Electrophysiological Correlates of Hysterical Hearing Loss

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Abstract

A female patient exhibiting hysterical hearing loss in her left ear consistently demonstrated reduced P3 amplitude for event-related potentials (ERPs) to left monaural stimulation, while displaying normal amplitude and latency for N1 to either ear stimulation. This result suggested that although stimuli in the affected ear were successfully conducted up to the auditory cortex, further processing was "repressed". ERP examination was concluded to be useful in clarifying the brain mechanisms underlying some hysterical disorders.

Introduction

A diagnosis of conversion disorder (a kind of hysteria) is usually established when medical examination fails to confirm the presence of any somatic disease causing the exhibited symptoms. However, such a procedure has the limitation of relying on circumstantial evidence which is based only upon "exclusive" diagnostic criteria. In this context, we have not yet identified any "positive" laboratory findings that confirm the diagnosis of hysteria.

Recently, EP (evoked potential) examinations have been introduced as diagnostic tool for hysteria. However, as concluded by Howard et al.(1986), they have so far failed to provide clinicians with an objective means to rule out the presence of somatic diseases. Howard et al. stated in their review that "in all reported cases, EP tests of the visual or auditory system in patients with hysterical sensory loss have given normal results." Although only a few studies have been conducted on the N1 and P3 components of ERPs (event-related potentials) in hysteria, these late EP components are expected to offer

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"positive" clues to the diagnosis, because they reflect the higher cognitive functions, such as attention and context updating, which are possibly disturbed in hysteria. Gordon et al.(1986) reported that the amplitudes and latencies of P3s in somatization disorders were not significantly different from those of normal controls. As for conversion disorders, Towle et al.(1985) reported that visual stimuli elicited P3s in a hysterically blind patient. These studies represent an extension of the EP results described above that do not necessarily give a "positive" diagnostic base.

Here, we report on an intriguing P3 finding observed in a female patient, diagnosed as having unilateral hysterical hearing loss, which might provide a "positive" basis for such a diagnosis.

Case report

NK, a 26-year-old nurse, first recognized a hearing loss in her left ear five years ago while listening through a stethoscope. Because the disturbance to her daily life caused by hearing loss was mild, she did not have it examined. The left hearing loss was clearly demonstrated by audiometry when she had a medical examination for new employment at the age of 25. Her hearing loss was diagnosed to be of psychological origin because further examination excluded any underlying otological disease. A spiral-shaped defect in her visual field also supported this diagnosis. Half a year later, at the suggestion of her employer, she visited the Department of Otorhinolaryngology, Tokyo University Hospital, where she was diagnosed as having unilateral functional hearing loss, and was then transferred to the Department of Neuropsychiatry.

During psychiatric interviews, she complained of left hearing loss and bilateral otodynia; however, she denied any apparent inconvenience in her daily life due to the hearing loss. She sometimes failed to hear the psychiatrist's questions and asked that they be repeated. She insisted on leaving the hearing problem untreated as long as no serious organic disease was found. She had no history of head trauma or substance abuse, and a magnetic resonance imaging (MRI) of her brain revealed no abnormal findings.

Otological examinations

Otological findings were as follows: 1) pure tone audiogram indicated a severe hearing loss in the left ear (0-10 dB in the right ear, and 50-110 dB in the left ear), with markedly variable results in the left ear across repeated examinations. Perception of consonant-vowel syllables ("speech discrimination") was also highly impaired in the left ear (no correct perception at 110dB). 2) Normal stapedius reflexes were elicited bilaterally at 75dB with normal tympanograms (type A) in both ears, which demonstrated no abnormal conductance up to the middle ear. 3) BAEP (brainstem auditory evoked potential) confirmed an absence of any clear abnormality in stimulus conduction through the brainstem in that waves I to V for 90dB tones were normal in both ears, and that wave V was observed even for 30dB tones in the left ear. These results indicated that she had

severe hearing loss for both pure tones and syllables in the left ear in the absence of any abnormality from the external acoustic meatus to the brainstem.

Event-related potentials

ERPs were recorded twice, four weeks apart, using the oddball paradigm from Pz in the first, and from Fz and Pz in the second examination. In the oddball paradigm, 200 tone bursts of 1000Hz (25%) and 2000Hz (75%) at 90dB were delivered during one session, with the former being assigned as targets to be counted mentally. Three sessions of binaural, right monaural and left monaural stimulation were conducted in this order. The amplitude and latency of N1 in Fz and the amplitude of P3 in Pz were determined in the ERP waveforms. The N1 (P3)-peak was defined as the most negative (positive) peak between 80 - 130 (260 - 600) msec after the stimulus onset. Further details of the procedures are described elsewhere (Niwa & Hayashida, 1992).

In the required task, the subject could perform mental counting of the target stimuli and answered correctly in the binaural and right monaural stimulations. However, she claimed that she could not hear any sound in the left monaural stimulation. Visual inspection of her ERP waveforms indicated that N1 was observed consistently across the three modes of stimulation, whereas the appearance of P3 was quite variable in the two examinations. P3 was clearly identified only in the binaural stimulation for the first examination, and only in the right monaural for the second examination, with no obvious peaks observed for the remaining stimulations (Fig. 1).

The amplitudes and latencies of the N1 peak of this patient will be described in the following, along with the corresponding data for 25 normal controls (in parentheses) of approximately the same age (male 14, female 11; mean age 23.9, SD 3.1) which were obtained in the binaural stimulation. N1 amplitudes in Fz were 9.46, 10.81 and 9.45 microvolts in the binaural, right monaural and left monaural stimulations, respectively (normal controls: mean 7.15, SD 2.82). N1 latency in Fz for each stimulation mode was 94, 88 and 90 msec, respectively (normal controls: mean 99, SD 14). The P3 amplitude in Pz for the first examination was 6.25 microvolts in the binaural stimulation and that for the second was 6.41 microvolts in the right monaural stimulation, with no obvious peaks being identified in the remaining stimulation modes (normal controls: mean 11.69, SD 5.05).

The results indicate the following. The N1 amplitude of the patient was slightly larger, and the N1 latency a little shorter, than the normal means. Three modes of stimulation elicited almost the same amplitude and latency of N1 compared to each other. In contrast, stimuli in the left ear consistently failed to elicit P3s; and, although elicited by right monaural or binaural stimuli, the P3 peak amplitude was approximately as small as 1SD below the normal mean.

Discussion

The clinical history and the results of the otological examinations described above

clearly demonstrate that the patient's hearing loss was "non-organic and functional" in otological terms, and "hysterical" in psychiatric terms. Based upon the ERP data, we were able to obtain insight into the brain mechanism of the hysterical process in this patient, which was made possible by the unusual fact that her hearing loss was unilateral.

According to the idea that the Fz-dominant N1 is generated in the temporal cortex (Nataanen, 1987), the normal amplitude and latency of N1 in this patient suggest that the auditory stimuli were successfully conducted up to the auditory cortex in the temporal lobe irrespective of the side of stimulation. However, the consistent absence of P3 in the left monaural stimulation in our two examinations suggests that further processing beyond the auditory cortex was disturbed. Because P3 was distinctly present in other stimulation modes, the P3-generating mechanism itself was thought not to be organically damaged. Instead, two possible explanations can be posited for the P3 reduction; that is, a possibility of a functional disturbance in the P3-generating mechanism, or the possibility that some brain mechanism presents itself as a negative ERP component which overlaps P3. At any rate, brain mechanisms leading to P3 reduction has been speculated to be important aspects of the pathophysiology in the conversion disorder, possibly constituting a physiological substrate of "repression", for instance subjective hearing loss in the left ear in this case. Although our patient was able to count the stimulus numbers correctly, P3s were not consistently elicited for the right monaural or binaural stimulation. This result suggests that the processing of the right ear stimuli was also affected to some extent in its later stage.

Thus, the combination of normal N1 and reduced P3 can be thought to be "positive" laboratory findings suggesting that the hearing loss of this patient is hysterical. In addition, ERP examinations may offer an objective means not only of diagnosing hysterical disorders but also of clarifying the underlying brain mechanisms of such disorders.

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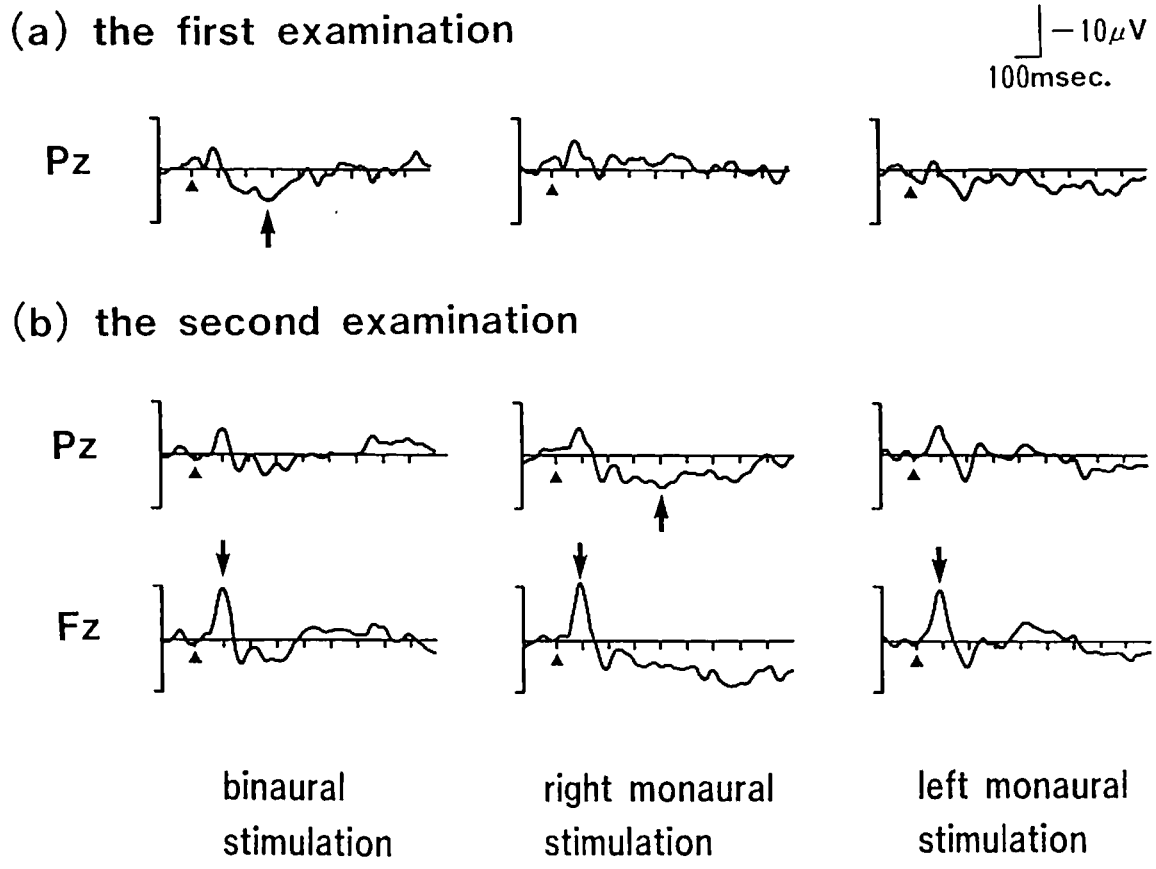

Figure 1 ERP waveforms in binaural (left column), right monaural (middle column), and left monaural (right column) stimulations for (a) the first and (b) the second examinations. The triangles and arrows indicate the stimulus onset and the peak of N1 or P3 components, respectively.