

Mismatch Negativity in the Neurophysiologic/Behavioral Evaluation of Auditory Processing Deficits: A Case Study

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Abstract

The subject of this case report is an 18-year-old woman with grossly abnormal auditory brain stem response (ABR), normal peripheral hearing, and specific behavioral auditory processing deficits. Auditory middle latency responses (MLRs) and cortical potentials N1, P2, and P300 were intact. The mismatch negativity (MMN) was normal in response to certain synthesized speech stimuli and impaired to others—consistent with her behavioral discrimination of these stimuli. Behavioral tests of auditory processing were consistent with auditory brain stem dysfunction. A neuropsychological evaluation revealed normal intellectual and academic performance. The subject was in her first year of college at the time of the evaluation. This case study is important because: (1) Although there have been several reports of absent/abnormal ABR with preserved peripheral hearing and deficits in auditory processing, little is known about the specific nature of the auditory deficits experienced by these individuals. Such information may be valuable to the clinical management of patients with this constellation of findings. (2) Of interest is the information that the mismatch negativity (MMN) cortical event-related potential can bring to the evaluation of patients with auditory processing deficits. The MMN reflects central auditory processing of small acoustic differences and may provide an objective measure of auditory discrimination. (3) From a theoretical standpoint, a patient with neural deficits affecting specific components of the auditory pathway provides insight into the relationship between evoked potentials and physiological mechanisms of auditory processing. How do various components of the auditory pathway contribute to speech discrimination? How might evoked potentials reflect the processes underlying the neural coding of specific features of speech stimuli such as timing and spectral cues?

There have been several reports of absent/abnormal ABR with normal peripheral hearing and deficits in auditory processing (Berlin, Hood, Cecola, Jackson,

& Szabo, 1993; Lenhardt, 1981; Starr, McPherson, Patterson, Luxford, Shannon, Sininger, Tonokawa, & Waring, 1991; Worthington & Peters, 1980). Reports of young patients with Brainstem Auditory Processing Syndrome (BAPS) have focused on babies and toddlers with absent ABR, but only mild-to-moderate loss of hearing sensitivity (Kraus, Özdamar, Stein, & Reed, 1984). Although it has been speculated and partially documented that these patients show deficits in auditory processing, the specific nature of these deficits is poorly understood. Little is known about how these individuals function in "real life" or how these deficits will affect them over time.

The awareness that such patients exist has resulted in the identification of an increased number of such patients in hearing/otology clinics. Starr et al. (1991) reported a case of severely impaired auditory temporal processing in which ABR was abnormal. The patient was totally unable to understand speech despite normal hearing sensitivity. It would be valuable to learn the communication capabilities of other patients with abnormal ABR but preserved peripheral hearing. Additional reports of the status of such patients would be valuable from a prognostic standpoint and could aid in making appropriate recommendations for treatment and counseling.

The subject of this case report is an 18-year-old woman with subclinical hydrocephalus, a grossly abnormal ABR, normal peripheral hearing, and specific deficits in auditory processing, as revealed by behavioral tests. Other evoked potentials measured included auditory middle latency responses (MLRs) and cortical potentials N1, P2, the mismatch negativity (MMN), and P300. The patient also underwent a battery of conventional behavioral tests of auditory processing in which the primary signal was altered through electronic filtering, was presented in competition with speech or noise, or required binaural processing. Additional behavioral testing included psychoacoustic discrimination of pairs of well-defined synthesized speech stimuli, which were the same stimuli used to elicit the cortical potentials. The patient also underwent a neuropsychological evaluation of intellectual and academic function.

One of the principal objectives for presenting this case is to illustrate what the MMN may bring to the evaluation of a patient with auditory processing

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deficits. The MMN is a neurophysiological reflection of sensory processing within auditory cortex for which important clinical applications are envisioned (Näätänen, 1992, review; Näätänen, Gaillard, & Mantysalo, 1978; Kraus, McGee, Micco, Sharma, Carrell, & Nicol, 1993a; Kraus, McGee, Sharma, Carrell, & Nicol, 1992; Kraus, Micco, Koch, McGee, Carrell, Sharma, Wiet, & Weingarten, 1993b; Korpilähti, Ek, & Lang, 1992). The MMN is a passively-elicited, objective measure that does not require attention or a behavioral response to the stimuli. It is elicited by a physically deviant stimulus occurring in sequence with a series of homogeneous, standard stimuli. The MMN is a neuronal response to minimal changes in acoustic parameters such as frequency, intensity, location, and duration (Näätänen, 1992; Näätänen, Paavilainen, Alho, Reinikainen, & Sams, 1987; Paavilainen, Karlsson, Reinikainen, & Näätänen, 1989; Sams, Aulanko, Aaltonen, & Näätänen, 1990; Sams, Paavilainen, Alho, & Näätänen, 1985) and can be elicited by speech stimuli (Aaltonen, Niemi, Nyrke, & Tuhkanen, 1987; Kraus et al, 1993a,b; Sams et al, 1990; Sharma, Kraus, McGee, Carrell, & Nicol, 1993). In addition to reflecting the processing of stimulus features such as frequency, intensity, and duration, the MMN is a response to stimulus change. Therefore, it may be a significant neurophysiological tool because it is stimulus change that fundamentally characterizes the acoustics of human speech. Current models of speech perception place special emphasis on the importance of patterns of stimulus change over time as the primary means of conveying linguistic information (Gordon, 1988; Kewley-Port, 1983; Remez, Rubin, Pisoni, & Carrell, 1981; Van Tasell, Soli, Kirby, & Widen, 1987).

Another principal objective in presenting this case is to highlight the benefit of combining electrophysiological and behavioral measures in assessing patients with central auditory deficits. The combined approach taken in the assessment of this patient reveals information relevant to the treatment and prognosis of other individuals with a similar constellation of symptoms. From a theoretical standpoint, results from a patient with specific neural deficits of the auditory pathway can provide insight into the relationship between evoked potentials, physiological mechanisms of auditory processing, and the neural coding of speech stimuli.

Case Report

The patient was an 18-year-old left-handed female who reported a long-standing "hearing" difficulty. Throughout her childhood, numerous behavioral audiometric tests revealed normal pure-tone sensitivity to sound frequencies in the speech range.

However, the patient reported particular difficulty hearing in the presence of background noise and perceived her hearing problem as frustrating and debilitating. Despite the behavioral and electrophysiological deficits described below, this patient was succeeding academically and was in her freshman year of college at the time of testing.

Magnetic resonance imaging, performed subsequent to evoked potentials testing, revealed moderate diffuse enlargement of the ventricular system (subclinical hydrocephalus) and no midline shift of structures, and was otherwise normal. The results of a recent neurological evaluation, her first, were unremarkable.

Evoked Potentials

With respect to generating systems, the ABR reflects the activity of the VIIIth nerve and brain stem auditory pathways (Hecox & Galambos, 1974; Møller & Jannetta, 1985; Picton, 1986; Starr & Don, 1988). The MLR reflects the activity of primary and nonprimary divisions of the auditory thalamo-cortical pathways with contributions from the mesencephalic reticular formation (Kraus & McGee, 1992, review). The N1/P2 complex has a complex generating system involving sensory and motor processes (Davis, 1939; Näätänen & Picton, 1987; Picton, Hillyard, Krausz, & Galambos, 1974). The generating system for the MMN is largely modality dependent (Ritter, Simson, & Vaughan, 1983), with major sources originating from the supratemporal plane (Alho, Paavilainen, Reinikainen, Sams, & Näätänen, 1986; Csépe, Karmos, & Molnár, 1987; Giard, Perrin, Pernier, & Bouchet, 1990; Hari, 1990; Kaukoranta, Sams, Hari, Hämäläinen, & Näätänen, 1989; Näätänen & Picton, 1987; Sams, Kaukoranta, Hämäläinen, & Näätänen, 1991). The P300 generating system is diffuse (Sutton, 1965; Donchin, 1981; Polich & Starr, 1984), involving numerous multisensory cortical and subcortical structures (Buchwald, 1989; Halgren, Stapleton, Smith, & Altafullah, 1986; Harrison, Buchwald, Kaga, Woolf, & Butcher, 1988; Knight, Hillyard, Woods, & Neville, 1980; Knight, Scabini, Woods, & Clayworth, 1989; McCarthy, Wood, Williamson, & Spencer, 1989).

ABR. ABR recordings were obtained from Cz, referenced to the ipsilateral earlobes with Fpz as ground. Click stimuli were presented at rates of 11 and 31/sec monaurally at 70 dB HL through insert earphones. Responses were filtered from 30 to 2000 Hz (12 dB/octave), and 20 msec of poststimulus time was recorded.

Bilaterally, atypical early waves (?I, II) reflecting activity from the VIIIth nerve and lower brain stem were present, with reduced/absent responses from

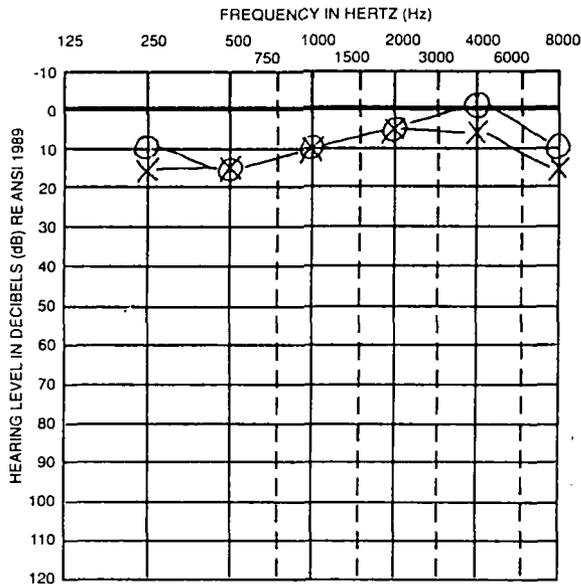
the rostral brain stem (IV and V). Response morphology was somewhat more distinct for the right ear. Replicated recordings in response to clicks presented at 31/sec are shown in Figure 1.

MLR. The MLR was obtained as previously described (Kraus, Özdamar, Hier, & Stein, 1982) using a coronal montage, with active electrodes at Cz and over each temporal lobe, referenced to the ipsilateral mastoid, with forehead as ground. Responses were filtered from 10 to 2000 Hz (12 dB/octave) and 80 msec of poststimulus time was recorded. The MLR was elicited by click stimuli delivered monaurally at 70 dB HL, at a rate of 11/sec, through insert earphones.

The MLR was normal bilaterally, consistent with our norms (Kraus et al, 1992; Özdamar & Kraus, 1983), as shown in Figure 2. Waves Na, Pa, and TP41 were of normal amplitude, latency, and topography. Wave Pa was largest at the vertex and symmetric over the temporal lobes.

Cortical Responses. Stimuli. The synthesized speech stimulus pairs were chosen based on parameters of theoretical and clinical interest. Although a wide variety of acoustic dimensions are used to distinguish phonemes, stimulus spectra, spectral (formant) transition, and transition duration are critical for speech perception (Stevens & Blumstein, 1978). Interestingly, the perception of rapid formant transitions in stop

AUDIOGRAM



MLR

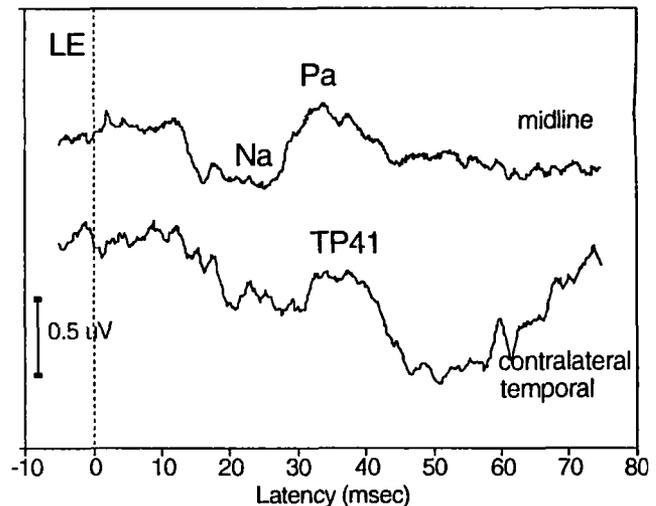
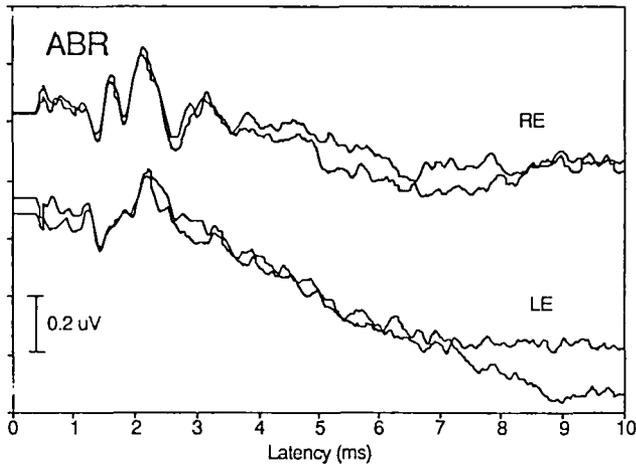
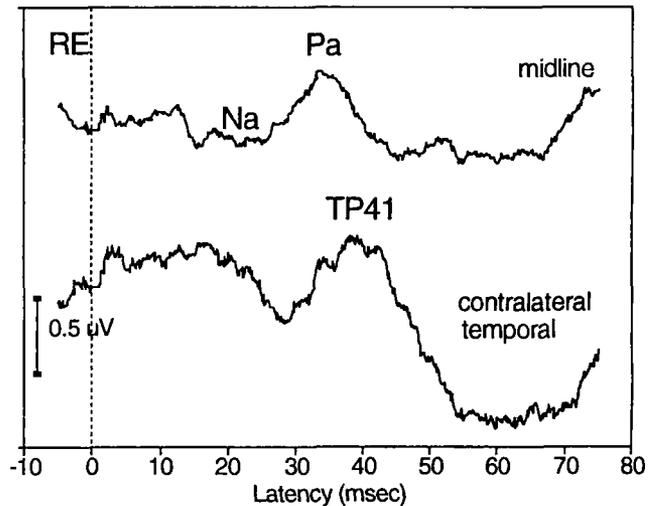


Figure 1. Top: Pure-tone audiogram indicating normal hearing sensitivity bilaterally. Bottom: Abnormal ABRs bilaterally, with atypical waves up to 3 msec after stimulus onset, and absent ABR thereafter.

Figure 2. MLRs showing normal waves Pa (midline) and TP41 (temporal lobe).

consonants has been shown to be particularly vulnerable in clinical cases of processing disorders (Elliott, Hammer, & Scholl, 1989; Tallal, Stark, & Mellitis, 1980). The specific reasons for this vulnerability are not clear, but the perception does seem to require very precise and complex neural processing at various levels in the auditory system.

The stimulus pairs /da-/ga/ differ in spectral (formant) transition, whereas /ba-/wa/ differ in the duration of the formant transition. The specific stimulus pairs used to elicit the MMN were chosen based on behavioral discrimination results of various pairs from a group of normal listeners (see Kraus et al, 1992, 1993a,b; Pisoni, Aslin, Perey, & Hennessy, 1982; Walley & Carrell, 1983, for speech synthesis and delivery parameters). In a two-alternative forced-choice procedure, subjects judged whether pairs were the same or different. P(c)max, a measure of behavioral discrimination (Green & Swets, 1974), was calculated for /da-/ga/ and /ba-/wa/ pairs. The average P(c)max for the 3 /da-/ga/ pairs and 3 /ba-/wa/ pairs are listed in Table 1 for 10 normal-hearing adults. From those P(c)max data, we labeled the pairs "easy," "hard," and "hardest" to discriminate.

In this patient, the MMN was elicited by synthetically generated variants of /da-/ga/ and /ba-/wa/. Acoustically, /da/ and /ga/ differed in the onset frequency of the second and third formant transitions. The differences in the onset frequencies of the "easy," "hard," and "hardest" stimulus pairs were 38, 15, and 8 Hz for F2 and 438, 175, and 88 Hz for F3. For all /da-/ga/ stimuli, the duration of the formant transition was 40 msec. Variants of /ba-/wa/ differed in the relative duration of formant transitions for F1 and F2. Pairs described as "hardest," "hard," and "easy" to discriminate differed in transition durations by 5, 10, and 15 msec, respectively, with the /ba/ transition being held constant at 40 msec.

The P300 was elicited by the easily discriminable stimulus pair /di-/da/, differing in the steady-state frequencies of F2 and F3. P300 also was tested in response to the "easy" /da-/ga/ pair.

Electrophysiological recording. Speech stimuli were presented to the right ear at 75 dB SPL through insert earphones. For the MMN, a modified oddball paradigm was used in which a deviant stimulus (probability of occurrence = 15%) was presented in a series of standard stimuli (probability of occurrence = 85%). The interstimulus interval was 1 sec. Evoked responses elicited by standard stimuli and those elicited by deviant stimuli were averaged separately. The evoked responses were collected in blocks of 25 deviant stimuli and approximately 140 standard stimuli. Eight blocks (1200 standard and 200 deviant stimuli) were collected for each stimulus pair. The MMN should occur in response to the deviant stimulus only when it is presented in the oddball paradigm and not when the deviant stimulus is presented alone. Therefore, as a control, 8 blocks of 25 presentations of "deviant" stimuli were presented alone as described previously (Kraus et al, 1992, 1993a,b).

To control for level of arousal and to minimize the subject's attention to the test stimuli, the subject was instructed to watch a captioned, videotaped movie and to ignore the stimuli used to elicit the MMN. During P300 recording, the patient was asked to count the number of deviant stimuli (probability of occurrence = 20%). Thus, the MMN was passively elicited, whereas P300 was assessed using an active task.

Evoked potentials were recorded from Fz/A2 with the forehead as ground. MMNs were obtained to the "hard" pairs first. If a response was obtained, testing progressed to the "hardest" pair. If no MMN was obtained to the "hard" pair, the testing progressed to the "easy" pair. Trials contaminated by eye movements (recorded with a supraorbital-to-lateral-canthus electrode montage) were eliminated from the averaged responses. The recording window included a 50-msec prestimulus period and 500 msec of poststimulus time, with a total of 512 sampling points/sweep (sampling rate = 1024 points/sec). Responses were analog band-pass filtered on-line from 0.1 to 100 Hz (12 dB/octave) and digitally low-pass filtered off-line at 40 Hz

Table 1. Behavioral discrimination data.

Stimulus Pair	Latency (msec)			Amplitude (μ V)		Area (msec \cdot μ V)	P(c)max (%)	
	Onset	Peak	Offset	Onset/Peak	Peak/Offset		Normal	Subject
/da-/ga/easy	152	215	320	3.1	2.3	404	98	58
/da-/ga/hard		No MMN			No MMN	No MMN	65	37
/da-/ga/hardest		Not tested			Not tested	Not tested	53	48
/ba-/wa/easy		Not tested			Not tested	Not tested	89	96
/ba-/wa/hard	153	238	336	5.7	3.3	596	67	93
/ba-/wa/hardest		No MMN			No MMN	No MMN	58	41

with a Blackman filter. Averaged response waveforms were converted to ASCII format and transferred to a spreadsheet for analysis. Waveforms were "baseline adjusted" by adjusting the baseline to a zero point defined by the mean voltage of the prestimulus baseline period.

Mismatch negativity. The MMN is more variable and more time consuming to record than the more familiar ABR, MLR, and P300 potentials. Thus, one cannot record numerous replications in order to ensure validity in a clinical setting. Therefore, we used a statistical method (Kraus et al, 1993a,b) to ensure that the MMN identified visually was, in fact, a significant negative deflection. We recorded MMNs in blocks of 25 deviant stimulus presentations and then used statistical procedures on the subaverages in order to validate the significance of the MMN observed in the grand averages (Kraus et al, 1993a,b). The MMN often is not readily apparent in the subaverages because too much physiological noise is still present, but the statistical procedures demonstrate whether the MMN observed in the grand average is indeed a statistically significant response.

The analysis procedure involved examining the pattern of three sets of grand average waveforms: the responses to the standard and the deviant, the response to the deviant stimulus presented repetitively (not in the oddball paradigm), and the difference waves (deviant minus standard, deviant minus deviant presented alone). The MMN was identified visually as a relative negativity following the N1. The MMN was evident in the deviant and the difference waveforms, but not the standard or deviant-alone waveforms. The N1 was apparent in the standard, deviant, and deviant-alone waveforms. The examination of this pattern allowed a visual identification of the onset, peak, and offset of the MMN. T-tests were performed on the points in the difference wave subaverages that corresponded in latency to the MMN onset, peak, and offset. Those tests determined whether a significant negative deflection occurred at the MMN peak.

This analysis confirmed that an MMN was obtained to the "easy" /ga/-/da/ pair and the "hard" /ba/-/wa/ pair, but not to the "hard" /da/-/ga/ or the "hardest" /ba/-/wa/ pair. Responses to the standard and deviant stimuli as well as the corresponding difference waves are shown in Figure 3. When present, MMNs were of normal latency and morphology and unusually large in amplitude as compared to a group of normal adults (Kraus et al, 1992, 1993a; Sharma et al, 1993). In our experience, peak latency of the MMN occurs at approximately 235 msec (± 30) in response to speech stimuli in

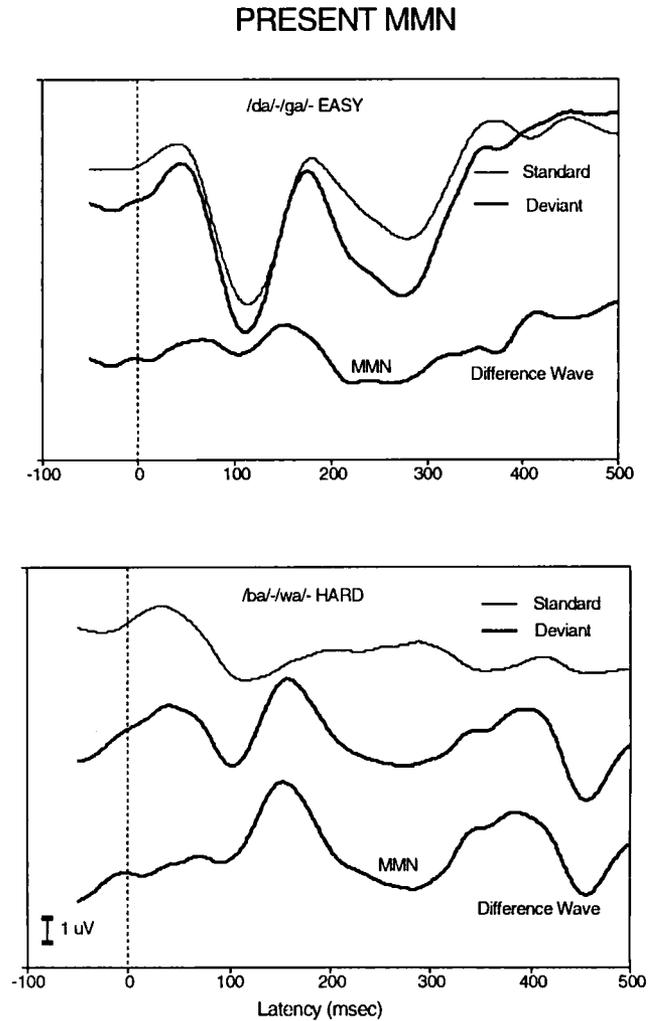


Figure 3. Cortical event-related potentials in response to (top) the standard stimulus /ga/ and deviant stimulus /da/ and (bottom) the standard stimulus /ba/ and deviant stimulus /wa/. Waves N1 and P2 occur in response to both stimuli. The MMN is evident in the difference wave obtained by subtracting the response to the standard stimulus from the response to the deviant stimulus.

normal-hearing subjects. MMN amplitude and area are approximately $2 \mu\text{V}$ (± 1) and $135 \mu\text{V} \times \mu\text{sec}$ (± 50), respectively.

For the "easy" /da/-/ga/ pair, the MMN occurred at a peak latency of 215 msec (152-320 msec onset-to-offset). MMN amplitudes were 3.1 and 2.3 μV from onset-to-peak and offset-to-peak, respectively, with an area of $404 \mu\text{V} \times \mu\text{sec}$. These stimuli were just perceptively different to the patient [P(c)max = 58%]. The MMN was absent in response to the "hard" /da/-/ga/ pair [P(c)max = 37%]. In comparison, normal subjects exhibit MMNs to both stimuli and show better behavioral discrimination [P(c)max = 98 and 65%, respectively].

For the /ba/-/wa/ stimuli, an MMN was obtained in response to the "hard" pair, with a peak latency of 238 msec (153-336 onset-to-offset). Wave amplitude was considerably larger than normal (5.7 and 3.3 μ V onset and offset-to-peak, respectively, with an area of 596 μ V \times msec). No MMN was elicited to the "hardest" /ba/-/wa/ pair. For the /ba/-/wa/ stimuli, the patient's behavioral performance compared favorably to normal subjects. Discrimination was 93% for the "hard" pair and 41% for the "hardest" pair, whereas the normal subject mean discrimination was only 67% for the "hard" pair and 58% for the "hardest" pair.

Statistical testing confirmed the absence of an MMN to "hard" /ga/-/da/ and "hardest" /ba/-/wa/ pairs. The absence of an MMN can be seen in Figure 4, where the responses to the deviant stimuli /da/ or /wa/ are compared in two conditions: (1) when they were presented in isolation (alone) and (2) when they occurred in the oddball paradigm. If an MMN had occurred, it would have been evident in the response to the deviant stimulus in the oddball paradigm. Notice instead that the responses overlap, indicating no MMN.

Figure 3 shows that the N1 waveform was normal. A positivity, possibly wave P3a, follows the MMN at 300 msec in the /ba/-/wa/ difference waveform (Roth, 1973; Squires, Squires, & Hillyard, 1975). P3a is a passively elicited component of P300 and is typically seen only when stimulus differences are well above discrimination threshold (Näätänen, Simpson, & Loveless, 1982; Snyder & Hillyard, 1976). Note that the positivity is only apparent for the

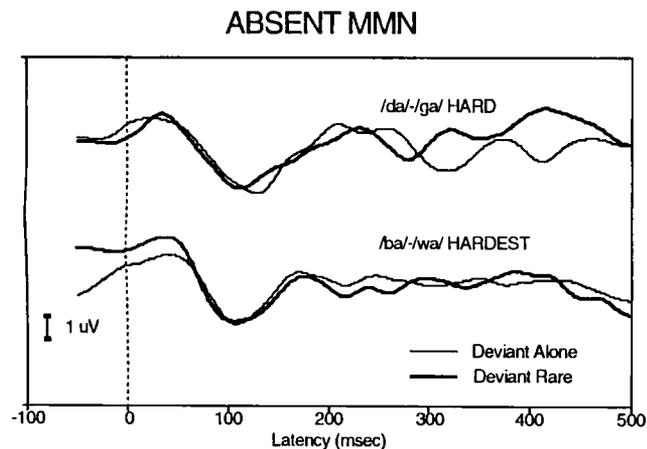


Figure 4. The absence of MMN is seen by comparing the response to the deviant stimuli /da/ and /wa/ (1) when they are presented alone (alone) and (2) when presented in the context of the oddball paradigm (deviant). If an MMN were present, it would be evident only in response to the deviant stimulus in the oddball paradigm. Evident from the figure is that the deviant and alone responses overlap, indicating no MMN.

/ba/-/wa/ stimuli, which were easier for the patient to discriminate.

P300. The P300 was present at about 300 msec in response to the stimuli /di/ and /da/, as shown in Figure 5. This wave was unusually large, on the order of 30 μ V. Normative values for our laboratory using these stimuli range from 10 to 20 mV. The subject correctly identified the occurrence of deviant stimuli used to elicit this response. However, no P300 was elicited by the "easy" /da/-/ga/ stimulus pair, which she had difficulty discriminating behaviorally. This result is consistent with reports that elicitation of P300 requires a stimulus pair that can be easily discriminated (Picton & Hillyard, 1988).

Summary of Electrophysiological Tests

ABRs were markedly abnormal. Auditory evoked potentials generated by structures central to and including the midbrain (MLR, N1, P2, MMN, P300) were elicited. The MMN was present when elicited by stimuli that the subject could distinguish behaviorally and was absent to stimuli that she discriminated below chance. MMN findings were consistent with behavioral observations of her impaired discrimination of stimuli differing in spectral content and her better discrimination of stimuli varying minimally in duration.

Behavioral Tests of Auditory Processing

Pure-tone testing indicated peripheral hearing sensitivity within normal limits for each ear (PTA = 10 dB HL). Speech reception thresholds were 10 dB HL bilaterally. Word recognition of familiar monosyllables (W-22 words) presented at 40 dB SL (re: speech

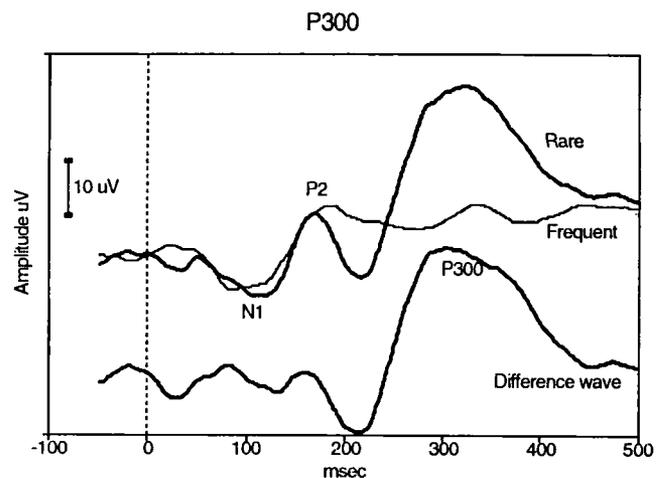


Figure 5. Responses to the standard stimulus /di/ and deviant stimulus /da/. P300 is evident in the corresponding difference wave at 300 msec. N1 and P2 responses are evident at normal latency and morphology in response to both stimuli.

reception thresholds) were 88% for the right ear and 100% for the left ear.

Tests of auditory processing abilities included Speech-in-Noise, Low-Pass Filtered Speech, Staggered Spondaic Word, Binaural Fusion, and Pitch Patterns Sequence Tests. Descriptions of these tests and the patient's performance are summarized in Table 2.

Abnormal performance was observed for Speech-in-Noise, Low-Pass Filtered Speech, and Staggered Spondaic Word Tests, reflecting her difficulty in perceiving low redundancy speech. On the Binaural Fusion Test, in which words possess greater acoustic and linguistic redundancy, performance was normal.

Performance on the Pitch Patterns Sequence Test was also normal. Her performance improved significantly when signal redundancy was increased by the addition of visual and/or linguistic cues. Overall findings are consistent with brain stem dysfunction.

Intellectual, Neuropsychological, and Academic Evaluation

A neuropsychologist administered the Wechsler Adult Intelligence Scale (WAIS-R), Halstead-Reitan Neuropsychological Test Battery for adults, Selective Reminding Test of Memory, Boston Naming Test,

Table 2. Test descriptions and patient performance.

Test	Description	Result
Speech in Noise	<p><i>Test 1:</i> Monosyllabic words were presented without visual cues under earphones with ipsilateral multispeaker babble^a at signal to noise ratio (S:N) of +5, and in sound field with competing babble at S:Ns of 0, +5, and +10.</p> <p><i>Test 2:</i> A second speech-in-noise test was in which words were presented with visual cues.</p>	<p><i>Test 1:</i> Performance under earphones was 45% for each ear, contrasted with 88% and 100% without any competing signal. Sound field performance deteriorated with decreasing S:N from 44% to 24% for +10 and +5 S:N, respectively. The patient was unable to do the test at 0 S:N.</p> <p><i>Test 2:</i> With visual cues scores improved to 96% at 10 S:N and 84% at 0 S:N.</p>
Low-Pass Filtered Speech*	Familiar monosyllabic words (NU#6) that have been filtered to remove or reduce high frequency cues (cut-off 1 kHz) were presented monaurally. This test assesses recognition of signals that are degraded to reduce the redundant acoustic cues that normally aid in the discrimination of speech.	Scores were 60% and 48% for right and left ears, respectively, significantly below normal limits bilaterally.
Staggered Spondaic Word (SSW, Katz 1962)	A dichotic listening test in which two spondaic words were presented one to each ear, in such a way that the second part of the first spondee overlaps in time with the first part of the second spondee. This test taxes binaural interaction ability to divide attention and keep information separate between ears.	Scores were obtained for competing and non competing portions of the test. The scores were overcorrected for the right ear and normal for the left ear. (The continuum of performance ranges from overcorrected to severely depressed).
Binaural Fusion	Spondaic words that have been electronically filtered to produce a high band (1.9–2.1 kHz) segment and low band (500–700 Hz) were presented dichotically. The subject hears one segment in each ear and repeats the whole word. The test requires that the patient effect closure or summation on the target and differs from low pass filtered speech test in that the words used possess greater acoustic and linguistic redundancy.	Scores were normal for each dichotic condition.
Pitch Patterns Sequence ^a	A three tone sequence was presented monaurally and the subject was asked to label the pattern (i.e., low-high-low, high-high-low etc). This test taxes cortical hemispheric integration, temporal sequencing, spatial abilities, auditory memory, pitch perception, pattern recognition (Pinheiro & Musiek, 1985).	Scores were 100% for each ear.

^a Tapes obtained from Auditec, 156 W. Argonne Dr., St. Louis, MO 63122.

Gordon Diagnostic Systems, Controlled Oral Word Fluency (FAS), Go-NoGo Test, Paced Auditory Serial Addition Task (PASAT), Auditory Discrimination Test, G-F-W Diagnostic Auditory Discrimination Test, Visual Aural Digit Span Test, Peabody Picture Vocabulary Test, Clinical Evaluation of Language Fundamentals, Test of Adolescent Language, Detroit Tests of Learning Aptitude, Woodcock-Johnson Tests of Cognitive Ability, Stanford Diagnostic Reading Test, Test of Written Spelling, Test of Written Language and portions of the Stanford-Binet Intelligence Scale, Wechsler Memory Scale, and Woodcock-Johnson Tests of Achievement.

On the basis of the intellectual, neuropsychological, and academic evaluation, the patient had excellent abstract reasoning skills and above-average verbal and nonverbal skills. She had difficulty in auditory discrimination when she was required to listen to two words and decide whether they were the same or different, yet she had superb auditory attention in tasks such as repeating digits and performing rapid-paced mental additions. She had excellent ability to discriminate whether rhythms were the same or different and to identify nonsense words that were spoken (with written, multiple choices to which she could refer).

The patient exhibited a mild disability in receptive and expressive language. Her language comprehension was average, but she had difficulty with higher level abstract language processing. Although expressive language skills were well developed, she demonstrated word retrieval difficulties and occasional awkward formulation. These difficulties may have been exacerbated by her bilingual background. She demonstrated average to above-average reading, writing, and mathematics skills.

Discussion

Summary of Test Results

This normal-hearing subject demonstrated specific deficits in auditory processing as revealed by behavioral tests and a grossly abnormal ABR. Auditory MLRs and cortical potentials N1, P2, and P300 were intact. The MMN was normal in response to certain stimuli and impaired in response to others. These results were consistent with her behavioral discrimination of the same stimuli. Behavioral tests of auditory processing were consistent with auditory brain stem dysfunction. The neuropsychological evaluation revealed normal intellectual and academic performance.

Behavioral Auditory Processing Battery

Behavioral auditory processing tests confirmed this patient's complaint of marked inability to process

speech under adverse listening conditions and suggested a brain stem-level site of signal disruption. Her performance improved significantly when signal redundancy was increased by the addition of visual and/or linguistic cues.

Specifically, her severely abnormal performance in speech-in-noise tests was consistent with her complaint of difficulty understanding speech. Generally, these tests are nonlocalizing with respect to site of lesion because those same abnormalities are observed for patients having dysfunction at any level of the auditory system from the cochlea to cortex (Morales-Garcia & Poole, 1972; Olsen & Noffsinger, 1975). Recognition of low-pass filtered speech has been shown to be severely compromised in patients with diffuse brain stem lesions (Lynn & Gilroy, 1977; Musiek & Geurnink, 1982). Staggered Spondaic Word Test performance was consistent with subcortical disorder (Katz, 1962, 1968, 1970). Poor performance on the Binaural Fusion Test also has been reported for patients having brain stem dysfunction (Linden, 1964; Matzker, 1959; Smith & Resnick, 1972). Pinheiro, Jacobson, and Boller (1982) and Pinheiro and Musiek (1985) have confirmed the usefulness of the Pitch Patterns Sequence Test in identifying cortical dysfunction that disrupts sequencing and temporal ordering abilities. On the other hand, patients having brain stem dysfunction tend to show normal performance (Musiek & Geurnink, 1982; Musiek, Weider, & Mueller, 1983) on this test, consistent with the present case.

Evoked Potentials

Detection and Discrimination as Revealed by Auditory Evoked Potentials. Auditory evoked potentials revealed abnormal ABRs, normal MLRs, identifiable N1 and P2 components, normal P300, and selective impairment of the MMN cortical potential. The presence of auditory evoked potentials reflecting activity of pathways beyond the brain stem provides neurophysiological evidence that, although auditory processing was impaired, this patient can make use of auditory signals successfully. These results support behavioral observations.

The MLR, N1, and P2 waveforms are neurophysiological indices underlying detection of acoustic events. The MMN can be considered a neurophysiological reflection of sensory discrimination of stimulus differences. The P300 reflects the cognitive processing of stimulus differences. P300 is not specifically diagnostic of auditory processing disorders because it is a complex, multimodality response generated by many auditory and nonauditory structures. In this case, P300s were not elicited by stimuli

that the patient could not easily discriminate behaviorally, consistent with the cognitive basis of the P300 (Picton & Hillyard, 1988).

In the present case, the MMN was a neurophysiological reflection of behavioral discrimination ability. The MMN was elicited by stimuli that were distinguished behaviorally and was absent to stimuli that were discriminated below chance. The fact that the MMN as elicited by stimuli that were perceived just above chance [$P(c)_{max} = 58\%$] suggests that a correspondence between the MMN and discrimination threshold may exist. MMN findings support behavioral observations of impaired discrimination for stimuli differing in spectral content and better discrimination for stimuli varying minimally in duration. These findings suggest that the MMN might be used diagnostically to determine which aspects of the acoustic signal are discriminated abnormally.

Consequently, the MMN may serve as a useful clinical tool. Previous studies have revealed that the MMN can be elicited to stimulus pairs that are at behavioral discrimination threshold (Kraus et al, 1993a; Sams et al, 1985). The present case is consistent with these reports because an MMN was elicited by the /da/-/ga/ "easy" stimulus pair that was just perceptibly different [$P(c)_{max} = 58\%$]. The presence of an MMN indicates that these stimuli are discriminated at a preattentive level.

This finding raises the question of whether behavioral training would enhance this patient's conscious discrimination abilities. It has been shown that training will improve performance on behavioral discrimination tasks (Pisoni et al, 1982). If an MMN is present, it indicates that the brain processes the stimulus differences. Whether training would improve behavioral discrimination and carry over to improve her speech discrimination ability is unknown.

How Do Subcomponents of the Auditory Pathway Contribute to Stimulus Processing? *Cortical versus Subcortical Processing.* Several studies have shown that cortical potentials and peripheral processing can be independent (Gravel, Kurtzberg, Stapells, Vaughan, & Wallace, 1989; Picton, Stapells, & Campbell, 1981; Picton, Woods, Baribeau-Braun, & Healy, 1977; Satya-Murti, Wolpaw, Cacace, & Schaffer, 1983). That is, normal peripheral hearing, abnormal ABR, and relatively normal cortical evoked potentials can exist in the same patient. This constellation of findings probably does not arise from a unitary phenomenon. Some researchers hypothesize disruption of type I fiber synapses (Starr et al, 1991). Others propose afferent-efferent disconnection (Berlin et al, 1993), whereas still others suggest brain stem

dysfunction (Kraus et al, 1984), particularly in the presence of hydrocephaly (Kraus, Özdamar, Heydemann, Stein & Reed, 1984). The patient in this case report falls in the last category, as evidenced by preservation of wave I and subclinical hydrocephalus.

Patients with this test result pattern exhibit a variety of behavioral symptoms, leading to different interpretations of the underlying mechanisms. For example, our patient clearly differs from the case described by Starr et al (1991) in which the patient suffered disruption of temporal processing, determined psychophysically. Their patient had no ABR. In contrast, our patient functions reasonably well. Her auditory deficit is apparent only in certain circumstances (in noise) and with certain auditory stimuli (fast frequency transitions). Interestingly, waves I and II of her ABR were present.

Implications for Neural Encoding. All of these studies hypothesize that the ABR shows abnormalities because of its dependence on synchrony of neural firing. The MLR waves are more resistant to disruptions of synchrony, as evidenced by their lack of sensitivity to changes in stimulus rise time (Vivion, Hirsch, Frye-Osier, & Goldstein, 1980). In Starr et al's case, the behavioral breakdown of temporal processing fits well with the supposition of disorganization of neural synchrony.

In our patient, the disruption in the ABR suggests a disruption of synchrony and, therefore, a disruption of temporal encoding. Her difficulty hearing in noise is consistent with the idea that temporal encoding is disrupted. In the presence of background noise, perception of vowel spectra is based only on temporal encoding (Sachs, Voigt, & Young, 1983; Miller, Barta, & Sachs, 1987).

Although noise has a particularly deleterious impact on stop consonant perception (Horst, 1989), it is not generally agreed whether the encoding of stop consonants involves temporal or rate-place mechanisms or both. Temporal representation (phase-locking) may account for neural responses to aperiodic stop consonants whose spectra are rapidly changing at the VIIIth nerve (Javel & Mott, 1988; Sachs et al, 1983). Phillips and Hall (1990) note that while cortical neurons do not demonstrate sufficiently rapid phase-locking, their temporal precision is sufficient to encode the rapid transitions of stop consonants. At cortical levels demonstrated differences in neural responses to stop consonants also show complex interactions between temporal encoding and tonotopic pattern (Steinschneider, Arezzo, & Vaughan, 1990). In our patient, her impairment may have affected the mediation of precise, rapid changes in neural firing pattern. Another possibility is that a portion of the tonotopic array is affected so that the disruption

of /da/-/ga/ is merely coincidental to the ABR desynchronization.

The /ba/-/wa/ distinction is also likely to be temporally encoded. However, the temporal contrast in /ba/-/wa/, 5 msec or more, is a much grosser difference than the /da/-/ga/ contrast. The perception of the /ba/-/wa/ contrast appeared to be unaffected in this patient, perhaps indicating that the imprecision in synchrony was not great enough to disrupt the coding of larger timing differences. Also, transition duration differences in /ba/-/wa/ may be encoded differently, involving timing differences of neural discharges to formant onset/offset, for example.

The selective impairment of MMN in this case study implies that the MMN may be used diagnostically to determine which aspects of the acoustic signal are abnormally discriminated. Several lines of evidence indicate that subcomponents of acoustic signals are processed distinctly by the auditory system. For example, single neurons are specifically responsive to elements of the speech signal (Makela, Hari, & Linnakivi, 1987; Mendelson & Cynader, 1985; Phillips, Mendelsen, Cynader, & Douglas, 1985; Steinschneider, Arezzo, & Vaughan, 1982; Steinschneider, Schroeder, Arezzo, & Vaughan, 1992; Whitfield & Evans, 1965). With respect to the MMN, "frequency" and "duration" MMNs appear to be processed distinctly (Giard et al, 1990; M. Sams et al, personal communication).

Functional Implications. Finally, our patient demonstrated a high level of auditory function as well as selectively intact cortical potentials. Atypically large MMN and P300 magnitudes may possibly reflect enhanced acoustic processing of certain stimulus features at the cortical level compared to normal listeners. The presence of those potentials in the absence of normal brain stem responses suggests that compensation and neural plasticity have occurred in the central auditory system of this patient. Abnormal transmission in the auditory brain stem could induce compensatory alterations similar to the plastic changes documented after sensory deprivation, such that non-impaired systems are heightened (Kujala, Alho, Paavilainen, Summala, & Naatanen, 1992; Neimeyer & Starlinger, 1981; Neville & Lawson, 1987; Neville, Schmidt, & Kutas, 1983; Wood, McCarthy, & Bentin, 1985).

Summary

From a practical and prognostic standpoint, it is encouraging that, despite the highly abnormal ABR, this patient was able to utilize auditory input effectively and to function well academically. The

abilities of other patients with absent/abnormal ABRs (Berlin et al, 1993; Kraus, Smith, & Grossman, 1985; Lenhardt, 1981; Starr et al, 1991; Worthington & Peters, 1980) range from the complete inability to use acoustic information (i.e., functionally deaf) to the high level of performance shown by this patient. It can be argued that the intact cortical potentials provide neurophysiological support for this patient's success in using acoustic information and for her high level of functioning. The MMN (as well as behavioral measures of discrimination) revealed which specific aspects of the speech signal were being discriminated abnormally. The results were encouraging to the patient because long-standing hearing complaints had received little support or understanding. We hope that the combination of evoked potential measures (reflecting the activity of multiple portions of the auditory pathway in response to specific acoustic cues) and behavioral information will aid in making the most appropriate management decisions for these patients.

Conclusions

This case study illustrates that the combination of several auditory evoked potential measures, which assess the auditory system from peripheral to central pathways, can identify dysfunctional areas neuroanatomically as well as provide diagnostic and prognostic information. The behavioral information (auditory processing, academic and intellectual performance) provides a picture of what might be expected functionally from such a combination of evoked potentials findings. In babies or subjects who are not as easily testable behaviorally, clinicians may have to rely heavily on electrophysiological information. Cortical potentials, especially the MMN, may provide important information for the prognosis and management of patients who have absent abnormal ABR, normal peripheral hearing, and auditory processing disorders.

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