

Attentional modulation of the mismatch negativity elicited by frequency differences between binaurally presented tone bursts

LEONARD J. TREJO,^a DAVID L. RYAN-JONES,^b AND ARTHUR F. KRAMER^a

^aDepartment of Psychology, University of Illinois at Urbana-Champaign, Champaign, IL, USA

^bTraining Research Department, Navy Personnel Research and Development Center, San Diego, CA, USA

Abstract

We examined the attentional sensitivity of the frequency-change mismatch negativity (MMN). Subjects listened to a binaural mixture of a narrative and a series of tone bursts that included 1200-Hz standards and two deviants (1000 and 1400 Hz). In the attend-tones condition, subjects responded to one deviant and ignored the narrative. In the attend-words condition, subjects responded to target words in the narrative and ignored the tones. Event-related potentials (ERPs) were recorded for the tones, and difference waves (deviant ERPs minus standard ERPs) were computed. Two negative peaks in the difference waves, DN1 (100–180 ms) and DN2 (200–300 ms), overlapped the known scalp distribution and latency of the MMN. Mean DN1 and DN2 amplitudes were greater in the attend-tones condition than in the attend-words condition. These data suggest that the frequency-change MMN is modulated by nonspatial shifts of auditory attention.

Descriptors: Auditory event-related potential, Mismatch negativity, Selective attention

One component of the auditory event-related potential (ERP)—the mismatch negativity (MMN)—is thought to reflect the action of neural mechanisms that automatically detect physical changes in a stream of auditory stimulation (Näätänen, 1990). The MMN is experimentally elicited by sudden changes in the physical properties of repetitive, brief auditory stimuli. Early evidence for the MMN appeared in studies that manipulated the probabilities of stimuli differing in intensity, frequency, or duration (Ford, Roth, & Kopell, 1976; Snyder & Hillyard, 1976; Squires, Squires, & Hillyard, 1975). In these studies, the MMN appeared as an increase in the voltage in the latency range of the N2 component of the ERP elicited by low-probability stimuli. Later studies isolated the MMN from the N2 and plotted it as a component of the difference wave formed by subtracting the ERP for high-probability stimuli (or standards) from the ERP for the low-probability stimuli (or deviants) (Näätänen, Gaillard, & Mäntysalo, 1978, 1980; Sams, Alho, & Näätänen, 1983; Sams, Paavilainen, Alho, & Näätänen, 1985).

Source localization results show that the MMN arises from two distinct sources that are spatially separate but temporally overlapping (Scherg, Vajsar, & Picton, 1989). The early source (MMNa) corresponds closely to the N1 dipole source—a vertically oriented dipole on the supratemporal plane in the auditory cortex. This source apparently does not respond to small amounts (less than 10%) of frequency deviance, and its peak activity closely overlaps that of the N1 source in time. The later source (MMNb) corresponds to a more anteriorly located dipole, and it responds to decreasing amounts of frequency deviance by decreasing in amplitude and increasing in latency. The scalp voltage of this later source tends to be greater over frontal areas than at the vertex.

The MMN has a special functional significance in models of auditory information processing (Näätänen, 1990; Novak, Ritter, & Vaughan, 1992). In these models, auditory information is processed by two parallel and simultaneously active mechanisms. One mechanism is voluntarily controlled and maintains an attentional trace, such as a stimulus set, which facilitates processing of stimuli with attended properties and is associated with the processing negativity (PN) and an N2–P3 complex in the associated ERPs. A second mechanism operates automatically, without the benefit of attention, and is triggered by a mismatch between physically deviant stimuli among a series of standard stimuli. In Näätänen's (1990) model, this process is part of a feature detector system that passes information about the physical properties of a stimulus to a sensory memory store, where a neuronal trace is maintained and reinforced with repeated presentations of the same stimulus. Each new stimulus is compared

This work was supported in part by a grant to L.J.T. from the Office of Naval Technology, monitored by Dr. Stanley C. Collyer and Dr. Terry Allard, and in part by a grant to A.F.K. from the Office of Naval Research, monitored by Dr. Harold Hawkins. The opinions expressed here are those of the authors, are unofficial, and do not necessarily reflect the views of the Navy Department.

We gratefully acknowledge Richard Ogle, Todd Braver, and Leticia Novelo for help in the data collection and analyses and Dr. Walter Ritter and two anonymous reviewers for helpful comments.

Address reprint requests to: Leonard J. Trejo, Department of Psychology, University of Illinois, 603 E. Daniel St., Champaign, IL 61820, USA.

with the trace, and if a mismatch is detected, the MMN generator is triggered. The precise role of the MMN generator is to provide, at least for certain types or degrees of stimulus deviance, an interrupt or attention switching signal to higher, executive mechanisms. In this way, executive mechanisms can shift attention to novel stimuli, which could be important for the organism, even though they are not in the current attentional set. Novak et al. (1992) further proposed that the automatic process takes priority in determining the timing of later operations, such as reaction times, whereas the attentionally controlled process takes priority in determining response accuracy.

One controversial claim of Näätänen's model is that the MMN generator fully encodes information about stimulus features with or without attention. This claim is supported by experiments in which the MMN appears to be uninfluenced by the direction of attention. For example, deviant stimuli in the unattended ear during dichotic listening elicited MMNs that were comparable to those elicited in the attended ear (Näätänen et al., 1978, 1980). In another experiment, subjects were asked to ignore auditory stimuli and read a book (Sams et al., 1985). Under these conditions, the MMN was elicited by deviants that differed from standards in frequency by less than 2%.

The insensitivity of the MMN to attention was questioned by results of dichotic listening experiments that produced strong attentional demands by presenting stimuli at high rates (Woldorff, Hackley, & Hillyard, 1991). Under these conditions, stimuli that deviated from standards by an intensity decrement in the unattended ear produced a smaller MMN than that produced in the attended ear. This finding suggests that the MMN is either enhanced in attended channels or attenuated in unattended channels. The earlier experiments showing insensitivity of the MMN to attention may be explained in part by insufficient focusing of attention away from the MMN-eliciting stimuli to make a measurable difference. The results of Woldorff et al. (1991) further suggest that auditory information processing is influenced by attention before the elicitation of the MMN. However, these experiments did not completely rule out a contribution of the N2 component or target- and response-related processes to the attention effect on the MMN.

More recently, the effects of attention on the MMN were re-examined under recording conditions that reduced the likelihood of confounding the N2 with the MMN (Näätänen, Paavilainen, Tiitinen, Jiang, & Alho, 1993). As in the earlier experiments, this experiment also involved dichotic listening and high stimulus rates. Additionally, a second (nontarget) deviant stimulus, which did not require a response, allowed elicitation of the MMN without contamination by target- or response-related processing. Attentional modulation of MMN amplitude was confirmed for intensity-deviant stimuli. However, for frequency-deviant stimuli, no effect of attention on the MMN was observed. The authors concluded that it would be unparsimonious to infer that attention should modulate sensory inflow for the stimulus attribute of intensity but not frequency; therefore, attentional modulation of the MMN for intensity deviants could only reflect gain control on the output of the intensity-deviant MMN generator. This explanation leads to the equally unparsimonious assertion that attention controls the gain of the MMN generator process for intensity but not frequency deviations.

The preceding experiments raise three interesting questions about attentional modulation of the MMN. First, because subjects in the Woldorff et al. (1991) study responded to the attended deviant stimuli, it is possible that the differences in MMN

amplitude that they reported were augmented by a target effect on another part of the N2 component of the ERP measured in the attended ear. Second, in both studies, intensity decrements were difficult to detect, and their failure to elicit MMN could be at least partly related to their low intensity. Hit rates for the intensity-deviant stimuli were low (45–70%) when they were attended. Thus, a large fraction of the unattended deviants may have been indiscriminable from other stimuli or from the background. Third, attention to locations in space may operate differently from attention to other stimulus properties at a single location. The ability to attend to stimulus attributes, such as frequency, at a single location may require first learning to attend to the relevant dimension and then associating a response with the appropriate value of that dimension (Kahneman, 1973, p. 99). Thus, a test for attentional modulation using attention to attributes in a single location may be stronger and more general than that provided in a dichotic listening paradigm.

We sought to address these questions by devising an experiment in which subjects listened to a mixture of an interesting narrative and a series of MMN-eliciting tones in central auditory space. Large stimulus differences in frequency between standards and deviants were used, and intensities were balanced so that hit rates were reasonably high and all stimuli were clearly discriminable. Attention was manipulated by asking subjects either to respond to target words in the narrative and ignore the tones or to respond to a deviant tone and ignore the narrative. The potential for confounding attention effects on the MMN with possible target- or response-related effects on the N2 was reduced by using two deviant tones—one that required a response and another that did not. MMNs for the nontarget deviants should reflect only the effects of attention and not response-related processes. The results provide evidence for attentional modulation of the MMN elicited by frequency differences in central auditory space. When subjects attended to the tones, the MMN was more than twice as large as when subjects attended to the narrative.

Method

Subjects

Subjects (five men, five women) were volunteer staff and college students ranging in age between 18 and 43 years. Each subject completed and signed an informed consent and biographical/medical history form. Only subjects with normal hearing (self-report) and who passed a pretest for discrimination of the experimental stimuli were allowed to participate (two subjects failed the pretest). All subjects were native English speakers.

Tone Task

The tone stimuli were tapered sinusoids with a duration of 60 ms, cosine taper of 30% (18 ms rising and falling, 24 ms level) with equal intensities of 86 dB SPL, as measured with Bruel & Kjaer Model 2900 sound pressure level meter (B&K Corp., Los Angeles, CA). The tones were arranged in a pseudorandom sequence of standards (80%) and two deviants (10% each), with the restriction that the local probabilities over successive 20-trial blocks were constant and that at least two standards were interposed between any two deviants. Standard stimuli had a frequency of 1200 Hz. The high deviant frequency was 1400 Hz and the low deviant frequency was 1000 Hz. The interstimulus interval (ISI) was randomized, with a mean of 310 ms and a range

of 210–410 ms. Subjects were instructed to respond to one deviant tone by pressing a key on a response pad with their right forefinger.

Narrative Task

In the narrative task, subjects attended to a recorded narrative (a commercial seminar on memory improvement techniques) averaging 65 dB SPL and were instructed to listen to the narrative and respond whenever they heard the target word *and*. The response was to increment a mental count of the target words. The average rate of the target word presentation was 10 targets/min.

Procedures

The experiment was performed in two sessions: training and testing. During training there was a single condition: attend tones. During testing, there were two conditions: attend tones and attend words. In all conditions subjects were presented with a monophonic mixture of the tones and the narrative presented binaurally through calibrated headphones. In both conditions, data were collected in blocks containing 500 tones or about 2.6 min of the narrative. In the attend-tones condition, subjects were instructed to respond to one deviant tone and ignore the tape. In the attend-words condition, subjects were instructed to respond to the target words and ignore the tones. The intensities, proportions, and presentation rates of the tones were invariant across conditions.

Subjects were given 2,000 trials of practice in responding to one of the deviant tones. Half the subjects were trained with the high deviant tone, and the other half were trained with the low deviant tone. The first 1,000 practice trials served as a pretest to determine whether subjects could discriminate the stimuli. The second 1,000 practice trials were presented jointly with a background narrative (a recorded fiction novel, averaging 65 dB SPL)—which subjects were instructed to ignore—to train them to respond to tones in the presence of a narrative.

After the practice session, electroencephalogram (EEG) and electrooculogram (EOG) electrodes were applied to the subjects. The testing session began immediately afterwards. Testing proceeded in 16 500-tone blocks in which the condition alternated between attend tones and attend words every four blocks, beginning with attend words.

Task Performance

Final levels of performance in the attend-tones condition were assessed in a separate posttest condition. Unlike the testing condition, performance was measured for deviant tones in eight 500-trial blocks, and ERPs were not recorded. Each subject performed four blocks for each deviant tone (tones that had been targets and nontargets in the ERP session for that subject). The mean (*SD*) hit rate and mean (*SD*) reaction time for tones that had been target deviants were 86.9% (12%) and 436 ms (42 ms), respectively. For tones that had been nontarget deviants, the mean (*SD*) hit rate and reaction time were 79.9% (16%) and 473 ms (47 ms), respectively. Mean (*SD*) false alarm rates for tones that had been target and nontarget deviants were 5.7% (3%) and 3.7% (2%), respectively. Mean (*SD*) d' estimates for tones that had been target and nontarget deviants were 2.92 (0.85) and 2.87 (0.82), respectively.

Performance in the attend-words condition was assessed during the testing condition. About 25 target words occurred in each of the four blocks of the attend-words condition. Mean

(*SD*) hit rates and false alarm rates for word-counting performance across subjects were 91.5% (14%) and 4.1% (7.2%), respectively.

EEG Recording

The EEG from Fz, Cz, Pz (Jasper, 1958) and right mastoid (left mastoid reference) and the vertical and horizontal EOG were recorded while subjects performed the two test conditions. Signals were amplified and band-pass filtered (0.1–100 Hz) with a Grass Model 12 Neurodata Acquisition System then digitized continuously at 256 samples/s and stored by a computer using the Neuroscan system (Neuroscan, Inc., Herndon, VA). Off line, epochs of 1 s duration, including a 200-ms prestimulus baseline, were extracted, and the vertical and horizontal EOG records were then used to reduce EOG contamination (Semlitsch, Anderer, Schuster, & Presslich, 1986). In addition, any epoch containing residual artifact voltages exceeding 50 μ V at Fz, Cz, or Pz was rejected. Across subjects, the epoch rejection rate averaged 26%.

ERP averages for each stimulus and experimental condition were created separately for each subject, arithmetically rereferenced to average mastoids, digitally low-pass filtered (windowed finite impulse response filter), and adjusted for zero-median prestimulus baseline voltage. The low-pass filter cutoff frequency (-3 dB) was set to 12 Hz to smooth the ERPs enough to allow for unambiguous location of peaks. This filter was linear in phase and did not delay the frequency components of the signal in the passband. Difference waves were computed by subtracting (a) the average ERP for standards from the average ERP for target deviants (TG – STD) and (b) the average ERP for standards from the average ERP for nontarget deviants (NT – STD).

The dependent measure for ERP component analyses was the peak voltage in a fixed measurement interval for each selected component. We chose each measurement interval by graphically locating the peak and temporal extent of the presumed component in grand average ERP waveforms (Figure 1). Our interest was limited to the negative deflections that approximate latency and scalp distribution criteria corresponding to the MMN. Attention effects on positivities such as the P1, P2, and P300 will not be discussed in this paper. For the ERP averages, the selected components were the N1 (105–180 ms) and the N2 (200–300 ms). Confirmatory analyses of N1 and N2 amplitude effects using the mean voltage within the measurement interval were also performed.

Two broad negative peaks were evident in the grand average difference waves (Figure 2), which we labeled DN1 (100–180 ms) and DN2 (200–300 ms). Among the single-subject TG – STD difference waves, at Fz, 8 of the 10 subjects had a clear peak in the DN1 measurement interval for the attend-tones condition. Only three subjects showed a peak in the attend-words condition. For the NT – STD waves at Fz, six subjects had a defined peak for the attend-tones condition and five had a defined peak for the attend-words condition. Where a peak was not defined, the DN1 merged smoothly with the following DN2 or no deflection was present. Similar individual differences in the occurrence of peaks in the DN2 range were observed. Therefore, we analyzed the DN1 and DN2 using the mean voltage in the measurement interval as the dependent measure. DN1 and DN2 peak amplitude and latency effects will not be considered here.

Repeated-measures analyses of variance (ANOVAs) of the dependent measures for each component were performed using

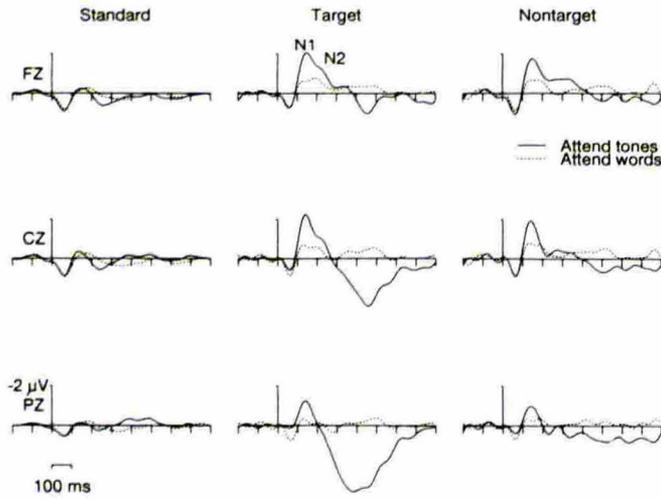


Figure 1. Grand average ERPs at Fz, Cz, and Pz for the standard (left), target (middle), and nontarget (right) tones in the attend-tones (solid lines) and attend-words (dashed lines) conditions. Standard and nontarget tones were not responded to in any condition. Target deviants were responded to only in the attend-tones condition. X-axis is time; scale bar is 100 ms. Time zero marks the stimulus onset. Y-axis is voltage; scale bar is 2 μ V; negative is up.

program 4V of the BMDP statistical package (Dixon, 1990). The significance level for all effects was .05, using degrees of freedom corrected for violations of the sphericity assumption where appropriate (Geisser & Greenhouse, 1958). Scalp distributions of components were compared by performing repeated-measures ANOVAs with electrode and component as factors and mean voltage in the measurement interval as the dependent measure. If the scalp distributions of the two components being compared

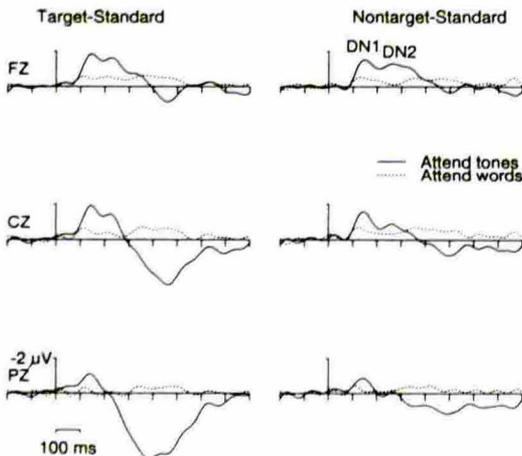


Figure 2. Grand average difference waves at Fz, Cz, and Pz. Target-standard difference waves (left) were computed by subtracting ERP averages for standard tones from those of the target deviant tones in the attend-tones (solid lines) and attend-words (dashed lines) conditions. Nontarget-standard difference waves (right) were computed by subtracting ERP averages for standard tones from those of the nontarget deviant tones in the attend-tones (solid lines) and attend-words (dashed lines) conditions. X-axis is time; scale bar is 100 ms. Time zero marks the stimulus onset. Y-axis is voltage; scale bar is 2 μ V; negative is up.

are different, then there should be a significant Electrode \times Component interaction. To avoid confounding an effect of field strength with true differences in the shapes of the scalp distributions, we applied a multivariate normalization to the mean voltages across electrodes (McCarthy & Wood, 1985). The values for Fz, Cz, and Pz were normalized by subtracting the mean and dividing by the root mean square value.

Results

Grand Average Waveforms

Standard tones. In both the attend-tones and attend-words conditions, grand average ERP waveforms for the standard tones (Figure 1, left) contained a triphasic frontocentral P1-N1-P2 complex. The N1 was small in amplitude, as expected with short ISIs (Nelson & Lassman, 1968). The peak of the N1 also appeared late, but the mean latency we observed (Cz, attend-words: 158.4 ms) is consistent with the reported N1 latency for ERPs elicited by binaurally presented unattended tones with short ISIs (138–160 ms; Hansen & Hillyard, 1980). Peak amplitudes of P1 and P2 appeared insensitive to the attentional manipulation, except for a possible P2 difference between the standard-stimulus ERPs. N1 and P2 appeared to show latency increases and amplitude decreases when attention was directed to the words versus the tones.

Target tones. Grand average ERPs for the target tones (Figure 1, center) contained a frontocentral P1-N1-N2 complex and a parietal P300 wave. The N1 and P300 components both clearly have lower amplitudes in the attend-words condition than in the attend-tones condition. The P300 appears to be completely absent in the attend-words condition. In the attend-tones condition, the N2 component appears as a shoulder on the descending limb of the N1 peak at Fz and Cz. In the attend-words condition, the N2 component appears as a peak following the N1, with a latency near 200 ms, and appears to be smaller than the N2 in the attend-tones condition.

Nontarget tones. Grand average ERPs for the nontarget deviant tones (Figure 1, right) contained a frontocentral P1-N1-N2 complex. The N2 component appears smaller than the N2 in the target ERPs in the attend-tones condition. Positivities around 225 ms and 500 ms at Pz suggest small P2 and P3 components in the attend-tones condition, but their amplitudes are questionably small. The ERP voltage for the attend-tones condition is greater than that for the attend-words condition between 100 and 400 ms at Fz and between 100 and 180 ms at Cz and Pz.

Difference waves. Grand average difference waves for TG – STD and NT – STD were computed as described above (Figure 2). These difference waves show two negative peaks between 100 and 300 ms at Fz and Cz, which we have labeled DN1 and DN2. Although we will not present an analysis of DN1 and DN2 peak latencies, we note that at Fz, DN1 had a mean latency of 143.4 ms across the single-subject NT – STD difference waves. The corresponding mean latency of the DN2 was 242.4 ms.

Average ERP Analyses

Three-way repeated-measures ANOVAs were performed on the mean amplitudes of the N1 and N2 components. The factors

Table 1. Significant Effects on ERP Components

Variable	<i>F</i>	<i>df</i>	ϵ^a
N1 amplitude			
Attention (A)	15.2**	1,9	
Stimulus (S)	15.7***	2,18	0.83
Electrode	21.0***	2,18	0.98
A × S	4.3*	2,18	0.82
A × E	9.6**	2,18	0.65
S × E	4.9*	4,36	0.62
N1 latency			
Attention	5.5*	1,9	
N2 amplitude			
Stimulus	13.9***	2,18	0.98
Electrode	12.6**	2,18	0.65
S × E	9.0***	4,36	0.64
A × S × E	3.8*	4,36	0.82
N2 latency			
Attention	6.5*	1,9	
A × E	7.1*	2,18	0.68

^aGeisser–Greenhouse ϵ used to adjust *df* for effects including stimulus or electrode.

* $p < .05$. ** $p < .01$. *** $p < .001$.

were attention (attend tones, attend words), electrode (Fz, Cz, Pz), and stimulus (standards, target deviants, nontarget deviants).

N1. For N1 amplitude (Tables 1 and 2), there were significant main effects of attention, stimulus, and electrode. There were also three significant two-way interactions: Attention × Stimulus, Attention × Electrode, and Stimulus × Electrode. Mean N1 amplitude was maximal for target stimuli at Cz in the attend-tones condition (Table 2). The greatest attention effect also occurred for this stimulus and electrode.

We examined the two-way interactions with a simple effects analysis of the effect of attention at pairs of stimuli and electrodes. No attention effect on N1 amplitude was significant for the standard tones at any electrode. For target tones, there were significant attention effects at Fz, $F(1,9) = 7.44$, $p < .0233$, Cz, $F(1,9) = 9.67$, $p < .0125$, and Pz, $F(1,9) = 5.89$, $p < .0381$. For

nontarget tones, significant attention effects occurred at Fz, $F(1,9) = 12.96$, $p < .0058$, and Cz, $F(1,9) = 9.84$, $p < .0120$.

For N1 latency (Table 1), there was a significant main effect of attention but no effect of stimulus or electrode nor any significant interactions. For most electrodes and stimuli, N1 latency was shorter in the attend-tones condition than in the attend-words condition, although the differences appear to be largest for the standard stimuli (Figure 1, Table 2).

Because of the attention effect of N1, we performed a confirmatory analysis using the mean voltage of the single-subject ERP waveforms in the N1 measurement interval as the dependent measure. As for the peak amplitude analysis, the main effect of attention was significant, $F(1,9) = 12.87$, $p < .0059$, as were the main effects of stimulus, $F(2,18) = 12.68$, $p < .0007$, $\epsilon = 0.87$, and electrode, $F(2,18) = 17.99$, $p < .0001$, $\epsilon = 0.88$. In addition, the Attention × Electrode interaction was still significant, $F(2,18) = 12.62$, $p < .0014$, $\epsilon = 0.76$, as was the Stimulus × Electrode interaction, $F(4,36) = 4.82$, $p < .0131$, $\epsilon = 0.62$. Unlike the peak amplitude measure, the Attention × Stimulus interaction was not significant for the mean voltage measure. The simple effects of attention for the deviant stimuli were replicated using the mean voltage measure of N1 amplitude including target tones at all electrodes (Fz: $F[1,9] = 7.15$, $p < .0255$; Cz: $F[1,9] = 15.11$, $p < .0037$; Pz: $F[1,9] = 6.07$, $p < .0360$) and nontarget tones at Fz, $F(1,9) = 7.83$, $p < .0208$, and Cz, $F(1,9) = 7.06$, $p < .0262$. Thus the confirmatory analysis shows that the effects of attention on N1 amplitude for the deviant stimuli are robust across measures.

N2. For N2 amplitude, there were significant main effects of stimulus and electrode and significant interactions of Stimulus × Electrode and Attention × Stimulus × Electrode (Table 1). Mean N2 amplitude was greatest for the target deviant stimuli, with a maximum at Fz followed closely by Cz (Table 3).

The interactions were examined with a simple effects analysis of the effect of attention at pairs of electrodes and stimuli. There were significant simple effects of attention on N2 amplitude for the standard stimuli at Fz, $F(1,9) = 45.80$, $p < .0001$, and Cz, $F(1,9) = 8.17$, $p < .0188$, but these effects are questionable, given that mean N2 amplitudes for the standard stimuli

Table 2. Mean (SE) Amplitudes and Latencies of the N1

Electrode	Attend tones		Attend words		Difference ^a	
	Amplitude (μ V)	Latency (ms)	Amplitude (μ V)	Latency (ms)	Amplitude (μ V)	Latency (ms)
Standards						
Fz	-0.3 (0.1)	149.6 (6.8)	-0.3 (0.6)	163.4 (4.6)	-0.0	-13.8
Cz	-0.4 (0.2)	137.9 (5.6)	-0.3 (0.1)	158.4 (5.8)	-0.1	-20.5
Pz	-0.3 (0.1)	144.9 (6.7)	-0.2 (0.1)	159.9 (5.9)	-0.0	-15.0
Targets						
Fz	-2.2 (0.4)	145.0 (3.9)	-0.9 (0.2)	146.9 (7.6)	-1.3*	-1.9
Cz	-2.4 (0.5)	143.0 (4.8)	-0.9 (0.2)	145.3 (7.1)	-1.5*	-2.3
Pz	-1.4 (0.3)	136.3 (5.4)	-0.5 (0.3)	147.6 (6.5)	-0.9*	-11.3
Nontargets						
Fz	-1.9 (0.4)	153.6 (5.4)	-1.0 (0.3)	150.5 (7.4)	0.9**	3.1
Cz	-2.0 (0.4)	140.4 (4.5)	-1.0 (0.3)	150.5 (7.2)	-1.0*	-10.1
Pz	-1.1 (0.3)	139.4 (6.2)	-0.6 (0.2)	153.9 (8.2)	-0.5	-14.5

^aDifferences were computed by subtracting values for the attend-words condition from values for the attend-tones condition.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 3. Mean (SE) Amplitudes and Latencies of the N2

Electrode	Attend tones		Attend words		Difference ^a	
	Amplitude (μ V)	Latency (ms)	Amplitude (μ V)	Latency (ms)	Amplitude (μ V)	Latency (ms)
Standards						
Fz	0.1 (0.1)	221.0 (12.2)	-0.2 (0.1)	201.8 (1.3)	-0.3***	19.2
Cz	0.1 (0.1)	257.7 (14.2)	-0.2 (0.1)	201.8 (1.3)	-0.3*	55.9**
Pz	-0.1 (0.0)	257.7 (13.7)	-0.2 (0.1)	209.6 (7.7)	-0.1	48.1**
Targets						
Fz	-1.7 (0.4)	230.5 (10.5)	-0.9 (0.2)	211.2 (6.9)	-0.9	19.3
Cz	-1.6 (0.3)	229.2 (11.4)	-0.7 (0.2)	206.1 (3.8)	-0.9	23.1
Pz	-0.3 (0.3)	209.6 (7.4)	-0.2 (0.1)	223.7 (9.5)	-0.2	-14.1
Nontargets						
Fz	-1.1 (0.3)	234.3 (14.2)	-0.8 (0.2)	225.0 (11.8)	-0.4	9.3
Cz	-0.9 (0.2)	258.5 (11.6)	-0.8 (0.2)	232.4 (13.2)	-0.2	26.1
Pz	-0.3 (0.2)	227.5 (12.0)	-0.4 (0.1)	243.6 (13.6)	0.1	-16.1

^aDifferences were computed by subtracting values for the attend-words condition from values for the attend-tones condition. * $p < .05$. ** $p < .01$. *** $p < .001$.

were not significantly different from zero at either Fz or Cz. At these electrodes, mean N2 amplitudes were negative in the attend-words condition and positive in the attend-tones condition (Table 3), which would suggest the unlikely conclusion that attention to the tones led to a polarity reversal of the N2. However, the grand average ERPs suggest that the N2 was not elicited by the standard stimuli. Thus, it is more likely that these simple effects reflect an attention effect on the P2 component, which was larger in the attend-tones condition than in the attend-words condition (Figure 1).

Focusing on the deviant stimuli, a simple effects analysis at pairs of electrodes and stimuli showed no significant N2 amplitude attention effect for either the target or nontarget tones. The effects of attention for target stimuli at Fz and Cz narrowly missed significance (Fz: $F(1,9) = 4.72, p < .0580$; Cz: $F(1,9) = 4.62, p < .0600$).

For N2 latency (Tables 1 and 3), the main effect of attention and the Attention \times Electrode interaction were significant. However, a simple effects analysis showed that these effects were confined to the standard-stimulus ERP averages. Significant attention effects on N2 latency occurred only for standard stimuli at Cz, $F(1,9) = 14.82, p < .0039$, and Pz, $F(1,9) = 12.50, p < .0064$. These results are questionable given the nonsignificant amplitude of N2 for standard stimuli and overlap of the P2 component (see above). No significant effects of attention on N2 latency for the deviant stimuli occurred at any electrode.

Given the ambiguity of N2 peak definition in some of the ERP averages, the significant N2 latency variation, and the absence of N2 from the standard-stimulus ERP averages, a confirmatory analysis was performed using the mean voltage in the N2 measurement interval as the dependent measure. This analysis focused on the deviant stimuli only and the Fz and Cz electrodes only, where the P300 component does not cancel the N2 negativity. Under these measurement conditions, there was a significant main effect of attention on N2 amplitude, $F(1,9) = 5.17, p < .0491$. No other effects or interactions were significant.

Difference Wave Analyses

Three-way repeated-measures ANOVAs were performed for baseline-to-peak amplitude and latency measures of the DN1

and DN2 components identified in the TG – STD and NT – STD difference waves (Figure 2). The variables were attention (attend tones, attend words), electrode (Fz, Cz, Pz), and stimulus (target, nontarget, i.e., first stimulus in the subtraction).

DN1. For DN1 amplitude, there were significant main effects of attention, $F(1,9) = 7.88, p < .0205$, and electrode, $F(2,18) = 10.86, p < .0008, \epsilon = 0.65$. Mean DN1 amplitude was greater at Cz and Fz than at Pz (Table 4). At Fz and Cz, for the TG – STD subtraction, DN1 was three times greater in the attend-tones condition than it was in the attend-words condition. For the NT – STD subtraction, the magnitude of the attention effect at Fz and Cz was over a factor of 2.

DN2. For DN2 amplitude, the main effect of attention narrowly missed significance, $F(1,9) = 5.06, p < .0511$. The only

Table 4. Mean (SE) Amplitudes (μ V) of the Difference-Wave Components

Electrode	Attend tones	Attend words	Difference ^a
DN1, TG-STD			
Fz	-1.5 (0.4)	-0.5 (0.2)	-1.0
Cz	-1.5 (0.4)	-0.5 (0.2)	-1.0
Pz	-0.9 (0.3)	-0.2 (0.2)	-0.7
DN1, NT-STD			
Fz	-1.2 (0.3)	-0.5 (0.2)	-0.7*
Cz	-1.3 (0.3)	-0.6 (0.2)	-0.7*
Pz	-0.7 (0.3)	-0.2 (0.1)	-0.5
DN2, TG-STD			
Fz	-1.3 (0.4)	-0.5 (0.1)	-0.8*
Cz	-0.9 (0.4)	-0.3 (0.1)	-0.6
Pz	0.7 (0.2)	0 (0.1)	0.6*
DN2, NT-STD			
Fz	-1.2 (0.3)	-0.2 (0.2)	-1.0**
Cz	-0.8 (0.2)	-0.4 (0.2)	-0.4
Pz	0 (0.2)	-0.2 (0.1)	0.2

^aDifferences were computed by subtracting values for the attend-words condition from values for the attend-tones condition. * $p < .05$. ** $p < .01$.

significant main effect was electrode, $F(2, 18) = 12.11, p < .0014, \epsilon = 0.67$. Unlike DN1, mean DN2 amplitude was greater at Fz than at Cz. There were two significant two-way interactions: Attention \times Electrode, $F(2, 18) = 11.78, p < .0018, \epsilon = 0.77$, and Stimulus \times Electrode, $F(2, 18) = 7.50, p < .0052, \epsilon = 0.94$. These interactions were examined by testing the simple effect of attention at pairs of stimuli and electrodes. The attention effect was significant for the TG – STD difference wave at Fz, $F(1, 9) = 5.82, p < .0391$, where mean DN2 amplitude was over two times more negative in the attend-tones condition than in the attend-words condition. The positive difference observed at Pz for the TG – STD stimulus probably arises from the P300 component (Figures 1 and 2) and also led to a significant attention effect, $F(1, 9) = 6.24, p < .0339$. The attention effect was also significant for the NT – STD difference wave at Fz, $F(1, 9) = 9.88, p < .0119$, where mean DN2 amplitude was six times more negative in the attend-tones condition than in the attend-words condition.

Scalp Topographies of Difference Negativities

So far, the results suggest that the MMN contributes to the DN1 or the DN2 components, or both, and that attention modulates the amplitude of the MMN. However, because of the considerable temporal overlap of the N1 with the DN1 and the N2 with the DN2 it is difficult to rule out contributions of N1- and N2-related effects to the apparent attentional modulation of the MMN. For example, the DN1 and DN2 attention effects could have been produced if there had been greater attention-related enhancements of N1 and N2 amplitudes for deviant stimuli than for the standard stimuli.

To rule out N1 as a mediator of the DN1 attention effect, we compared the scalp distributions of N1 and DN1. To rule out N2 as a mediator of the DN2 attention effect, we compared the scalp distributions of N2 and DN2. We structured these comparisons as two-way ANOVAs with electrode (Fz, Cz, Pz) and component (N1, DN1; N2, DN2) as factors.

N1 versus DN1. For N1, the mean voltage in the N1 measurement interval for the standard stimulus ERPs in the attend-tones condition was the dependent measure. For DN1, the mean voltage in the DN1 measurement interval for the NT – STD difference wave in the attend-tones condition was the dependent measure.

After normalization (see Method), both the N1 and the DN1 showed a maximal negativity at Cz (Figure 3). However, relative to the N1, normalized mean amplitudes of the DN1 were greater at Fz and Cz and lower at Pz, indicating a more frontal maximum in the DN1 scalp distribution. The main effect of electrode was significant, $F(2, 18) = 6.56, p < .0148, \epsilon = 0.75$, as was the Electrode \times Component interaction, $F(2, 18) = 4.37, p < .0417, \epsilon = 0.77$. This interaction suggests that the DN1 arises from a different, possibly more frontal source than does the N1 component.

N2 versus DN2. For N2, the mean voltage in the N2 measurement interval for the target deviant stimulus ERPs in the attend-tones condition was the dependent measure. For DN2, the mean voltage in the DN2 measurement interval for the NT – STD difference wave in the attend-tones condition was the dependent measure.

The normalized mean amplitude of the N2 was maximally negative at Fz, whereas the DN2 was maximal at Cz (Figure 3). In this respect, the normalized scalp distribution of the DN2

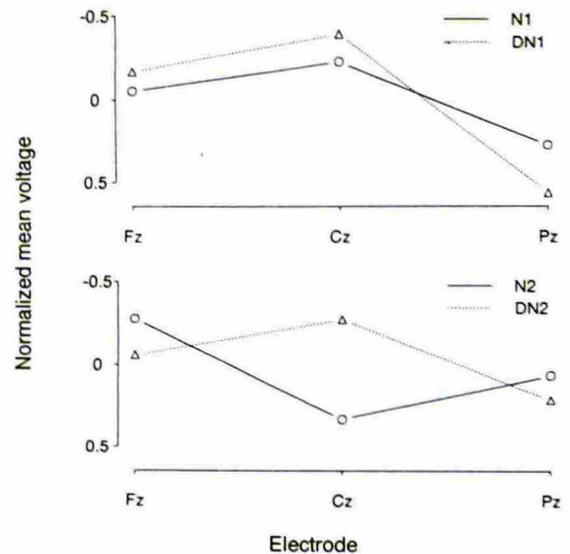


Figure 3. Electrode \times Component interaction plots for scalp distribution comparisons of N1 versus DN1 (upper) and N2 versus DN2 (lower). Circles/solid lines: ERP components. Triangles/dashed lines: difference-wave components. Mean voltages within the component measurement intervals were normalized.

resembled that of the N1 and the DN1, whereas the scalp distribution of the N2 did not. The main effect of electrode was significant, $F(2, 18) = 7.19, p < .0061, \epsilon = 0.94$, as was the Electrode \times Component interaction, $F(2, 18) = 8.84, p < .0041, \epsilon = 0.83$. The interaction suggests that the N2 arises from a different, possibly more frontal source than does the DN2 component.

Discussion

Attentional Modulation of the MMN

The most important results of this study are the attention-related differences in the amplitudes of the DN1 and DN2 deflections in the TG – STD and NT – STD difference waves. In these difference waves, when attention was directed toward the tones, mean voltages of the DN1 and DN2 were two or more times more negative than they were when attention was directed toward the narrative. Thus, the latency and scalp distribution properties of the DN1 correspond to the MMN component, and the DN2 may also receive a contribution from the MMN component. Accordingly, our data provide evidence for attentional modulation of the MMN for frequency differences among binurally presented tones.

DN1. The DN1 component falls within an interval that is known to contain an MMN elicited by frequency differences comparable to those we used. For example, using a 1000-Hz standard stimulus, May, Tiitinen, Reinikainen, and Näätänen (1992) found that MMN peak latency decreased from 200 ms with a frequency separation of 10 Hz to 120 ms with a frequency separation of about 100 Hz. With separations greater than 100 Hz, peak latency of the MMN remained nearly constant at 120 ms. The duration of the MMN leveled off near 115 ms for frequency separations greater than 40 Hz. Thus, the MMN for a frequency separation of 200 Hz should be found at 120 ± 55 ms. This latency range considerably overlaps the latency range of our DN1 component, 100–180 ms.

To conclude that the effect of attention on DN1 amplitude is an effect on the MMN, we must rule out other possible amplitude enhancements in the latency range of the DN1. Such effects include N1 enhancement (Hillyard, Hink, Schwent, & Picton, 1973; Näätänen & Picton, 1987) and PN (also called the negative difference wave or Nd; Hansen & Hillyard, 1980; Näätänen et al., 1978; Woods, 1990). For these effects to explain our DN1 attention effect, there must have been an interaction of the attention-related enhancement with stimulus type. If the same enhancement had occurred for standards and deviants, it would have cancelled in the subtraction and could not explain our DN1 effect.

One possible source of a Stimulus \times Attention effect interaction is the different rates at which the deviant and standard stimuli occurred. A DN1-like attention effect could have occurred if attention had produced more N1 enhancement or PN for the lower rate deviant stimuli than for the higher rate standard stimuli. This explanation seems unlikely for two reasons. First, Parasuraman (1980) found no effect of stimulus rate on the slow negative shift, a PN-like component of the auditory ERP elicited in selective or divided attention conditions. Stimulus rate was varied from two stimuli/s to four stimuli/s, which includes the rate of our standard stimuli (about 2.6/s). More recently, Teder, Alho, Reinikainen, and Näätänen (1993) found that the PN is reduced in amplitude only at stimulus rates substantially higher than our rate for standard stimuli (e.g., 6–12/s).

Another possible source of a Stimulus \times Attention effect interaction is that more N1 enhancement or PN could have occurred for the target stimuli because their sequence formed a channel to which attention was selectively directed in the attend-tones condition. This explanation also is unlikely because the target stimulus rate was too low (about 0.32/s) to create a well-defined channel upon which attention could be sustained. When relevant stimuli occur at low rates, the PN is greatly attenuated, presumably because the PN results from a matching process that requires an attentional trace whose strength depends on the stimulus rate (Alho, Lavikainen, Reinikainen, Sams, & Näätänen, 1990). A related possibility is that the two deviant stimuli formed a channel to which attention was directed in the attend-tones condition. This deviant-stimulus channel explanation is unlikely for the same reason—the combined deviant stimulus rate was too low to form a good channel (about 0.65/s). Moreover, this explanation is unlikely because the deviant stimulus frequencies (1000 and 1400 Hz) were physically and perceptually more different from each other than they were from the standard frequency (1200 Hz).

Yet another possibility is that the DN1 attention effect was produced by an interaction of stimulus-specific habituation of N1 amplitude with attention-related enhancement of N1 amplitude. Stimulus-specific habituation of the N1 component was described by Butler (1968) and is sometimes referred to as stimulus-specific *refractoriness* (e.g., Woldorff & Hillyard, 1991). For example, a DN1-like attention effect could have occurred if the attention-related N1 enhancement had been greater for less habituated stimuli (deviants) than for more habituated stimuli (standards). This would have produced greater N1 amplitudes for deviants in the attend-tones condition than in the attend-words condition, which did occur. Woldorff and Hillyard (1991) found some evidence of an Attention \times Stimulus-specific habituation interaction in a dichotic listening paradigm. However, the direction of this interaction for the negative difference in the latency range of the DN1 (N125/Nd125) was opposite of

what would be needed to explain our DN1 effect. That is, there was greater attention-related enhancement of the more habituated ERPs (such as our standard-stimulus ERPs) than of the less habituated ERPs (such as our deviant-stimulus ERPs). So it seems unlikely that this type of interaction could account for our DN1 effect.

Our scalp distribution comparisons provided additional support for the DN1 as MMN versus N1 enhancement. The Electrode \times Component interaction analysis showed that the N1 and DN1 scalp distributions were significantly different. The energy of the DN1 electric field was distributed more frontally than was that of the N1, which is consistent with source localization and magnetoencephalographic data (Scherg et al., 1989; Tiitinen et al., 1993). Together with the preceding arguments, this result leaves only MMN as an explanation of our DN1 attention effect.

DN2. Several studies have shown that the MMN latency may extend into the range of the DN2 for frequency separations comparable to those we used. For example, using 1000-Hz standards and a frequency separation of 150 ms, Näätänen et al. (1980) observed an MMN with a latency of about 175 ms. Scherg et al. (1989) found the MMN for a frequency separation of 100 Hz at a latency of about 190 ms. With a greater frequency separation (1000 Hz), the MMN appeared to consist of two peaks: an early peak near 96 ms and a later peak near 132 ms. Using 1000-Hz standards and 1500-Hz deviants, Böttcher-Gandor and Ullsperger (1992) found the MMN between 100 and 250 ms, with a mean peak latency near 200 ms. Magnetoencephalographic recordings of the magnetic equivalent of the MMN for a 100-Hz frequency separation with 1000-Hz standards placed the maximum difference between deviants and standards between 200 and 250 ms (Tiitinen et al., 1993). When considered with the MMN duration data, these latency data suggest that the energy of the MMN component can extend to 270 ms. Thus the MMN may extend into the range of our DN2 measurement interval, 200–300 ms. Thus, latency estimates indicate that the DN2 could have received a contribution from the MMN. Our observation that the distinction between DN1 and DN2 was not clear in all subjects also suggests that our DN1 and DN2 measures reflect a range of individual MMNs at varying latencies. It is also possible that our DN2 partly reflects the later MMNb source identified by Scherg et al. (1989).

The same arguments used against a PN-based explanation of our DN1 attention effect apply to our DN2 attention effect. The critical question about the DN2 is whether it can be wholly explained by an attention effect on the N2 component. This question is difficult to address using latency estimates because the N2 did not exhibit a well-defined peak in our ERP averages. Inspection of these averages suggests that the N2 was present only in the attended-deviant ERP averages but was slightly larger in the target-deviant ERPs than in the nontarget-deviant ERPs. Other work has also shown that the N2 is elicited by both target and nontarget deviant stimuli in attended channels (Ritter et al., 1992) but is absent in unattended channels. This finding is consistent with the significant effect of attention on our N2 mean voltage measure restricted to deviant tones and the Fz and Cz electrodes. Thus, we cannot rule out a contribution of N2 to the DN2 attention effect. However, we did find a difference between the normalized scalp distributions of N2 and DN2, as shown by the significant Electrode \times Component interaction. The normalized scalp distribution of the DN2 was fronto-central, with a maximum at Cz. This is the same general pat-

tern shown by the N1 and DN1. The N2, however, showed a maximum at Fz, which is consistent with the normalized N2 scalp distributions reported by Ritter et al. (1992). Thus, from the expected latency range and duration of MMN and these scalp distribution differences, it appears that a portion of the DN2 attention effect can be ascribed to the MMN.

Relationship to Other Studies

At least three other studies have recently reexamined the influence of attention on the MMN. Our results are consistent with those of Woldorff et al. (1991), who found attentional modulation of the intensity-change MMN in a dichotic listening paradigm. However, our results suggest that attentional modulation of the MMN is a more general phenomenon, which extends to binaural hearing and to frequency deviance. In addition, our DN1 and DN2 effects for the NT – STD difference waves were based on stimuli that did not require responses from our subjects. Thus, unlike the Woldorff et al. (1991) results, our DN1 and DN2 attention effects cannot be discounted by response-related or target-related processing. Our data also show that even when the attended channel is defined by a complex set of attributes, such as those that distinguish a narrative from a tone sequence, a substantial attention effect on the MMN occurs.

Our results are also consistent with those of another recent study, which also used binaural auditory stimuli and found a small enhancement of a frequency deviance-related negativity at Fz (probably the MMN) when attention was directed to auditory stimuli that were difficult to detect (Alho, Woods, Algazi, & Näätänen, 1992). In contrast, our results suggest that a large relative reduction in MMN (a factor of 2 or more) occurs when attention is directed away from the eliciting stimuli within the auditory modality and instead is directed to another channel of auditory information.

Our results differ in part from those of Näätänen et al. (1993), who observed attentional modulation of MMN for intensity deviants but not for frequency deviants in a dichotic listening paradigm that also included target and nontarget deviants. In reconciling their result with the aforementioned result of Alho et al. (1992), Näätänen et al. (1993) concluded that the frequency-change MMN is elicited even in the complete absence of attention, but under some conditions its amplitude can be attenuated. Our results show that sustained withdrawal of attention from the MMN-eliciting stimuli in central auditory space is one more condition under which the frequency-change MMN can be attenuated. In addition to the binaural/dichotic distinction between our study and that of Näätänen et al. (1993), there were several other methodological differences. First, their stimulus sequences contained a mixture of intensity-deviant and frequency-deviant stimuli, whereas ours contained only frequency-deviant stimuli. Other potentially significant differences between their experiment and ours included a lower amount of frequency deviance (factor of 4), lower deviant stimulus probabilities (factor of 5), and lower interstimulus interval (factor of 3). A reconciliation of our results with theirs must consider whether attentional modulation of the frequency-change MMN depends on the mixture of deviant stimulus types, the degree of frequency deviance, the deviant stimulus probability, and the deviant stimulus rate.

Theoretical Considerations

Taken together with the studies that have shown attentional modulation of the intensity-change MMN, our results ques-

tion the position that the MMN signals a mismatch detection based on preattentive, automatic, and complete processing of physical or sensory stimulus properties. Instead, the MMN appears to be influenced by the operation of an early attentional filter in a way similar to that of other components of the auditory ERP. The role of an attentionally modulated mismatch detector in Näätänen's (1990) auditory information processing model is unclear. If the MMN can be attenuated by attentional control, as suggested by the data of Woldorff et al. (1991), then the mismatch process it represents may not be an all-or-none phenomenon but instead may signal some likelihood of a mismatch given the current level of attention to the eliciting stimuli.

In his reply to an early version of the data presented by Woldorff et al. (1991), Näätänen (1990, p. 262) argued that as long as attention is not required for the MMN to occur, the MMN generator can still subserve fully automatic mismatch detection. According to this view, attentional modulation could be "due to selective sensitization of the MMN generator itself rather than to larger-quantity or higher-quality sensory information being stored in the attended channel."

More recently, Näätänen et al. (1993) clarified this view by arguing that attention might affect the gain of an amplification process that regulates the output of the MMN generator after the mismatch computation process is complete. In this view, the amplitude of MMN does not necessarily reflect the quantity or quality of sensory information that is involved in mismatch detection. However, this view was based on the assertion that (a) it would be unparsimonious to conclude that basic processing leading to initial stimulus representations differs for intensity and frequency and (b) that the frequency-change MMN is insensitive to attention. Since both assertions are contradicted by our results, we think it likely that the amplitude variation of MMN reflects variation in the certainty of mismatch detection. Such variation could partly arise from effects of attention on the quantity or quality of sensory information available to the MMN generator. This view is also consistent with the demonstration of attentional enhancement of ERP components, such as the P20-50, that precede the latency range of the MMN (Woldorff & Hillyard, 1991).

The precise influence of attention on the MMN generator is still unclear. An attentional filter appears to change inputs to the MMN generator, but either inhibition in the unattended channel or enhancement in the attended channel could be involved. Also, a combination of inhibition and enhancement may be involved. This view is consistent with models of auditory selective attention that include inhibition of unattended channels. For example, a model proposed by Hawkins and Presson (1977) to account for peripheral recognition masking effects has a mechanism that selectively inhibits unwanted acoustic information before this information gains access to an echoic sensory store. Selection of what to inhibit requires a frame of reference, which could be provided by a voluntarily maintained attentional trace such as that proposed in Näätänen's (1990) model. The data suggest that the MMN in the unattended channel is attenuated when the frame of reference for attentional control is well defined. In all the experiments that have shown attenuated MMNs for unattended channels, the frame of reference was continuous and natural for subjects to select. In lower rate dichotic listening or when using visual distractions such as reading, the frame of reference for auditory comparisons is not continually or clearly defined.

Attentional modulation of the MMN does not rule out a role for the MMN generator in detecting and comparing sensory differences, as required for neuronal-mismatch initiation of the orienting response (e.g., Sokolov, 1975) or for phenomena such as the breakthrough of the unattended. Instead, the effect of attention would be to render the output of the MMN generator more or less reliable for these purposes, depending on whether it operated on attended or unattended inputs. In any case, the evidence

shows that attentional filtering is a flexible process that influences the MMN generator and operates when attention is focused on spectral, intensity, and spatial stimulus attributes. Our data further suggest that this filter does not simply select the auditory modality or one location in auditory space; instead, this filter can select complex spectral patterns within a single spatial location.

REFERENCES

- Alho, K., Lavikainen, J., Reinikainen, K., Sams, M., & Näätänen, R. (1990). Event-related potentials in selective listening to frequent and rare stimuli. *Psychophysiology*, *27*, 73-86.
- Alho, K., Woods, D. L., Algazi, A., & Näätänen, R. (1992). Intermodal selective attention: II. Effects of attentional load on processing of auditory and visual stimuli in central space. *Electroencephalography and Clinical Neurophysiology*, *82*, 356-368.
- Böttcher-Gandor, C., & Ullsperger, P. (1992). Mismatch negativity in event-related potentials to auditory stimuli as a function of varying interstimulus interval. *Psychophysiology*, *29*, 546-550.
- Butler, R. A. (1968). Effect of changes in stimulus frequency and intensity on habituation of the human vertex potential. *Journal of the Acoustical Society of America*, *44*, 945-950.
- Dixon, W. J. (Ed.). (1990). *BMDP statistical software manual: To accompany the 1990 software release* (Vol. 2). Berkeley: University of California Press.
- Ford, J. M., Roth, W. T., & Kopell, B. S. (1976). Auditory evoked potentials to unpredictable shifts in pitch. *Psychophysiology*, *13*, 32-39.
- Geisser, S., & Greenhouse, S. W. (1958). An extension of Box's results on the use of the *F* distribution in multivariate analysis. *Annals of Mathematical Statistics*, *29*, 885-891.
- Hansen, J. C., & Hillyard, S. A. (1980). Endogenous brain potentials associated with selective auditory attention. *Electroencephalography and Clinical Neurophysiology*, *49*, 277-290.
- Hawkins, H. L., & Presson, J. C. (1977). Masking and preperceptual selectivity in auditory recognition. In S. Dorne (Ed.), *Attention and performance VI* (pp. 195-211). New York: Academic Press.
- Hillyard, S., Hink, R., Schwent, V., & Picton, T. (1973). Electrical signs of selective attention in the human brain. *Science*, *162*, 177-180.
- Jasper, H. (1958). The ten-twenty electrode system of the international federation. *Electroencephalography and Clinical Neurophysiology*, *43*, 397-403.
- Kahneman, D. (1973). *Attention and effort*. Englewood Cliffs, NJ: Prentice-Hall.
- May, P., Tiitinen, H., Reinikainen, K., & Näätänen, R. (1992, May). *The mismatch negativity (MMN) as a function of the magnitude of change in tone frequency*. Paper presented at the Tenth International Conference on Event-Related Potentials of the Brain, Eger, Hungary.
- McCarthy, G., & Wood, C. C. (1985). Scalp distributions of event-related potentials: An ambiguity associated with analysis of variance models. *Electroencephalography and Clinical Neurophysiology*, *62*, 203-208.
- Näätänen, R. (1990). The role of attention in auditory information processing as revealed by event-related potentials and other brain measures of cognitive function. *Behavioral and Brain Sciences*, *13*, 201-288.
- Näätänen, R., Gaillard, A. W. K., & Mäntysalo, S. (1978). Early selective-attention effect on evoked potential reinterpreted. *Acta Psychologica*, *42*, 313-329.
- Näätänen, R., Gaillard, A. W. K., & Mäntysalo, S. (1980). Brain potential correlates of voluntary and involuntary attention. *Progress in Brain Research*, *54*, 343-348.
- Näätänen, R., Paavilainen, P., Tiitinen, D., Jiang, D., & Alho, K. (1993). Attention and mismatch negativity. *Psychophysiology*, *30*, 436-450.
- Näätänen, R., & Picton, T. (1987). The N1 wave of the human electric and magnetic response to sound: A review and an analysis of the component structure. *Psychophysiology*, *24*, 375-425.
- Nelson, D. A., & Lassman, F. M. (1968). Effects of intersignal interval on the human auditory evoked response. *Journal of the Acoustical Society of America*, *44*, 1529-1532.
- Novak, G., Ritter, W., & Vaughan, H. G., Jr. (1992). The chronometry of attention-modulated processing and automatic mismatch detection. *Psychophysiology*, *29*, 412-430.
- Parasuraman, R. (1980). Effects of information processing demands on slow negative shift and N100 amplitude in selective and divided attention. *Biological Psychology*, *11*, 217-233.
- Ritter, W., Paavilainen, P., Lavikainen, J., Reinikainen, K., Alho, K., Sams, M., & Näätänen, R. (1992). Event-related potentials to repetition and change of auditory stimuli. *Electroencephalography and Clinical Neurophysiology*, *83*, 306-321.
- Sams, M., Alho, K., & Näätänen, R. (1983). Sequential effects on the ERP in discriminating two stimuli. *Biological Psychology*, *17*, 41-58.
- Sams, M., Paavilainen, P., Alho, K., & Näätänen, R. (1985). Auditory frequency discrimination and event-related potentials. *Electroencephalography and Clinical Neurophysiology*, *69*, 437-448.
- Scherg, M., Vajsar, J., & Picton, T. W. (1989). A source analysis of the late human auditory evoked potentials. *Journal of Cognitive Neuroscience*, *1*, 336-355.
- Semlitsch, H. V., Anderer, P., Schuster, P., & Presslich, O. (1986). A solution for reliable and valid reduction of ocular artifacts, applied to the P300 ERP. *Psychophysiology*, *23*, 695-703.
- Snyder, E., & Hillyard, S. A. (1976). Long-latency evoked potentials to irrelevant, deviant stimuli. *Behavioral Biology*, *16*, 319-331.
- Sokolov, E. N. (1975). The neuronal mechanisms of the orienting reflex. In E. N. Sokolov & O. S. Vinogradova (Eds.), *Neuronal mechanisms of the orienting reflex* (pp. 217-235). New York: Wiley.
- Squires, N. K., Squires, K. C., & Hillyard, S. A. (1975). Two varieties of long-latency positive waves evoked by unpredictable auditory stimulus in man. *Electroencephalography and Clinical Neurophysiology*, *38*, 387-401.
- Teder, W., Alho, K., Reinikainen, K., & Näätänen, R. (1993). Interstimulus interval and the selective attention effect on auditory ERPs: "N1 enhancement" versus processing negativity. *Psychophysiology*, *30*, 71-81.
- Tiitinen, H., Alho, K., Houtilainen, M., Ilmonemi, R. J., Simola, J., & Näätänen, R. (1993). Tonotopic auditory cortex and the magnetoencephalographic (MEG) equivalent of the mismatch negativity. *Psychophysiology*, *30*, 537-540.
- Woldorff, M. G., Hackley, S. A., & Hillyard, S. A. (1991). The effects of channel-selective attention on the mismatch negativity wave elicited by deviant tones. *Psychophysiology*, *28*, 30-42.
- Woldorff, M. G., & Hillyard, S. A. (1991). Modulation of early auditory processing during selective listening to rapidly presented tones. *Electroencephalography and Clinical Neurophysiology*, *79*, 170-191.
- Woods, D. L. (1990). The physiological basis of selective attention: Implications of event-related potential studies. In J. W. Rohrbaugh, R. Parasuraman, & R. Johnson, Jr. (Eds.), *Event-related brain potentials. Basic issues and applications* (pp. 178-209). New York: Oxford University Press.

(RECEIVED August 30, 1993; ACCEPTED August 3, 1994)

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.