APHASIOLOGY, 1998, VOL. 12, NO. 7/8, 499–507

A comparison of the mismatch negativity (MMN) event-related potential to tone and speech stimuli in normal and aphasic adults

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Abstract

We evaluated the mismatch negativity (MMN) event-related potential (ERP) in normal and aphasic adults to tone and speech stimuli to determine aphasic patients' auditory discrimination and the relationship between MMN measures and severity of aphasia. MMNs were present in 89% of normal subjects and 79% of aphasic subjects to tone stimuli. MMNs were present in 100% of normal subjects and 54% of aphasic subjects to speech stimuli. The duration of the MMN ERP to speech stimuli was significantly related to severity of aphasia on the Western Aphasia Battery, Porch Index of Communicative Ability, and the Token Test. Thus, not all aphasic people show an early, preconscious orientation response to tone and speech stimuli. However, the duration of this response, when present, to speech stimuli appears to be related to the severity of aphasia.

Introduction

The mismatch negativity (MMN) is an event-related potential (ERP) which is generated by the presentation of a deviant, 'oddball' stimulus in a series of standard stimuli. A standard stimulus is presented 80–90% of the time and a deviant stimulus is presented 10–20% of the time. Responses to the standard and deviant stimuli are averaged separately.

As shown in figure 1, the MMN peaks at approximately 200 ms after stimulus onset and is seen in the difference waveform produced by subtracting the response to the standard stimulus from that to the deviant stimulus. It is related to the discrimination of stimulus differences and reflects the earliest, automatic processing of these differences (Naatanen and Alho 1995, Naatanen *et al.* 1982, Sams *et al.* 1985). These differences include frequency, time, and intensity (Naatanen *et al.* 1982, Naatanen *et al.* 1987, Sams *et al.* 1985) as well as complex phonemic differences (Aaltonen *et al.* 1987, Kraus *et al.* 1992, Sams *et al.* 1991). The MMN is

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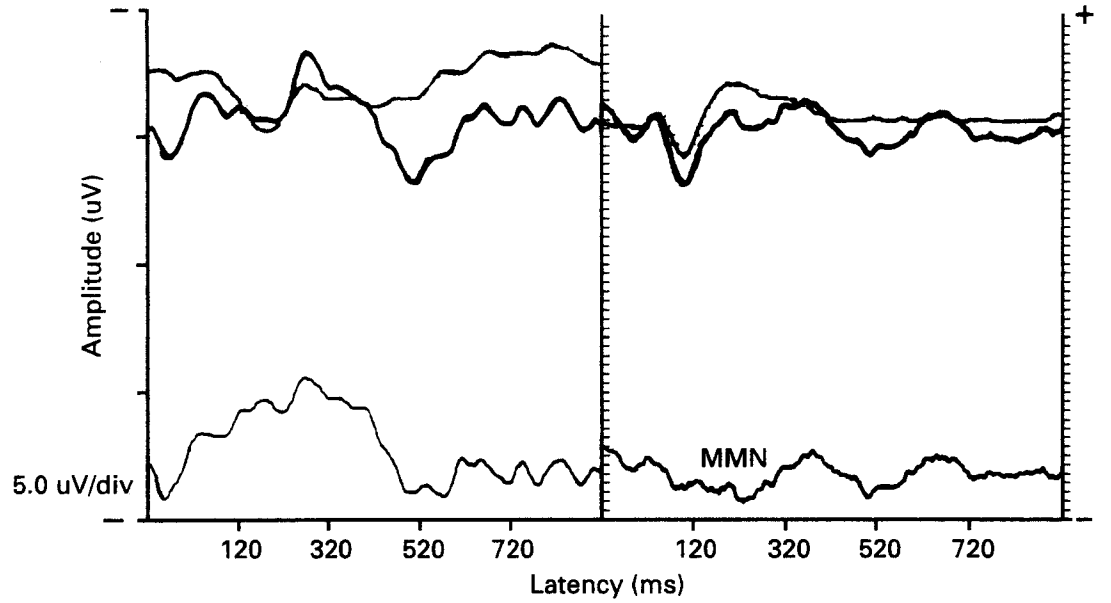

Figure 1. Mismatch negativity (MMN) demonstrated at the vertex. Event-related potentials to the deviant ('oddball') stimulus (dark line) and standard stimulus (light line). On the left, the MMN event-related potential is absent. On the right, the MMN event-related potential is present.

generated in the primary auditory cortex (Alho *et al.* 1986, Sams *et al.* 1991) and probably has an additional component generated in the frontal cortex (Giard *et al.* 1990).

Peach *et al.* (1992) collected MMNs to tone bursts in four aphasic patients and concluded that MMN latencies suggest that automatic attending is preserved in aphasia. Aaltonen *et al.* (1993) examined MMNs to tones and vowels in two aphasic subjects with anterior lesions and two aphasic subjects with posterior lesions. All subjects displayed MMNs to the tone stimuli, but only the subjects with anterior lesions displayed MMNs to the vowel stimuli.

Because the MMN has been shown to change with changes in discrimination ability, it may be useful in assessing auditory discrimination ability in aphasic patients for whom traditional behavioural tests may be confounded because they require volitional responses. We evaluated the MMN in normal and aphasic adults to tone and speech stimuli to determine aphasic patients' auditory discrimination and the relationship between MMN measures and severity of aphasia. Specifically, we asked: does the MMN event-related response differ between normal and aphasic adults to tone and speech stimuli, and do MMN measures in aphasic patients predict severity of aphasia?

Method

Nine normal adults with no history of neurological involvement and 24 adults who were aphasic subsequent to a left hemisphere ischaemic stroke participated in the study. Descriptive data for normal and aphasic subjects are shown in table 1. The groups did not differ significantly ($p < 0{\cdot}05$) in age or education. Months post-onset in the aphasia group ranged from 1 to 96 months with a mean of 8·59 months.

The Porch Index of Communicative Ability (PICA) (Porch 1967), Western Aphasia Battery (WAB) (Kertesz 1982), Aphasia Quotient sub-tests, and the Token Test (Spreen and Benton 1977) were administered to all aphasic subjects. Severity

Table 1. Subject descriptive data

	Group			
	Normal		Aphasic	
Variable	$\bar{x}$	SD	$\bar{x}$	SD
Age (years)	66·00	8·47	60·25	10·15
Education (years)	13·33	4·09	12·38	2·52
Months post-onset			8·59	21·27
PICA overall			9·58	2·90
WAB AQ			48·80	32·68
Token Test			59·63	62·94

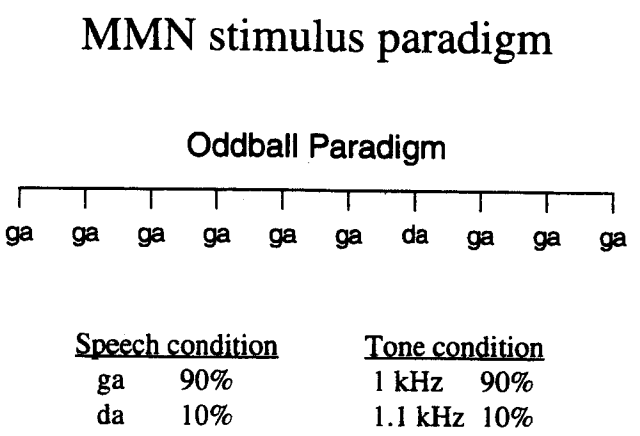

Figure 2. The MMN stimulus ('oddball') paradigm. In the tone condition, a 1 kHz tone is the standard stimulus, presented 90% of the time, and a 1·1 kHz tone is the deviant ('oddball') stimulus, presented 10% of the time. In the speech condition, /ga/ is the standard stimulus, presented 90% of the time, and /da/ is the deviant ('oddball') stimulus, presented 10% of the time.

of aphasia ranged from severe to mild across subjects, for example, the PICA overall score ranged from 4·34 to 14·67 with a mean of 9·58. All subjects in both groups demonstrated an estimated speech reception threshold (SRT) of at least 40 dB HL in the better ear determined by the Carhart method (average of 500 and 1000 Hz–2 dB) (Carhart 1971).

MMN procedures

As shown in figure 2, subjects were tested with two pairs of stimuli: a pair of pure tones (1000 and 1100 Hz) and a pair of synthesized consonant–vowel syllables (/ga/ and /da/). For tones, 1000 Hz was the standard stimulus, presented 90% of the time, and 1100 Hz was the deviant, 'oddball' stimulus, presented 10% of the time. For syllabic stimuli, /ga/ was the standard stimulus, presented 90% of the time, and /da/ was the deviant, 'oddball' stimulus, presented 10% of the time. MMN stimuli were presented at approximately 85 dB SPL to the right ear via an insert earphone at a rate of one per second. The stimuli were presented until event-related potentials to 100 deviant stimuli were recorded.

MMNs were recorded with a PC-based system that allowed stimulus presentation and triggering of the Biologic Navigator while subjects watched a video tape, *Mr Bean*. The video tape, without sound, maintained an alert state in the subject while

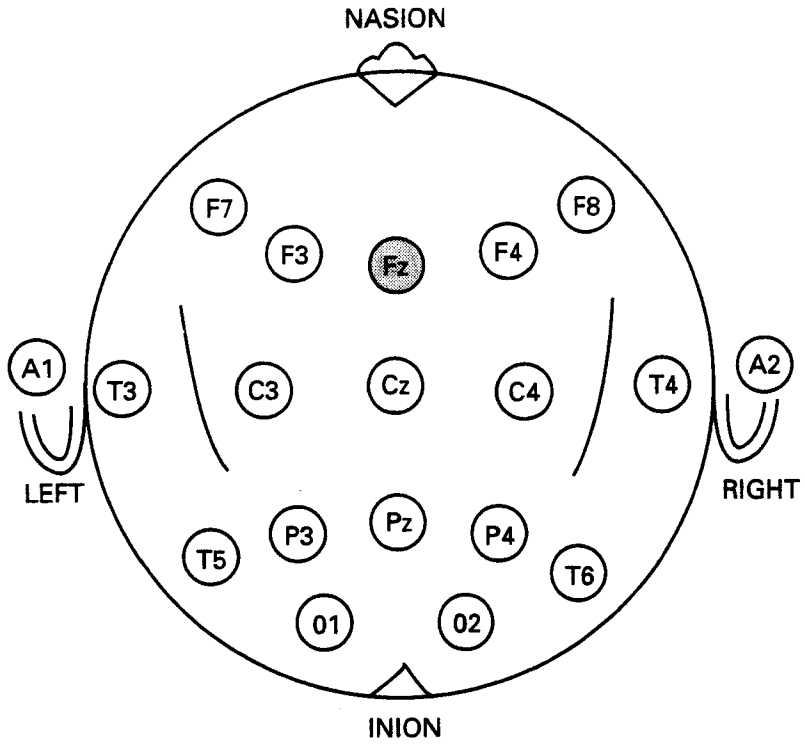

Figure 3. MMNs were recorded from a midline electrode site, Fz, referenced to the right ear, A2.

reducing attention to the experimental stimuli. For example, as shown in figure 2, in the syllabic condition, the subject watched the video tape, *Mr Bean*, without sound, and heard a series of /ga/ stimuli with an occasional, interspersed, /da/ stimulus. Figure 3 indicates that MMNs were recorded from a midline electrode site, Fz, referenced to the right ear, A2. A second electrode was placed near the eye to monitor and reject artifact from eye movements. Responses were band-pass filtered from 0·1 to 30 Hz on-line.

MMN data

Responses to standard and deviant stimuli were averaged separately. Then, response to the standard stimulus was subtracted from the response to the deviant stimulus for each subject for each stimulus pair. Presence of the MMN was confirmed by visual inspection of the standard, deviant, and difference waveforms. An MMN was identified visually if a negative-going peak was seen in the latency range of 100 to 400 ms.

We examined three components of the MMN event-related potential—latency, duration, and peak amplitude. MMN peak latencies, in milliseconds, were chosen at the maximum negativity occurring between 100 and 400 ms after stimulus presentation. Onset and offset of the MMN were marked at the points corresponding to the maximum positivities at the beginning and end of the MMN waveform. MMN duration, in milliseconds, was calculated as offset minus onset latency. MMN onset-to-peak amplitude, in microvolts, was calculated by subtracting the onset amplitude from the peak amplitude value.

Results

Table 2 indicates that eight of the nine (89 %) normal subjects and 19 of the 24 (79 %) aphasic subjects showed an MMN event-related potential to tone stimuli.

Table 2. Number of normal ($n = 9$) and aphasic ($n = 24$) subjects showing (+) and not showing (−) MMN event-related responses to tone and speech stimuli

		MMN			
		Tone		Speech	
Groups	*n*	+	−	+	−
Normal	9	8	1	9	0
Aphasic	24	19	5	13	9

Table 3. Normal (N) and aphasic (A) group mean comparisons for MMN tone stimuli measures

	Group		
Measure	N	A	Difference
Latency	235·25	214·05	21·20
Duration	166·66	99·00	67·66*
Peak	−3·48	−3·26	0·22

* Significant at $p < 0{\cdot}02$.

Table 4. Normal (N) and aphasic (A) group mean comparisons for MMN speech stimuli measures

	Group		
Measure	N	A	Difference
Latency	294·11	309·92	−15·81
Duration	127·77	64·86	62·91*
Peak	−5·20	−1·71	3·49**

* Significant at $p < 0{\cdot}02$.
** Significant at $p < 0{\cdot}001$.

For speech stimuli, all nine (100 %) of the normal subjects and 13 of the 24 (54 %) aphasic subjects showed an MMN event-related potential.

Table 3 shows MMN measures—latency, duration, peak amplitude—for the normal and aphasic groups for tone stimuli. There was no significant difference in latency ($t = 0{\cdot}75$, d.f. = 25, $p = 0{\cdot}47$) or peak amplitude ($t = 0{\cdot}23$, d.f. = 31, $p = 0{\cdot}82$) between groups. However, duration was significantly shorter ($t = 2{\cdot}37$, d.f. = 31, $p = 0{\cdot}02$) in the aphasic group.

Table 4 shows MMN measures—latency, duration, peak amplitude—for the normal and aphasic groups for speech stimuli. There was no significant difference in latency ($t = -0{\cdot}62$, d.f. = 20, $p = 0{\cdot}54$) between groups. However, duration was significantly shorter ($t = 2{\cdot}50$, d.f. = 29, $p = 0{\cdot}02$), and peak amplitude was significantly lower ($t = -4{\cdot}65$, d.f. = 29, $p = 0{\cdot}001$) in the aphasic group.

Table 5. Correlations between MMN tone and speech measures—latency (L), duration (D), and peak (P)—and severity of aphasia

	MMN measures					
	Tone			Speech		
Aphasia measures	L	D	P	L	D	P
WAB AQ	0·11	0·16	0·21	0·11	0·49*	0·20
WAB auditory	0·04	0·24	0·19	0·19	0·59**	0·33
PICA overall	0·06	0·19	0·20	0·04	0·54**	0·28
PICA auditory	0·07	0·02	0·40	0·04	0·42*	0·22
Token Test	0·00	0·26	0·13	0·20	0·49*	0·10

* Significant at $p < 0·05$.
** Significant at $p < 0·01$.

To determine the relationship between MMN measures and severity of aphasia, we computed correlations between tone and speech measures and aphasic subjects' performance on the WAB AQ, WAB auditory subsection, PICA overall, PICA auditory modality, and Token Test total score. Table 5 shows that none of the tone measures was significantly related ($r = 0·00$–$0·40$, d.f. $= 24$, $p = 0·08$–$0·88$) to any measure of aphasic severity. For the speech measures, duration correlated significantly with all aphasia measures—WAB AQ ($r = 0·49$, d.f. $= 24$, $p = 0·04$), WAB auditory subsection ($r = 0·59$, d.f. $= 24$, $p = 0·01$), PICA overall ($r = 0·54$, d.f. $= 24$, $p = 0·01$), PICA auditory modality ($r = 0·42$, d.f. $= 24$, $p = 0·04$), and Token Test ($r = 0·49$, d.f. $= 24$, $p = 0·04$). Positive correlations imply that the duration of the MMN event-related potential was significantly related to the severity of aphasia—the more severe the aphasia, the shorter the MMN duration. No significant correlations were observed between severity of aphasia and MMN latency or peak amplitude ($r = 0·04$–$0·33$, d.f. $= 24$, $p = 0·31$–$0·98$).

Discussion

The data provide answers to our two questions. First, the MMN event-related potential to tone stimuli was present in more normal subjects (89%) than aphasic subjects (79%). Similarly, all normal subjects, but only 54% of the aphasic subjects, produced an MMN event-related potential to speech stimuli. Among the MMN measures, duration to tone stimuli was significantly shorter in the aphasic group. Also, for speech stimuli, duration was significantly shorter and peak amplitude was significantly lower in the aphasic group. Thus, the presence and the configuration of the MMN event-related potential, especially for speech stimuli, appears to differ between normal and aphasic adults.

Second, although none of the MMN measures for tone stimuli was significantly correlated with aphasic severity, duration of the MMN event-related potential to speech stimuli was significantly correlated with aphasic severity on all language measures. Thus, duration of the MMN event-related potential to speech stimuli appears to predict severity of aphasia.

The MMN response reflects an early, preconscious orientation to stimuli.

Moreover, the presence of the MMN response implies the ability to discriminate between stimuli—standard and deviant. Most of our aphasic subjects displayed early, preconscious orientation to non-speech (tone) stimuli, implying that they were able to discriminate between the standard and deviant stimuli. Conversely, only approximately half of our aphasic subjects displayed an MMN response to speech stimuli, implying the absence of early, preconscious orientation in some aphasic people to speech and, perhaps, inability to discriminate between standard and deviant speech stimuli. Only the duration of the MMN response to speech stimuli was significantly related to severity of aphasia. Thus, it appears that some aphasic people do not orient, early and preconsciously, and, perhaps, discriminate, speech stimuli. This may suggest that exploration of the MMN response in aphasia, a language disorder, should employ language stimuli.

Previous investigations of the MMN event-related potential in aphasic adults used the measure to test attention allocation and to compare the effects of anterior and posterior left hemisphere damage on the presence or absence of the MMN. Peach and colleagues (1992), using tone stimuli, concluded that even though MMN latencies suggest that automatic attending is preserved in aphasia, aphasic people have reduced attentional resources available to make discriminative decisions. In a subsequent investigation, Peach and colleagues (1994) reported that their MMN data indicated that aphasic subjects require more time to allocate fewer attentional resources to the detection of changes in an auditory signal and engage attention differently from normal subjects, even at the earliest, preconscious phases of orientation.

Conversely, as indicated above, we found some aphasic subjects (21 %) who did not show an MMN event-related potential to tone stimuli, and only 54 % showed an MMN event-related potential to speech stimuli. These results imply that some aphasic subjects do not display an early, preconscious phase of orientation, especially for speech stimuli. Our results do not indicate whether this early orientation is essential for engaging and allocating attention or whether its presence implies attention has been engaged and/or allocated.

Aaltonen *et al.* (1993), using tone and vowel stimuli, observed aphasic subjects with either anterior or posterior lesions who produced the MMN event-related potential to pure tones. However, only subjects with anterior lesions produced the MMN event-related potential to vowel stimuli. The authors concluded 'that vowels and pure tones are at least partly processed in different cortical areas' (p. 149). However, the presence of the MMN to vowels in the subjects with an anterior lesion did not indicate that subjects could discriminate the vowels. Moreover, 'auditory discrimination as measured by MMN... is not directly related to language comprehension' (p. 151).

Most of our aphasic subjects (79 %) produced an MMN event-related potential to tone stimuli, but only 54 % produced an MMN event-related potential to speech stimuli. Unlike Aaltonen *et al.* we found that the duration of the MMN event-related potential to speech stimuli was significantly related to overall severity of aphasia—WAB AQ and PICA overall—and severity of auditory comprehension–WAB auditory subsection, PICA auditory modality, and Token Test total score. However, the magnitude of the relationships was not strong, $r = 0{\cdot}59$ and below.

Obviously, there are methodological differences between Peach *et al.*'s, Aaltonen *et al.*'s, and our efforts. For example, the stimuli differ—Peach *et al.* employed pure tones, Aaltonen *et al.* employed pure tones and vowels, and we employed pure

tones and syllables. In addition, MMN measures differed. Peach *et al.* measured latency and peak amplitude; Aaltonen *et al.* measured peak amplitude; and we measured latency, duration, and peak amplitude.

There is a need to define and standardize MMN measures. Similar assumptions are being made, for example, that shorter latency, longer duration, and larger peak amplitude imply better discrimination. However, criteria for what constitutes an MMN event-related potential may differ among investigators, specifically the criterion for differentiating a 'weak' MMN—long latency, short duration, and reduced peak amplitude—from the absence of an MMN.

Our interest in the MMN event-related potential is its potential for providing an electrophysiological confirmation of aphasic severity observed on behavioural measures. In addition, we speculate that the presence, absence, and, when present, configuration of the MMN event-related potential may be prognostic. For example, the presence of the MMN event-related potential in an aphasic person who fails to respond to the same stimuli behaviourally may predict the emergence of the behavioural response in the future. This, of course, is speculation in search of empirical evidence. Careful collection of MMN event-related potentials and behavioural responses to the same stimuli, early post-onset, and systematic re-evaluation over time would provide the evidence required.

Acknowledgement

This research was supported by the Department of Veterans Affairs Rehabilitation Research and Development Program and the Department of Veterans Affairs Medical–Surgical Research, Historically Black Colleges and Universities (HBCU) Program.

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