

NOTE

PSYCHOACOUSTICAL DEFICITS RELATED TO BILATERAL SUBCORTICAL HEMORRHAGES A CASE WITH APPERCEPTIVE AUDITORY AGNOSIA

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ABSTRACT

We report a case of acute deafness secondary to bilateral hemorrhages involving the external capsule and extending to both temporal isthmi. The lesions probably disrupted both auditory radiations. Deafness disappeared within 2 weeks leading to a transient auditory agnosia for environmental and verbal sounds. Performance on audiological and neurolinguistic tests were consistent with the hypothesis of a deficit of non-specific auditory processes. Psycho-acoustical deficits related to subcortical lesions are very rare and might differ from those due to cortical lesions by the lack of aphasia. The present case and both previous cases with subcortical lesions might suffer from auditory agnosia of apperceptive type. Its characteristics and the role of non-specific auditory processes are discussed using the data obtained from this third case.

INTRODUCTION

Cortical deafness and auditory agnosia are usually related to cortical lesion of the temporal lobe and are frequently associated with aphasia (Lhermitte, Chain, Escourolle et al., 1971). The underlying cognitive disorders accounting for auditory agnosia have mainly been studied in patients with word deafness, and remain controversial (Praagstra, Haggort, Maassen et al., 1991). Thus, cases of auditory agnosia with pure subcortical lesions and without aphasia might be of major interest to clarify its underlying mechanisms. To our knowledge, only 2 cases of acute deafness (Clark and Russel, 1938; Hirano, 1973 and 1983) and 2 cases of auditory agnosia (Motomura, Yamadori, Mori et al., 1986; Kazui, Naritomi, Sawada et al., 1990) with audiological assessment related to pure subcortical lesions have been reported. New cases of auditory agnosia with pure deficits might contribute to a better comprehension of the deficit underlying auditory agnosia.

We report a third case of acute deafness evolving to auditory agnosia and related to subcortical lesions.

CASE REPORT

The patient was a 58 year-old right-handed French-speaking woman with 9 years of education. She had a 2-year history of arterial hypertension, treated with oral nifedipine (60 mg a day), and a 1-year history of diabetes mellitus, treated with oral metformine (1,700 mg a day). She suddenly experienced a mild right-sided sensory loss. Twenty-four hours

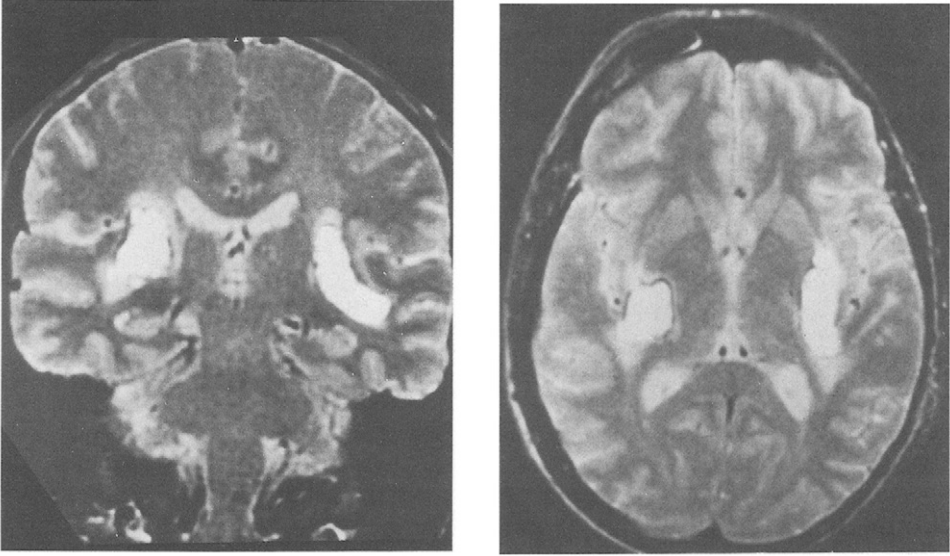


Fig. 1 – MRI: axial (a) and coronal (b) planes showing the bilateral hemorrhages in both external capsules extending to temporal isthmi.

later, a mild left-sided weakness occurred. In the ambulance, she suddenly felt that the word had become silent. She was able to understand speech only by carefully observing lips and she spontaneously asked speakers to write down their questions. She had never had previous hearing difficulties. The patient was attentive, her blood pressure was 180/90 mmHg and her heart rate was 80/min. Her neurological examination revealed left-sided sensory loss with a mild hemiparesis, associated with weakness and mild sensory loss of the right leg. Visual field campimetric examination was normal. There was no evidence of aphasia, gestural apraxia, visual hemineglect or anosognosia for the deafness.

A computed tomographic (CT) scan, performed within 2 hours after the onset of deafness, revealed 2 deep subcortical hemorrhages. Magnetic resonance imaging (MRI) performed 1 week post-onset showed signals of high intensity located in both external capsules, bilaterally extended to temporal isthmi; temporal cortices and thalami were spared (Figure 1).

A ^{15}O positron emission tomographic (PET) scan was performed 2 months after the onset using procedures previously reported (De Reuck, Van Aken, Decoo et al., 1991). Values of cerebral blood flow, oxygen extraction ratio and consumption were compared with those of controls and because of repeated measures, only p values lower than 0.001 were considered as significantly abnormal (De Reuck, Van Aken, Decoo et al., 1991) The decrease in oxygen consumption reached significance in the parietal and temporal cortices (Table I).

The complete deafness was confirmed by pure tone threshold audiometry using pulsed pure tone sounds. Otologic examination, tympanogram, aural reflex and vestibular function testing were normal. Brainstem (BAEPs) and Middle Latency (MAEPs) auditory evoked potentials were assessed using Nicolet Pathfinder II. Each trial utilized condensation and rarefaction clicks (80 and 20 dB) of 0.1 msec duration at 20 Hz (BAEPs) and 5.7 Hz (MAEPs). Stimuli were monaurally presented and contralateral masking was used for all trial. Three records, representing at least 2,000 responses, were obtained for each ear. Recording electrodes were derived from the vertex and referenced to the ear lobe of the stimulated side. Filters were set at 150-3,000 Hz (BAEPs) and 5-1,500 (MAEPs). All BAEPs components were elicited bilaterally at all click intensities (Figure 2) and waves I to VI were recorded with normal latencies and amplitudes (Table II), indicating integrity of the

TABLE I
 Values of Regional Cerebral Blood Flow (CBF), Oxygen Extraction Ratio (OER) and Oxygen Consumption (CMRO₂)

	Left side			Right side		
	rCBF ml/min/100g	rOER (%)	rCMRO ₂ ml/min/100g	rCBF ml/min/100g	rOER (%)	rCMRO ₂ ml/min/100g
Frontal cortex	61.96	26.72	2.34	58.97	31.34	2.11
Temporal cortex	36.16	34.56	2.05*	37.48	29.48	1.61*
Parietal cortex	32.47	27.80	1.61*	43.98	21.7	1.51*
Occipital cortex	47.65	31.95	2.43	58.30	33.75	2.95
Striatum	54.34	24.14	2.04	54.23	25.16	2.03
Thalamus	39.77	16.04	1.15	30.85	24.43	1.02

* = p < 0.001.

cochlear mechanism and auditory brainstem pathways. Conversely, MAEPs components were not obtained.

Deafness resolved within 2 weeks, leading to a mild bilateral sensori-neural hearing loss of -20 dB (mean of audiometric threshold for 500, 1,000 and 2,000 Hz frequencies (Albert, Sparks, Von Stockert et al., 1972)). Despite this good recovery, the patient still complained

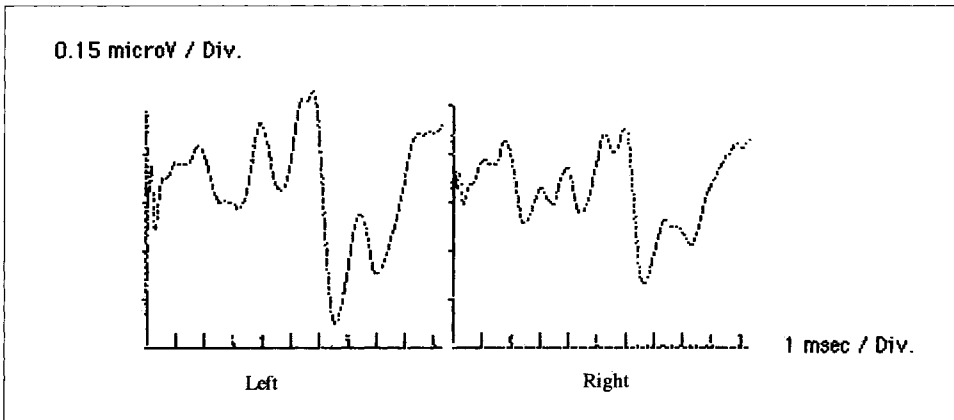


Fig. 2 - Brainstem Auditory Potentials. Recordings from left (Left) ear and right (Right) ear stimulations.

TABLE II
 Latency and Amplitude Ratios of Brainstem Auditory Evoked Potentials Components of the Patient (Left and Right Ears) and Controls

	Left ear	Right ear	Controls
Latencies			
I	1.85	1.75	<2.09
I-III	2.05	2.25	<2.6
V	5.87	5.93	<6.1
I-V	4.02	4.18	<4.36
Amplitudes ratios			
I/V	>1	>1	>0.5

TABLE III
Results of Neuropsychological and Language Tests

	Patient	Normal range
MMSE (Folstein et al., 1975)	30	≥28
WAIS (Wechsler, 1970)	VIQ: 77 PIQ: 94	
PM 38 (Raven, 1960)	40	percentile 50: 27
Spans (digit, "spatial")	4*, 6	6, 6
Peterson paradigm (Cermak, 1976)	total score 0 sec. score 3 sec. score 9 sec. score 18 sec. score	81±8.25% 50±0.00% 40.74±6.74% 38.10±7.00% 33.10±6.64%
Battery 144 (Signoret, 1991)	total score	86* 107±20
Gestural apraxia (Godefroy et al., 1994)	20	20
Cancellation test (Albert, 1973)	no omissions	no omissions
Rey's figure copying (Rey, 1941)	34	percentile 50: 32
Shortened Token Test (De Renzi and Faglioni, 1978)	35	>29
BDAE (Goodglass and Kaplan, 1972)		
Oral comprehension	119	114
Repetition	25	23.7
Naming	103	105
Fluency (fruits)	15	13.6±3.3

* = significant impairment.

of difficulties in identifying sounds. This impairment was not explained by the mild hearing loss and suggested the presence of auditory agnosia (Bauer and Rubens, 1979; Heffner and Heffner, 1989). In order to assess the presence of auditory agnosia, general neuropsychological, language, sounds identification and auditory evaluations were performed.

MATERIALS AND METHODS

Neuropsychological and language evaluations were performed 2 weeks after the acute stage and included tests assessing (1) general intellectual ability, (2) episodic and short term memory, (3) gestural ideomotor apraxia, (4) visuospatial hemineglect and (5) the French version of the Boston Diagnostic Aphasia Examination (Goodglass and Kaplan, 1972) and the shortened Token Test (De Renzi and Faglioni, 1978) (Table III).

The assessment of sounds recognition included tests evaluating identification of environmental noises, music and syllables. The environmental noise recognition test required the subject to name 48 sounds and to match them with drawings. The music recognition test required to name title, author or player and category of 33 well-known music tunes. The syllabic identification test required to repeat 34 consonant-vowels pairs, delivered binaurally with earphones, and to match them with one of the 34 written syllables. The patient's scores were compared with those of 7 healthy controls matched for age, education

level and music abilities and scores below cut-off scores (mean - 1.96 standard deviation) were considered as impaired.

The audiological assessment was performed 2 weeks post-onset and used 4 tests: speech audiometry, verbal dichotic listening test (Michel and Péronnet, 1975), loudness discrimination (Sisi-test, Jerger, Weikers, Sharbrough et al., 1969), and temporal auditory acuity tests. To assess temporal auditory acuity, we used a click fusion test: clicks were of 0.1 msec duration and separated by interval varying from 230 to 50 msec. This last test was repeated 2 month post-onset. Each ear was evaluated separately, except for the dichotic listening test.

Results

This patient used to listen to music, but was unable to play it (type I of Wertheim and Botez, 1959). Neuropsychological evaluations showed memory impairment and low IQ contrasting with better performance on Progressive Matrices (Table III). Digit span was impaired but when the assessment of verbal short term memory was carried out with written consonants (Peterson's paradigm), the performance was normal. Language evaluations were within normal range (Table III).

Scores on sound recognition did not differ according to the criteria used (matching or naming) and only the mean scores are reported in Table IV. Eighteen days after the stroke, sound recognition tests were impaired for environmental noise and syllable recognition but were in the normal range for music tune identification. These deficits resolved partly 2 months later.

The audiological assessment showed impairments of all tests (Table V). The patient reliably fused at intervals of 99 msec 2 weeks post-onset and at intervals of 5 msec 2 month later, whereas normals can distinguish 2 clicks 2 msec apart (Wallach, Newman, Rosenzweig, 1949). Impairment on speech audiometry was more prominent on the right ear and dichotic listening test showed complete right ear extinction.

TABLE IV
Percent Correct of the Patient (from day 8 to 60) and Controls (means ±SD) on Sound Recognition Test

		D 8	D 18	D 30	D 60	Controls
Pure tone audiometry threshold	dB SPL	-60	-20	-20	-10	—
Environmental noise identification	(%)	11*	48*	73*	85	88 (±3.1)
Music identification	(%)	21*	64	64	65	49 (±11)
Syllabic identification	(%)	6*	32*	63*	90	95 (±4.5)

* = scores below means - 1.96 S.D.

TABLE V
Audiological Assessment

	Patient		Controls	
	Left ear	Right ear	Left ear	Right ear
Pure tone audiometry threshold	-20	-20	—	—
Speech audiometry	50% at 40 dB 100% at 75 dB	50% at 70 dB max. 70%	—	—
Sisi-test ±2 dB	0%	0%	100%	100%
Click fusion test	99 msec.	99 msec.	2 msec.	2 msec.
Verbal dichotic listening test	62.5%	0%	≥70%	≥72.5%

Comment

The impaired recognition of environmental noises and syllables 18-day after the stroke was not explained by the mild hearing loss and fits the criteria of auditory agnosia (Bauer and Rubens, 1979; Heffner and Heffner, 1989). This auditory agnosia was associated with a deficit of auditory processes, and both the auditory agnosia and the deficit of temporal resolution resolved 2 months post-onset. To test the hypothesis that verbal auditory agnosia was related to the deficit of auditory processes, neurolinguistic testing was performed. As in visual agnosia, 2 main types of auditory agnosia have been distinguished: in the apperceptive type, auditory processes would be impaired leading to a deficit of sound discrimination, whereas in the associative type, access to specific processes (mainly phonetic) would be impaired leading to a deficit of sound identification (Spinnler and Vignolo, 1966; Buchtel and Stewart, 1982; Eustache, Lechevalier, Viader et al., 1990). A more precise characterization of apperceptive auditory agnosia rests on 2 features: the pattern of verbal sound discrimination deficits and, to a lesser extent, the benefit provided by access to a lexical representation. Unfortunately, both kinds of investigations have been rarely performed in the same patient, probably because of the frequent association of aphasia. In apperceptive agnosia, Auerbach, Allard, Naeser et al. (1982) showed that the pattern of defective verbal sound discrimination is consistent with an impairment of temporal auditory processing: the disorder involves sounds with rapid frequency changes, like consonants, whereas it spares vowels which are characterized by slow frequency changes. Second, the discrimination impairment between syllabic pairs mainly affects pairs differing by one feature, and amongst them, those differing by the place of articulation feature (Chocolle, Chedru, Bolte et al., 1975; Denes and Semenza, 1975; Auerbach et al., 1982; Miceli, 1982; Tanaka, Yamadori and Mori, 1987; Yaqub, Gascon, Al-Nosha et al., 1988; Eustache et al., 1990). Finally, the type of verbal stimuli used (syllables, words and sentences) would be critical, since the performance would benefit from the use of semantic or syntactic cues and access to a lexical representation (Saffran, Marin and Yeni-Komshian, 1976; Buchtel and Stewart, 1989; Eustache et al., 1990). Thus, increasing the constraints of the verbal stimuli e.g., by embedding single syllables in words, would improve their identification.

We suggest that the pattern of neurolinguistic impairment of apperceptive auditory agnosia is characterized by (1) a prominent impairment of the discrimination of syllabic pairs differing by 1 only feature and the place of articulation contrasts, (2) sparing of vowels identification and (3) improvement of identification with increasing constraints. We evaluated this hypothesis in the neurolinguistic study.

Neurolinguistic assessment. The syllabic discrimination test required discrimination of 108 pairs of syllables. All items were in consonant-vowel format and the vowels was always "a". Time interval between syllabic pairs was 3 sec (Albert and Bear, 1974). Forty percent used non-nasal stop consonants ("ba, da, ga, pa, ta, ka"). Thirty pairs were identical, 34 pairs differed by 1 feature (26% differed in regard to the manner of articulation, 21% by the voicing feature, 53% in regard to the place of articulation), 31 pairs differed by 2 features and 11 differed by 3 features. The decision task was binary: identical or different. Vowel repetition required to repeat 5 isolated vowels ("a, e, i, o, u") which were presented five times in pseudorandomized order. The performance on vowels identification in syllables was evaluated with the previous test of syllabic identification. Ten meaningful syllables from the previous test of syllabic identification were embedded in 30 tri-syllabic words, in initial, middle and final position (e.g. "ra" ("smooth"): *ratelier* ("rack"), *parapluie* ("umbrella"), *choléra* ("cholera")). This 30 words had to be repeated and scores were compared with those of the repetition of the 10 isolated syllables. For all these tests, sounds were natural stimuli delivered binaurally with earphones. All results are given in percentage correct.

Results

Syllabic pair discrimination was impaired (Table VI), especially when 2 syllables differed by 1 distinctive feature: errors were more prominent on pairs differing by the place of articulation feature whereas the discrimination of voicing feature was spared. The patient repeated all the isolated vowels correctly (Table VI), whereas identification of vowels in

TABLE VI
 Percent Correct on Neurolinguistic Tests of the Patient and Controls (mean scores \pm S.D.)

		D 8	D 18	D 30	D 60	Controls
Vowel repetition (Single)		100	100	100	100	100 (\pm 0)
Vowel repetition (Syllables)		23.5*	50*	100	100	100 (\pm 0)
Syllables repetition		0*	40*	70*	100	90 (\pm 4)
Word repetition		87*	93*	97*	97*	99 (\pm 0.7)
Syllabic discrimination	Total	74*	89	98	95	95.3 (\pm 3.3)
number of	0	78*	91	100	97	95.3 (\pm 6)
differing	1	69*	79	94	99	91 (\pm 6.5)
feature	2	84*	94*	100	100	99.5 (\pm 1.2)
	1 Manner artic.	90*	100	100	100	100 (\pm 0)
	1 Place artic.	47*	59*	94	76*	92.2 (\pm 5.7)
	1 Voicing	90	100	100	100	94.7 (\pm 8.3)

* = score below means -1.96 S.D.

consonant-vowel syllables was still impaired 18 days after the acute stage. Repetition of syllables embedded in words was clearly better than repetition of the same isolated syllables and did not differ according to the position of the syllable in the word.

To sum up, the patient's pattern of neurolinguistic deficit was characterized by (1) the prominence of syllabic discrimination errors on pairs differing by 1 feature, especially on the place contrast, (2) the sparing of isolated vowel identification contrasting with its impairment when a consonant was added and (3) the improvement of identification after embedding syllables in words.

DISCUSSION

A patient free of previous hearing disturbances suddenly exhibited a complete deafness, evolving 2 weeks later to an auditory agnosia for environmental and verbal sounds. The neurophysiological and neuroimaging investigations strongly suggested that these psychoacoustical deficits were due to bilateral damage to the auditory radiations. According to Ozdamar, Kraus, Curry et al. (1982), deafness with normal BAEPs components and absence of MAEPs components, such as that found in the present patient, suggests a cortical deafness. The generator sites of MAEPs still remain controversial. However, Woods, Clayworth, Knight et al. (1987) and Kaseda, Tobimatsu, Morioka et al. (1991) suggested that *Pa* wave abnormalities might reflect a temporal cortex dysfunction, while *Na* wave would originate from subcortical structures, possibly the auditory radiations. In the present patient, the absence of *Na-Pa* waves might suggest a bilateral disruption of auditory radiations, which run in the internal and external capsule, and in the temporal isthmus (Luxon, 1981). These structures were damaged bilaterally by the lesions, which spared the thalamus and temporal cortex, although we cannot exclude a transient dysfunction of temporal cortices.

Cortical deafness is usually due to bilateral lesion of the temporal lobes involving both auditory cortices. However, aphasia is usually associated with cortical deafness (Lhermitte et al., 1971; Graham, Greenwood and Lecky, 1980), and its absence is an argument in favor of a subcortical form of deafness. To our knowledge, only 3 cases of acute deafness were related to bilateral disruption of auditory radiations. Clark and Russel's (1938) patient suffered from a persisting deafness after a bilateral infarction of the external capsule; since no audiological testing was performed, it is not possible to rule out that auditory agnosia was secondary to true pure tone sounds deafness. The patient of Barraquer-Bordas, Pena-Casanova and Pons-Irazazabal (1980) had deafness without aphasia following bitemporal infarcts. The authors emphasized the prominent role played by the subcortical lesion of the auditory

radiations. Hirano's case (Hirano, 1973, 1983) became completely deaf after the second of 2 bilateral hemorrhages in the external capsule. The case reported by Tabira, Tsuji, Nagashima et al. (1981) is more difficult to interpret since imaging failed to reveal any abnormality. Tanaka et al. (1991) maintain that bilateral lesion of auditory radiations due to extensive damage of the external capsule would lead to persistent deafness. The comparison of MRI data between their patients and ours suggests that secondary resolution of deafness in the present case might be due to restricted damage of auditory radiations.

In the present case, agnosia for verbal sounds was characterized by the prominence of syllabic discrimination errors on pairs differing by the place contrast, the sparing of isolated vowel identification and the improvement of identification of syllables embedded in words. We used a syllabic discrimination task only based on the heard stimuli whereas, in most of the previous studies, subjects had to match the heard stimuli with written syllables. Our procedure does not demand access to a phonetic representation to discriminate between syllables and, consequently, the deficit is ascribable to the acoustical differences between syllables. The analysis of the auditory stimuli may require a storage lasting up to several seconds (Darwin, Turvey and Crowder, 1972), and the possibility of a short term memory deficit has to be taken into account. In the present case, verbal short term memory was spared, especially at interval of 3 sec., which was similar to the interval separating the syllables in the discrimination test. Moreover, a deficit of short term memory would not explain the prominent deficit of discrimination of place of articulation. The benefit provided by increasing the lexical constraints indicates the efficiency of lexical representations when they are spared in apperceptive agnosia. Finally, the evaluation of vowels identification usually focused on isolated vowels and did not take into account the possible role of an added consonant, except in the study of Praamstra et al. (1991). The finding that isolated vowels were perfectly repeated but that the performance dramatically decreased when a consonant was added, is consistent with the hypothesis that the unit used to decode fluent speech might be larger than individual phonemic features (Shankweiler and Studdert-Kennedy, 1967) and could be the syllable itself (Blumstein, Tartter and Michel, 1977; Massaro, 1977; Mehler, 1981; Pierrel, 1989).

We believe that 3 converging arguments support the hypothesis that the impairment of non-specific auditory processes accounts for the deficit observed in apperceptive auditory agnosia. *First*, to the best of our knowledge, a case with apperceptive auditory agnosia and without auditory processing disorder has never been reported. The only exception might be the patient with non verbal auditory agnosia reported by Lambert, Eustache, Lechevalier et al. (1989), whose pattern of syllabic discrimination errors was close to ours. Temporal acuity measures were not clearly impaired; however, interstimuli intervals smaller than 10 msec were not tested, and loudness and tone frequency discrimination were impaired. It is also worth reminding that, in the present case, the temporal resolution assessed with the click fusion test returned near the normal range when agnosia resolved. *Secondly*, the pattern of neurolinguistic impairment is consistent with a deficit at the stage of auditory processes. This is strongly suggested by the acoustic properties of verbal stimuli (Auerbach et al., 1982). Speech sounds are characterized by 3 universal attributes: spectra usually exhibit series of narrow peaks and valley, the amplitude of the sound rises and falls at a rate of about 3-4 per second and spectral changes are rapid, occurring over time-intervals lower than 40 msec (Stevens and Blumstein, 1981). Processing verbal sounds required fine temporal discrimination. Not surprisingly, our patient's performance was higher for syllabic pairs differing in 2 features than for the one-features contrast. The latter probably requires more accurate auditory processing than the former. The sparing of feature voice discrimination and the impairment of feature place can also be explained by the deficit of auditory processes. Perceiving feature voice requires the detection of low frequency periodicity due to glottal pulses within the first tens of msec after the onset of the burst, whereas the discrimination of the feature place lies in the analysis of the first tens of msec of the stimulus (Stevens and Blumstein, 1981). Moreover, the neurolinguistic evaluations in patients with probably associative form of auditory agnosia suggest a higher vulnerability of the feature voice discrimination (Saffran et al., 1976; Basso, Casati and Vignolo, 1977; Paamstra et al., 1991). *Thirdly*, cases with auditory agnosia related to subcortical lesions (Motomura et al., 1986; Kazui et al., 1990) showed deficits close to those of our patient, i.e. impairment of loudness, tone discrimination, click fusion test and phonemic discrimination. Experimental data support the hypothesis that

the disruption of auditory radiations induces a prominent impairment of non-specific auditory processes. The bilateral ablation of the temporal lobes impairs the processing of temporal pattern of sounds and the analysis of complex sound components (Neff, 1961; Dewson, Cowey and Weiskrantz, 1970; Heffner and Heffner, 1989).

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