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Auditory Agnosia

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Auditory agnosia

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Auditory agnosia can be defined as the defective recognition of non-verbal sounds and noises. The clinical picture of this disorder is described and the scarcity of knowledge of auditory agnosia derived purely from single cases is discussed. Next, experimental studies on unselected series of brain-damaged patients, especially designed to clarify the relation of auditory agnosia to aphasia and to the hemispheric locus of the lesion are reported. The results consistently point to the existence of two types of auditory agnosia, a semantic–associative one, specifically associated with lesions of the left hemisphere and aphasia, and a discriminative one, specifically associated with lesions of the right hemisphere. The hypothesis is advanced that the semantic–associative variety of auditory agnosia is part of a wider cognitive disorder.

The term 'auditory agnosia' came into use in the neurological literature after Freud's monograph of 1891, to define the impaired recognition of non-verbal sounds and noises.

I shall briefly give a concrete idea of the clinical characteristics of patients who show this disorder, as it presents itself to the clinician, and then go on to report the results of some experimental studies that we have undertaken to try to clarify the matter.

Evidence from clinical cases

Agnosia for non-verbal sounds and noises hardly ever comes about alone (there is one single case in which it did) but rather it is accompanied by other, much more striking symptoms, which puzzle and distress the patient and attract the clinician's interest. The typical patient is a person over the age of 50 who suddenly becomes unable to understand spoken language, to repeat spoken words and to write from dictation. In contrast, he still can speak, read and write spontaneously quite well. These patients show such a severe impairment of all performances involving auditory language input that they tend to be mistaken for deaf persons (unlike most Wernicke's aphasics, who are sometimes mistaken for confused patients). However, they are not at all deaf, at least in the sense that pure-tone audiometry is virtually normal. This condition, which is conspicuous and rather rare, has been defined as 'pure *word* deafness' (Lichtheim 1885) precisely to stress the fact that the defect was strictly limited to the perception of spoken language. These patients complained first and foremost that they could not perceive what people said to them, and much more rarely that they could not perceive ordinary everyday noises and sounds. This explains why clinicians often neglected to investigate auditory agnosia at all, and, if they did, it was merely to verify that it was *not* there, and that the patient's imperception was indeed confined to speech sounds.

No wonder, then, that the procedures and criteria for assessing it were variable and often inadequate. Some clinicians satisfied themselves with the observation that the patient reacted spontaneously to environmental noises, while others looked for more convincing evidence of

preserved recognition through non-systematized clinical tests in which the patient was asked to identify different kinds of auditory stimulus. These were of course restricted to sounds or noises that could be easily reproduced in the patient's environment. Only after the 1950s did the use of tape recordings allow a wider selection of sounds. The most commonly used auditory stimuli were common noises (such as coughing, whistling, clapping hands, knocking on the door, turning a key in the lock, crackling a sheet of paper, rattling a matchbox, lighting a match, jingling a bunch of keys, running water), sounds of musical instruments (e.g., toy trumpet, a toy drum, a bell, sometimes a piano) or noises of animals (such as the barking of a dog, the crowing of a cock). Before the introduction of the tape recorder these sounds had to be imitated on the spot by the examiner, who sometimes used onomatopoeic words.

All these factors probably contributed to the scarcity of studies devoted to analysing this symptom. While its absence in cases of pure word-deafness was stressed by clinical investigators because it emphasized the purity of the language disorder, the possible implications of its presence were seldom discussed.

An interesting suggestion can be found, however, in a purely theoretical paper by Kleist (1928). This author emphasized that the inability to perceive isolated sounds or noises ('perceptive Geräuschtaubheit') or to grasp sequences of them ('Geräuschfolgetaubheit') should be distinguished from the inability to understand the 'meaning' of noises ('Geräuschsinntaubheit'), where the patient is unable to associate the well-perceived sound with what it stands for (e.g. barking with dog). The first two types of disorder may be considered to be perceptual or discriminative in nature, since they both reflect an impairment in discriminating the acoustic structure of the stimulus, while the third type may be defined as associative or semantic, because it consists of the inability to associate the acoustic pattern with its meaning.

If one uses this dichotomy to categorize the published case-reports to date, one finds that, whenever the records were sufficiently detailed to permit a qualitative analysis of the auditory recognition disorders, they appeared to be perceptual–discriminative rather than semantic–associative in nature. The trouble seemed to consist essentially of a difficulty in grasping the acoustic structure of the auditory stimuli. Sometimes several different noises (keys, bell, drum, etc.) were consistently defined by a vague, stereotyped term, as if they awoke only an acoustically undifferentiated impression, e.g. as 'rattles' or 'squeaks', 'something like a grasshopper's sound'. Whenever errors were more specific, they were acoustic, not semantic, in nature: the noise of an elevator in motion was mistaken for the vibration of a tuning fork, the cry of a cat for someone singing, the tinkling of a glass for the jingling of keys. Some features of the defect, such as the selective impairment of high-pitched or low-pitched noises or of more musical as opposed to less musical noises also afford a perceptual–discriminative explanation rather than an associative one (for a more detailed analysis of symptoms see Vignolo (1969)).

Indeed, in the published cases of auditory agnosia associated with word-deafness, the perceptual component was so evident that some authors advanced the hypothesis that these symptoms may be due simply to partial deafness of either peripheral or central origin. If by partial deafness one means (as they did) a loss of threshold acuity for certain tones of the scale, this view is no longer tenable, since accurate examinations with pure-tone audiometry have established beyond doubt that auditory agnosia and word-deafness may exist in patients without significant hearing loss. There are several other clinical features of these patients that certainly cannot be attibuted to defective sound discrimination. A remarkable symptom is *auditory inattention*, which may be so severe as to abolish the normal motor reaction to unexpected

noises, as in Liepmann's (1898) patient, who ran the risk of being run over by street-cars because he paid no attention to the sound of the horn. Such a complete absence of motor reaction to a sudden, very loud noise – so abrupt and loud as to elicit a kind of jump from other people in the room – is perhaps what really causes the impression that the patient must be deaf, and it is surprising to hear him say, after a few minutes, in a calm and matter-of-fact voice, that he has in fact perceived the noise. Hemphill & Stengel (1940) rightly observed that 'what is lacking . . . is the reflex turning towards the person speaking or the origin of the noise', but it is not known whether this is due to an auditory–motor disconnection (Kleist 1928) or to loss of cortical inhibition and selection of auditory inputs (as maintained by Ombrédane (1944)).

The prognosis of these patients is poor; the clinical picture, far from improving, is often aggravated by auditory hallucinations and by depression with paranoid ideas. Finally, the aetiology of this condition is mostly vascular, and the clinical history usually points to a bilateral lesion: the disorder has a sudden onset, following an infarct either in the right or in the left temporal lobe, in patients who had been struck in the past by another infarct in the opposite temporal lobe.

A review of the published autopsy findings disclosed that most of these patients indeed had a bilateral damage involving the posterior third of the temporal lobe. Strictly unilateral lesions were found in three cases: they damaged the left hemisphere in two cases and the right hemisphere in one.

Experimental studies

The published records of individual cases (29 patients according to a recent review by Ulrich (1977)) offer but a very approximate picture of the relations between non-verbal auditory recognition and the brain. This is due to three main causes: biased sampling (virtually only patients with clinically conspicuous word-deafness were studied, and these are rare patients, who certainly cannot be considered representative of the entire brain-damaged population), heterogeneous testing conditions, and incomplete anatomical data. In particular, the single cases tell us little about the internal structure of auditory agnosia as well as about its relations with aphasia and the hemispheric side of the lesion. We then thought that, in order to disentangle auditory agnosia from other high-level auditory or speech defects, we should change the strategy of research and try to examine non-verbal auditory recognition by means of quantitative tests in unselected samples of patients with lesions confined to one hemisphere.

A pilot study (Spinnler & Vignolo 1966) convinced us that the division of auditory agnosia into a perceptual–discriminative and an associative–semantic aspect, implied by Kleist's (1928) analysis, could be a promising working hypothesis for research. We therefore undertook two studies, in which the following *ad hoc* tests were used.

Testing procedure

The auditory recognition tests consisted of tape recorded non-verbal sounds or noises that were presented through a loudspeaker located about 75 cm in front of the patient. Testing sessions took place in a quiet, though not sound-proofed, room. Of the two tasks, one (the Meaningless Sounds Discrimination Test) was intended to test the ability to discriminate the exact *acoustic pattern* of sounds, while the other (the Meaningful Sounds Identification Test) was intended to test the ability to identify the exact *meaning* of sounds.

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Meaningless Sounds Discrimination Test

The patient was told that he was going to hear two noises, one after the other. He was asked to listen carefully and say whether they were the same or different. He was then given six items (pairs of noises) as examples. Patients who failed on more than two of them were not given the test. The test proper consisted of twenty items. The paired noises were identical in five items, quite different in five items and very similar in ten items. These three types of items were intermingled in the test series. Each test noise was produced by mixing two or more recorded noises taken from sound tracks for radio programmes (e.g. a crackling sound on a buzzing background) and recording the mixed signals on magnetic tape. No attempt was made to analyse acoustically the complex patterns resulting from these manipulations. Patterns varied widely in structure; changes in loudness and pitch were sometimes gradual, sometimes abrupt. They lasted from 6 to 10 s, the duration being held constant for the members of each pair. The interval between the first and second noise of any pair was 2 s. Correct responses given within 10 s were rated 1 point, wrong or delayed (over 10 s) responses were rated 0 points.

Meaningful Sounds Identification Test

This consisted of twenty meaningful sounds or noises, each of which was presented to the patient with a four-picture display. The patient was asked to listen to the noise (e.g. the braying of a donkey), and then indicate which of the four pictures shown to him corresponded to the natural source of the noise he had just heard (figure 1).

Of the four objects represented in the display, one was the natural source of the sound (donkey), two belonged to the same semantic category of the real source but which produced either an acoustically different noise (a bellowing cow, a trumpeting elephant) or, in other items, no particular noise, and one was entirely unrelated to the presented noise (an ambulance in action). Pointing to the correct picture was given one point, yielding a maximum score of 20. Before beginning the test the examiner ascertained that the patient could visually recognize all the pictures correctly, by asking him to name them or, if he was aphasic, by pointing to each picture and naming it for him. The patient was then given two examples (siren wailing, sheep bleating), each of which was repeated twice. Patients who failed on the examples (on second presentation) were not given the tests.

Study 1

In the first study (Faglioni *et al.* 1969) we confined ourselves to unilateral lesions: in addition to 49 control patients without cerebral lesions, we examined 41 patients with lesions confined to the right hemisphere and 60 with lesions confined to the left hemisphere. Diagnosis of the affected hemisphere relied upon clinical symptoms, supplemented by e.e.g. and by available arteriography and isotope brain-scan findings. Hemisphere-damaged patients were excluded from the study who were left-handed or ambidextrous, had a clinical picture or past history pointing to involvement of both cerebral hemispheres, or could not be given the tests because of physical or mental disability (e.g. confined to bed or impaired consciousness). Right and left hemisphere-damaged patients were not significantly different with respect to age, educational level, aetiology and length of illness. Visual field defects, aphasia and simple visual reaction times, considered as a measure of attention, were systematically assessed.

All patients perceived the ticking of a stop-watch held at about 2 cm from either ear; none of them had been clinically deaf before the onset of the illness, nor complained of hypoacusis

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at the time of testing. Since the hemispheric groups were balanced with respect to age, there is no reason to believe that physiological hearing loss affected one group more than the other.

Results

The mean test scores (corrected for age and educational level) of the control, right and left hemisphere-damaged groups are shown in table 1. A bivariate analysis of covariance was carried out and the significantly defective mean scores are printed in italic type.

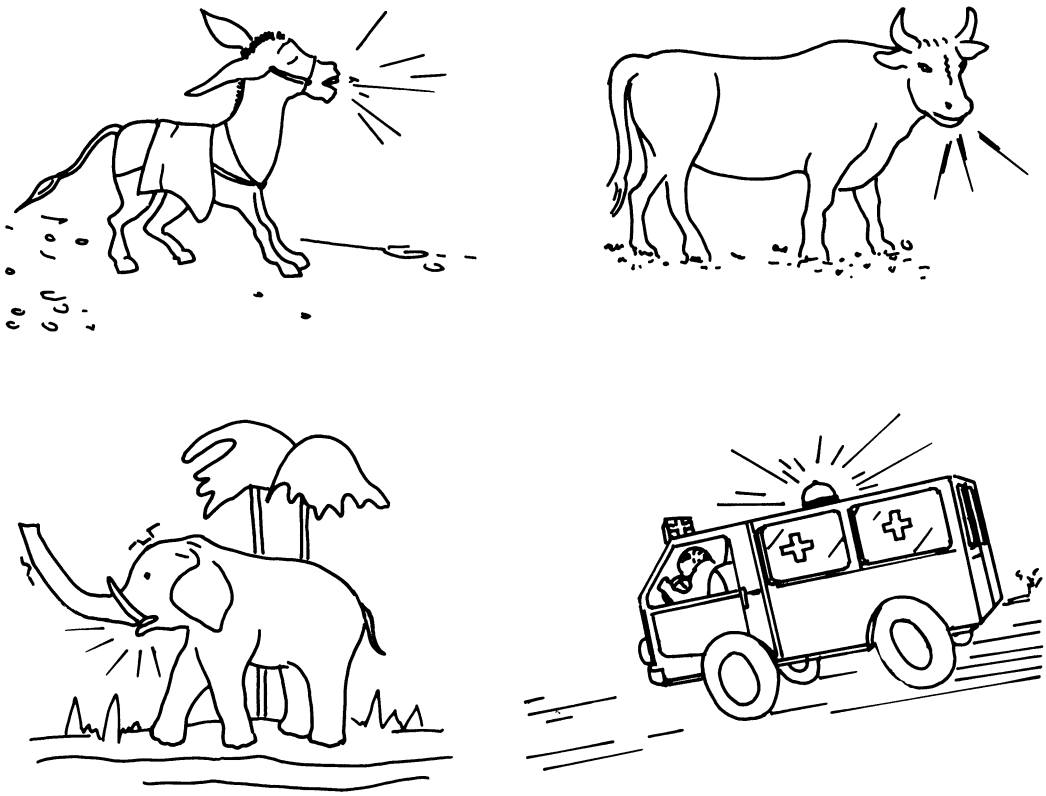

FIGURE 1. Example of a four-picture display of the Meaningful Sounds Identification Test.

It is apparent that failure on each test was specifically associated with damage to a different hemisphere. On the Meaningless Sounds Test, the left hemisphere-damaged group did as well as the controls, while the performance of the right group was significantly inferior to that of the other two groups. The reverse occurred with the Meaningful Sounds Test, on which the right brain-damaged group obtained the same mean score as the controls, while the left brain-damaged patients performed significantly worse than the other two groups. In other words, it was statistically established that there is a 'double dissociation' between failure on the Meaningless and the Meaningful Sounds Tests with respect to the hemispheric side of the lesion. Another analysis proved that such double dissociation was not due to chance.

As clinicians, we were less interested in the general means of the groups than in the so-called 'deviant' patients, i.e. those who could be defined as having a relevant degree of auditory agnosia. Therefore, we tried to study rather more closely the individual patients who had a selective defect on any one particular auditory test. These patients were singled out of the entire brain-damaged sample by using the following criteria. A given score on each test was defined

as *defective* if it was lower than the mean (M) minus two standard deviations (s.d.) of the control scores on that test. Likewise, the discrepancy between the two test scores in any given patient was defined as abnormal if it exceeded ($M \pm 2$ s.d.) of the inter-test differences in the control group. A patient was considered to have a selective impairment on one test if (1) his score was defective on that test and not on the other and (2) he showed abnormal discrepancy between the two test scores. By these rather stringent criteria, eight patients were found to be selectively impaired on the Meaningless Sounds Test and all of them had a lesion in the right hemisphere; twenty-two were selectively impaired on the Meaningful Sounds Test, and all of them had a lesion in the left hemisphere and aphasia. The two groups were not significantly different with respect to age, educational level, reaction time scores and aetiology; visual field defects tended to be more frequent in the left group.

TABLE 1

(Mean scores (corrected).)

		brain-damaged patients	
	controls (49)	right (41)	left (60)
Meaningless Sounds Discrimination Test	16.0	*12.9*	16.0
Meaningful Sounds Identification Test	17.5	17.5	*14.8*

Results in italic type indicate means that are significantly different from the highest mean in the same row.

Study 2

The purpose of this study was to verify the dissociation disclosed by the previous research on another series of patients, whose brain lesions had been assessed for site and extent by means of computed tomography (c.t. scan). This non-invasive neuroradiological procedure provides the most accurate routine method available today for localizing a brain lesion *in vivo*. In particular, it enables us to visualize old vascular lesions in clinically silent areas of the brain. We can ascertain whether lesions are confined to one hemisphere or involve both hemispheres, while we can determine with varying degrees of approximation the locus of the lesion within one hemisphere.

In this study (Guidotti, Papagno & Vignolo, in preparation), only patients whose c.t. scans showed circumscribed ischaemic lesions of the hemispheres were examined. In addition to patients with unilateral (14 right, 20 left) damage, all patients with bilateral lesions (5) on the c.t. scan were also included. None of these patients complained of hypoacusis; in particular, patients with bilateral lesions did not show any clinical auditory disorder of cerebral origin, such as word-deafness. There was no significant difference between groups with respect to the usual variables, except for the bilateral patients, who were older than the unilateral ones.

Results

The mean scores (not corrected) obtained by the three brain-damaged groups on the sound recognition tests are shown in table 2. Here, too, the Meaningless Sounds Test was performed significantly worse by the right brain-damaged patients than by the left brain-damaged patients, while the reverse occurred with the Meaningful Sounds Test. Moreover, the patients with bilateral lesions did poorly on both tests. When patients with a defect on either or both tests were singled out with the usual criteria, again, those with a selective discrimination deficit

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had lesions confined to the right hemisphere, whereas those with a selectively semantic deficit had lesions confined to the left hemisphere and were aphasics; in addition, patients with deficits on both tests had bilateral lesions (table 3).

In conclusion, then, this second piece of research, in which lesions were localized with more accurate techniques, essentially confirmed the findings of the first study.

TABLE 2

(Mean scores (uncorrected).)

	brain-damaged patients		
	right (14)	left (20)	bilateral (5)
Meaningless Sounds Discrimination Test	*12.5*	15.6	*10.0*
Meaningful Sounds Identification Test	18.4	*14.9*	*12.6*

Results in italic type indicate means that are significantly different from the highest mean in the same row.

TABLE 3. PATIENTS WITH SELECTIVE DEFECTS

	right	left	bilateral
Meaningless Sounds Discrimination Test	3	–	–
Meaningful Sounds Identification Test	–	4	–
both	–	–	3

DISCUSSION

I mentioned in the introduction that auditory agnosia *per se*, as defined here, is not a conspicuous clinical symptom and it may easily escape the neurologist's notice. In spite of the fact that the tests we have used were rather rudimentary, the neuropsychological method must be credited with the merit of bringing out consistent hemispheric asymmetries of auditory recognition that are difficult to detect at the clinical level.

The reasons for this double dissociation and the nature of the defect underlying failure on either test are open to discussion. The poor performance of the right hemispheric group on the Meaningless Sounds Test appears to be due to a specific inability to carry out auditory perceptual discriminations, rather than to a non-specific impairment of attention, because covarying scores for reaction time did not change the results. This conclusion is in agreement with the findings of previous investigations, carried out both in brain-damaged patients (Milner 1962; Shankweiler 1966*a*, *b*) and in normal subjects (Kimura 1964), indicating that the anatomical structures that subserve the recognition of non-verbal and perceptually complex auditory patterns belong to the right hemisphere.

On the other hand, the finding of an auditory recognition defect confined to the Meaningful Sounds Test provides empirical evidence of Kleist's Geräuschsinntaubheit ('deafness to the meaning of noises'), with the further qualification that this disorder is specifically associated with those lesions of the left hemisphere that also produce aphasia. Not only was the impairment on the Meaningful Sounds Test confined to the aphasic group, but all the patients showing an isolated defect on this test were aphasic. A close association was found between impairment on the semantic test and the defect of auditory verbal comprehension. In the first study, patients with global and severe Wernicke's aphasia, whose auditory verbal comprehension is poor by definition, did significantly worse than the rest of the aphasic group and constituted more than 80% of patients with selective semantic impairment. Moreover, a remarkable correlation of

0.65 was found between the Meaningful Sounds scores and scores on the Token Test – a test of auditory verbal comprehension of sentences.

Compared to these findings, it is surprising that aphasics performed well on the Meaningless Sounds Test and that no relation whatsoever could be found between auditory discrimination and language comprehension. Patients with global and severe Wernicke's aphasia performed the test as well as the remaining aphasic and non-aphasic left hemisphere-damaged patients and the correlation with the Token Test was very low and not significant ($\rho = 0.21$). One may add that the specific impairment of left brain-damaged patients with aphasia on a sound–object matching test has been confirmed by other studies (see, for example, Doehring *et al.* 1967; Strohner *et al.* 1978).

All the above evidence suggests that one of the basic disorders underlying the poor recognition of meaningful auditory material (verbal as well as non-verbal) is not so much a perceptual deficit but it consists, rather, of the inability to *associate* the auditory percept to a definite source or event. A number of investigations suggest the possibility that such a defect may transcend the auditory modality and be cognitive rather than sensory in nature. In a series of studies performed in our laboratory, aphasics have been found to be specifically impaired on several non-verbal matching tasks, such as associating a picture with the corresponding object (De Renzi *et al.* 1969), colour with picture (De Renzi & Spinnler 1967), gesture with object (De Renzi *et al.* 1968), as well as on a classic test of conceptual thinking, such as Weigl's Sorting Test (De Renzi *et al.* 1966). Moreover, when we studied the intercorrelations existing among three associative tasks (sound–picture, object–picture, colour–object) in a group of 99 patients (42 with right and 57 with left lesions) who had been examined with all three tasks, we found that, while the correlations within the right hemispheric group were trifling and not significant, those within the left group were highly significant (Vignolo 1972). These findings tend to support the view that impairment on such apparently different tasks may be due to one basic cognitive–associative disorder, which could be defined as the inability to put together different aspects of the same concept.

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