the sound lasted 4 minutes and was interrupted for 300 ms at intervals ranging from 8 to 20 ms. There were 24 interruptions, 12 per ear, to which the patient had to respond by pressing the space bar of a computer keyboard. The sound intensity could be adjusted independently in each channel in order to compensate for hearing deficits, so that a subjectively identical signal was provided to both ears in any case. The sound generator was linked to an Apple II E computer by a versatile interface adaptor (6522). A computer program controlled the interruption pattern, specifically the 300 ms duration, the intervals between interruptions and the side of interruption in the binaural test. Signal decay at interruption was very fast, about 3 ms. The test was presented in two versions, one mono-aural and the other binaural. In the *mono-aural test* the sound was first sent to one ear until the 12 interruptions had occurred and then to the other ear, so that the patient knew where he had to focus attention. The order of ear presentation was pseudo-random. In the *binaural test* both ears received the sound, but the interruptions occurred quasi-randomly to either ear, with the restriction of no more than three consecutive interruptions to the same ear. In this case, therefore, the patient had to attend to both ears in order to perceive when the sound was interrupted. The following day the entire session was repeated and thus patients received 48 signals per test, 24 in the right ear and 24 in the left ear. In the first session, patients were first familiarised with the procedure by the administration of a short version of either test with five 300 ms interruptions.

Auditory extinction

Stimuli were delivered with two sound generators (75 dB), kept manually at a distance of approximately 10 cm from the ears, and activated by pushing a button. Ten bilateral stimuli were administered, intermingled with 20 unilateral stimuli, 10 to the right and 10 to the left ear. Responses were given by pointing with the forefinger to the ear stimulated.

Visual neglect

Two tests were given:

(1) *Reading test:* The patient was presented with a cardboard sheet, 41 cm × 29·5 cm, where the sentence *"La pera é caduta sotto l'albero"* ("The pear has fallen under the tree"), was written in 15 mm high Letraset letters on a single line, 39 cm long. The patient was requested to read it aloud. Aphasics were encouraged to attempt reading, not worrying about the errors they might make. If reading proved to be impossible, they were requested to point to each word of the sentence.

Table 1 *Clinical characteristics of brain-damaged patients*

	Aetiology		*Length of illness**				
	Vascular	*Neoplastic*	*< 30 days*	*> 30 days*	*Visual neglect*	*Aphasia*	*Parietal† involvement*
RBD patients (No: 30)	22	8	16	6	16	—	15
LBD patients (No: 15)	11	4	6	3	—	7	8

*Vascular patients only.
†Based on CT scan evidence.

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(2) *Circle test:* A white cardboard sheet, 70 × 50 cm, displaying 10 red circles, 15 mm in diameter, symmetrically arranged, five in the left half and five in the right half, was presented for the patient to point to the circles with the forefinger ipsilateral to lesion. The test was repeated with the same cardboard sheet presented upside-down, so that the total number of circles to point to was 20.

Results

Number of omissions

Control patients never failed to report interruptions. One LBD patient omitted one ipsilateral signal in the binaural test. Seven RBD patients made errors of omissions in the mono-aural test and nine patients did the same in the binaural test (table 2). All patients who omitted on the former did the same on the latter, markedly increasing the number of their omissions.

Neglect was always present in the contralateral ear. It also affected the ipsilateral ear of four patients in the mono-aural test and five patients in the binaural test, but contralateral omissions outnumbered ipsilateral omissions in every case, with a mean difference of 2·22 in the mono-aural test and 10·11 in the binaural test. The scores of the nine patients who omitted on either test were submitted to a two-way within-subjects analysis of variance, with ear (right and left) and test (mono-aural and binaural) as factors. The two main effects and their interaction were significant: omissions prevailed in the left ear ($F: 19{\cdot}15$, $p < 0{\cdot}005$ with 1·8 DF), in the binaural test ($F: 19{\cdot}27$, $p < 0{\cdot}005$ with 1·8 DF) and the ear difference was significantly more marked in the binaural than mono-aural test ($F: 11{\cdot}01$, $p < 0{\cdot}02$ with 1·8 DF).

The prevalence of omissions in the contralateral (left) ear was not significant when the analysis was confined to the mono-aural test. Table 2 shows that the ear difference was of approximately the same size in the five patients (No 3, 4, 16, 22, 23) with exclusively right-sided damage and in the four patients (No 12, 15, 28, 30) who also had evidence of a left, clinically silent lesion.

Relation of auditory neglect to locus of lesion and other manifestations of hemi-inattention

Table 3 summarises clinical and CT findings of the patients with auditory neglect. Eight of them had recent vascular lesions of the right hemisphere that in two cases (No 12 and 15) were coupled with a lacuna and an old infarct, respectively, of the left hemisphere. None of these left brain lesions had been manifest in the patients' history.

Patient No 28 had a right parietal metastasis and two smaller metastases in the parieto-occipital and occipital regions of the left hemisphere, which were clinically silent. The locus of the right sided lesion was parietal in five patients, thalamic in three and occipital + thalamic in one. Among RBD patients with neglect auditory extinction was severe in four patients (with 9 to 10 contralateral stimuli out of 10 missed); mild in three patients (with 2 to 4 stimuli extinguished) and absent in two patients (No 12 and 28). Auditory extinction was also present in seven RBD patients without neglect.

Visual neglect was found in 16 RBD patients, nine without and seven with auditory neglect. In the latter group it was severe in six patients (only the last word of the sentence was read and all circles lying to the left of the midline were neglected) and mild in one (only the first two words of the sentence were omitted). Two patients with auditory neglect (the same who did not extinguish on auditory simultaneous stimulation) did not show visual neglect.

Among LBD patients, one showed massive auditory extinction and none visual neglect.

Follow-up

The eight patients with vascular lesions with neglect were re-tested approximately 2 weeks after the first examination and five of them still showed neglect (table 2). In four of these patients a third testing session was carried out approximately a month later and it showed that there were still three patients who missed more than half of contralateral interruptions 2 to 3 months after the stroke.

Table 2 *Number of omissions made by RBD patients with auditory neglect*

	1st Examination				*2nd Examination*				*3rd Examination*			
	Mono-aural		*Binaural*		*Mono-aural*		*Binaural*		*Mono-aural*		*Binaural*	
Pt. No.	*LE*	*RE*	*LE*	*RE*	*LE*	*RE*	*LE*	*RE*	*LE*	*RE*	*LE*	*RE*
3	3	2	7	4	0	0	1	1	—	—	—	—
4	2	0	10	2	0	0	0	0	—	—	—	—
12	3	0	5	3	0	0	1	1	—	—	—	—
15	10	3	14	3	3	0	11	3	2	0	13	0
16	0	0	7	0	0	0	3	0	0	0	2	1
22	2	0	8	0	0	0	2	0	—	—	—	—
23	7	3	23	9	7	6	15	9	6	2	20	2
28	2	1	15	0	—	—	—	—	—	—	—	—
30	0	0	23	0	0	0	24	0	0	0	24	0

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Table 3 *Clinical and CT findings in RBD patients with auditory neglect*

Pt. No.	*Aetiology*	*V. Ext.*	*T.Ext*	*A. Ext*	*. Visual neglect*	*Locus of lesion*
3	V	nt	nt	10/10	severe	R. inferior parietal infarct
4	V	10/10	nt	10/10	mild	R. thalamic-capsular haematoma
12	V	0/10	0/10	0/10	absent	R. inferior parietal + L. inferior parietal (old) infarct
15	V	nt	nt	4/10	severe	R. parietal haematoma + L. fronto-parietal (old) infarct
16	V	10/10	10/10	2/10	severe	R. inferior parietal infarct
22	V	nt	4/10	4/10	severe	R. thalamic-capsular infarct
23	V	nt	nt	9/10	severe	R. thalamic-capsular haematoma
28	N	10/10	10/10	0/10	absent	R. parietal + L. parieto-occipital + L. occipital metastasis
30	V	nt	nt	10/10	severe	R. occipital and thalamic infarct

nt = not testable, because of severe somato- sensory or visual field defect.
V. Ext.: visual extinction; T. Ext.: tactile extinction; A. Ext.: auditory extinction.

Discussion

The outcome of the present study provides clear evidence that in RBD patients contralateral neglect (that is, failure to detect a stimulus coming from the left ear) extends to the auditory modality, provided care is taken to confine the signal to one ear. Although the phenomenon is already apparent in an attenuated form when the patient focuses on the left ear, its intensity is greatly enhanced in conditions of diffuse attention, when the side of interruption is not known in advance and the patient has to attend to the sound of both ears. In this condition two RBD patients (No 23 and 30) missed practically all left ear stimuli and two others (No 5 and 28) more than half. In three of them (No 15, 23 and 30) omissions were still very marked 2 to 3 months after the stroke. The length of the testing session and the relatively rare occurrence of interruptions have probably been instrumental in favouring lapses in sustained attention, but the prevalence of left ear omissions points to a specific, lateralised deficit, which in the present series was associated with damage to the right parietal lobe or right thalamus, two structures known to play a pivotal role in the circuit of lateralised attention.

It is noteworthy, however, that auditory neglect was consistently associated neither with auditory extinction, nor with visual neglect. The finding of two patients who had neglect, but not extinction disputes the widely held view that extinction is but a minor form of neglect. Taken in conjunction with comparable data found in the visual modality,[5] and with the three patients of the RBD group without neglect who extinguished the majority of left ear stimuli, this dissociation indicates that extinction does not necessarily lie on a continuum of attentional disorders, having neglect at its end, but may result from discrete mechanisms and that any generalisation from extinction to neglect must be guarded.

The possibility first provided by the present experiment to demonstrate the existence of auditory neglect allowed us to compare its occurrence with that of visual neglect and to gain information on the organisation of the neural apparatus deploying attention to contralateral space. Though neglect has been investigated mainly in the visual modality, current theory[7 8] tends to conceive of it as a multimodal phenomenon, which affects the detection of contralateral stimuli, whatever the sensory channel through which they are conveyed. This may be so because a lesion of the posterior parietal cortex, which is the most frequent anatomical correlate of neglect in humans[9 10] tends to block the multiple unimodal inputs converging on it. Alternatively, it might be held that this area is equipped to provide the neural template for a superordinate surveyor of novel and significant events occurring in contralateral space, the damage of which results in failure to attend to any type of stimuli located therein. Such a hypothesis would imply that neglect must necessarily involve all sensory inputs independently of the modality through which they are transmitted, an assumption at variance with the outcome of the present study, which found nine RBD patients with visual but not auditory neglect and two RBD patients with auditory but not visual neglect. These instances of dissociation are more compatible with a model envisaging a plurality of autonomous attentional control systems decentralised at the level of the single modalities, as argued by Bisiach *et al* [11] with reference to anosognosia, and liable to produce unimodal neglect.

So far we have focused on neglect affecting the contralateral ear, but a failure to detect interruptions was also apparent in the ipsilateral ear of five RBD patients, though never with the same severity as in the contralateral ear. This finding cannot be disregarded as an unspecific consequence of brain damage, since it never occurred in LBD patients or in the RBD patients without contralateral auditory neglect, even those who showed visual neglect. In two of the five patients there was CT evidence of a previous clinically silent infarct of the left hemisphere. Although a recent and clinically manifest left brain lesion never produces contralateral neglect when occurring in isolation, the question may

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be raised of whether an old lesion may play an additive effect when occurring in combination with a recent right-sided damage. On this account one would expect ipsilateral omissions to be more frequent following bilateral than unilateral (right) brain-damage, but table 2 shows that this was not the case. It is, therefore, legitimate to assume that in these patients, not differing from patients with disease confined to the right hemisphere, right neglect depended on right brain damage and reflected the unique property of this hemisphere to also exert attentional control on the ipsilateral space. This hypothesis was advanced[7 8] to account for the much more frequent occurrence of neglect after right than left brain damage and found some support in normal studies, where EEG[12] and regional cerebral glucose metabolism data[13] suggested a right hemisphere participation in the processing of stimuli presented to the right space. There is also some pathological evidence pointing in the same direction. RBD patients have been found by Weintraub and Mesulam[14] to manifest, in addition to the classical left neglect syndrome, signs of inattention for the right space, where they missed some targets in a visual cancellation test. The interpretation of this finding is confounded by the fact that rightwards scanning unavoidably brings part of right-sided stimuli in the patient's left visual field. However, it receives some support by a study[15] showing that when single stimuli were flashed in different positions of the right visual field, those located to the left were responded to more slowly by right parietal patients. Weintraub and Mesulam's RBD patients were also found to be mildly, but significantly slower than controls on a tactile searching task when the target was in the right space, while no impairment in the left space was shown by LBD patients. This finding hints at a delayed attention in the tactical exploration of the ipsilateral space, but does not demonstrate a true neglect, comparable to that found in the contralateral space.[16] The right ear omissions, found in the present study, provide definite evidence for a substantial right hemisphere contribution to the monitoring of right-located events, because the absence of exploratory movements in the auditory task avoids the discrepancy between body-centred space and eye-centred space entailed by visual tasks. What is surprising is that while left brain damage is apparently compensated by a well-functioning right hemisphere, the opposite is not always true. Is this asymmetry to be taken as evidence that right space surveillance is exerted more powerfully by the ipsilateral right hemisphere than the contralateral left hemisphere? More data are needed, particularly on the behaviour of LBD patients, before a conclusion can be reached. The auditory attention test appears to provide a good paradigm for pursuing this line of research.

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