Neural Mechanisms of Involuntary Attention to Acoustic Novelty and Change

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Abstract

■ Behavioral and event-related brain potential (ERP) measures were used to elucidate the neural mechanisms of involuntary engagement of attention by novelty and change in the acoustic environment. The behavioral measures consisted of the reaction time (RT) and performance accuracy (hit rate) in a forcedchoice visual RT task where subjects were to discriminate between odd and even numbers. Each visual stimulus was preceded by an irrelevant auditory stimulus, which was randomly either a "standard" tone (80%), a slightly, higher "deviant" tone (10%), or a natural, "novel" sound (10%). Novel sounds prolonged the RT to successive visual stimuli by 17 msec as compared with the RT to visual stimuli that followed standard tones. Deviant tones, in turn, decreased the hit rate but did not significantly affect the RT. In the ERPs to deviant tones, the mismatch negativity (MMN), peaking at 150 msec, and a second negativity, peaking at 400 msec, could be observed. Novel sounds elicited an enhanced N1, with a probable overlap by the MMN, and a large positive P3a response with two different subcomponents: an early centrally dominant P3a, peaking at 230 msec, and a late P3a, peaking at 315 msec with a right-frontal scalp maximum. The present results suggest the involvement of two different neural mechanisms in triggering involuntary attention to acoustic novelty and change: a transient-detector mechanism activated by novel sounds and reflected in the N1 and a stimulus-change detector mechanism activated by deviant tones and novel sounds and reflected in the MMN. The observed differential distracting effects by slightly deviant tones and widely deviant novel sounds support the notion of two separate mechanisms of involuntary attention.

INTRODUCTION

It is a common experience that, even during intensive task performance, our attention can be involuntarily engaged by acoustic changes occurring unexpectedly in the environment. The present study used a combination of behavioral and event-related brain potential (ERP) measures to elucidate the neural mechanisms involved in such involuntary attention. Irrelevant novel sounds and slight sound changes were used to cause involuntary attention to auditory stimuli in a forced-choice visual discrimination task. In this task, each visual stimulus was preceded by a task-irrelevant auditory stimulus that was a repetitive tone occasionally replaced by a slightly higher ("deviant") tone or by a natural, "novel" sound. It was predicted that these unexpected acoustic events would prolong the reaction time (RT) to visual stimuli and decrease the performance accuracy (hit rate) by engaging the subject's attention. ERPs elicited by the auditory-visual stimulus pairs were simultaneously recorded in order to reveal neural events underlying the expected behavioral effects.

Behavioral evidence of attention switching to irrelevant stimuli was first reported by Cherry (1953). His subjects noticed changes in the voice delivering an unattended speech message to one ear while they were attending to the speech message presented to the opposite ear. Further evidence was provided by RTs to probe stimuli in a secondary task (Dawson, 1990; Posner, 1978). These RTs were prolonged when target stimuli were preceded by unexpected, irrelevant changes in the repetitive stimuli delivered in the primary task, indicating, according to the authors, that processing resources were engaged by these stimulus changes occurring in the primary task (Dawson, Fillion, & Schell, 1989; Fillion, Dawson, Schell, & Hazlett, 1991; Siddle, 1991; Woodward, Brown, March, & Dawson, 1991).

Involuntary attention shifts were originally explained by the orienting-reflex (OR) theory (Sokolov, 1963), proposing that a neuronal model is built from the repetitive features of the external environment, inhibiting the OR to identical but not to different stimuli. Further, Öhman (1979, 1992) proposed that a "call" for reallocation of central processing resources is issued by preattentive mechanisms detecting significant changes in incoming stimuli.

Attention switching can also be elicited by stimulus onsets (regardless of repetition/change), in particular if they appear after long "silent" intervals, and by offsets of continuous stimulation (Folk, Remington, & Johnston, 1992; Gati & Ben-Shakhar, 1990; Hikosaka, Miyauchi, & Shimojo, 1993; Öhman, 1979; Theeuwes, 1994; Yantis, 1993: Yantis & Hillstrom, 1994). According to Näätänen's (1990, 1992) model, stimulus onsets and offsets activate a transient-detector system reflected, in the auditory modality, in the supratemporal and nonspecific components of the N1 wave, peaking at about 100 msec from stimulus onset (Näätänen & Picton, 1987). The supratemporal N1 component is generated by bilateral dipoles located in the auditory cortices on the supratemporal plane (Giard et al., 1994; Vaughan & Ritter, 1970), and its amplitude increases with an increasing interval from the previous sound (Davis, Mast, Yoshie, & Zerlin, 1966; Hari, Kaila, Katila, Tuomisto, & Varpula, 1982; Mäkelä, Hari, & Leinonen, 1988; Ritter, Vaughan, & Costa, 1968). This component is sensitive in particular to the transient aspects of stimulation (Davis & Zerlin, 1966; McMillan, 1973; Pfefferbaum, Buchsbaum, & Gips, 1971), which suggests that the neuronal process generating the supratemporal N1 triggers an attention-capturing signal for conscious perception of the stimulus (Näätänen, 1990, 1992). The nonspecific N1 component is even more sensitive to interstimulus interval manipulations than the supratemporal component (Näätänen & Picton, 1987), and, in contrast to the supratemporal N1 component, it can be elicited also by stimuli of other modalities (Lehtonen, 1973; Velasco & Velasco, 1986; Velasco, Velasco, & Olvera, 1985). The function of the nonspecific N1 neural generators probably is to trigger a transient arousal burst, facilitating sensory and motor responses to the eliciting stimulus (Näätänen & Picton, 1987). However, Giard et al. (1994) described quite recently a frontal subcomponent of the N1 wave. This subcomponent might be a better candidate for the attention-switching function originally attributed to the supratemporal-N1 generator.

The stimulus-change detector mechanism postulated by the OR theory is represented in Näätänen's (1990, 1992) model as the neuronal process generating the mismatch negativity (MMN). The MMN, elicited by any discernible change in the physical features of a repetitive, even unattended, sound (e.g., Näätänen, 1992), is generated mainly in the supratemporal auditory cortex (for an overview, see Alho, 1995), as revealed by ERPsource modeling (Giard et al., 1995; Giard, Perrin, Pernier, & Bouchet, 1990; Scherg, Vaisar, & Picton, 1989), neuromagnetic data (Hari et al., 1984), and intracranial recordings in animals (Csépe, Karmos, & Molnár, 1987; Javitt, Schroeder, Steinschneider, Arezzo, & Vaughan, 1992) as well as in humans (Halgren, Baudena, Clarke, Heit, et al., 1995; Kropotov et al., 1995). According to Näätänen's (1990, 1992) model, the physical features of auditory stimuli are fully analyzed and encoded into neural traces of auditory sensory memory, the MMN being automatically elicited when the auditory input does not match with the neuronal trace formed by the repetitive standard stimulus (Näätänen, Paavilainen, Alho, Reinikainen, & Sams, 1989) while the trace still is in an "active" state (Cowan, Winkler, Teder, & Näätänen, 1993). Further, the model proposes that the MMN-generating process is involved in triggering a signal for attention switching after the automatic, preperceptual detection of auditory change (Näätänen, 1990).

Converging evidence supports the notion that the process generating the MMN may be associated with involuntary attention switching. First, the MMN is of preattentive nature because it does not depend on whether the subject is engaged in an easy or a difficult visual task (Alho, Woods, Algazi, & Näätänen, 1992; Duncan & Kaye, 1987).¹ Second, in addition to its supratemporal generators, MMN appears to get a further contribution from the frontal lobe activity (Giard et al., 1990; Molnár, Skinner, Csépe, Winkler, & Karmos, 1995), a critical structure for controlling both voluntary and involuntary attention (Fuster, 1989). Third, the MMN tends to be accompanied, especially in the beginning of the stimulus sequence, by autonomous nervous system (ANS) responses even when stimulus change is of very small magnitude (Lyytinen, Blomberg, & Näätänen, 1992). These autonomic responses indicate, apparently, attention switching triggered by the automatic change-detection process reflected by the MMN.

The strongest evidence supporting the causal relationship between the neural process generating the MMN and involuntary attention switching was recently obtained by Schröger (1996). He found that MMN-eliciting deviant tones occurring in a repetitive sequence of standard tones presented to the unattended ear prolonged the RT and decreased the performance accuracy to subsequent target tones delivered to the other ear. This effect was observed only for irrelevant tones preceding target stimuli by a short interval of 200 msec, but not for longer intervals of 560 msec, and was strengthened with increasing frequency difference between the deviant and standard tones. Both of these findings support the functional role of the MMN-generating process in triggering this attention switch.

Another ERP component that has been associated

with orienting of attention is the P3a. This positive component is elicited by irrelevant rare tones and novel sounds in a sequence of repetitive, standard tones and can be distinguished from the P300 or P3b component to target stimuli by its shorter latency and its more frontal scalp distribution (Courchesne, Hillvard, & Galambos, 1975; Ford, Roth, & Kopell, 1976; Squires, Squires, & Hillyard, 1975). The association of the P3a-generating process with the orienting response is supported by its elicitation by widely deviant or novel sounds and also by the frontal-lobe and hippocampal contributions to its generation (Knight, 1984, 1996), these brain regions being involved also in the orienting response (Fuster, 1989; Sokolov, 1975). The role of the P3a generator process in involuntary engagement of attention is further supported by studies showing delayed RTs to target stimuli following irrelevant novel sounds (Grillon, Courchesne, Ameli, Gever, & Braff, 1990; Woods, 1992). When novel sounds occurred in the attended sequences containing the target stimuli, the P3a elicited was larger in amplitude as compared with the unattended-sequence P3a to novel sounds, and the RT to consecutive targets was delayed (Woods, 1992). This attentional modulation suggests that the P3a reflects an actual reorientation of attention more closely than the MMN or the different subcomponents of the N1, which would rather be associated with the involuntary call for attention.

The purpose of the present experiment was to investigate the neural mechanisms of involuntary attention to acoustic novelty and change by analyzing the ERP concomitants elicited by these irrelevant auditory events during visual performance. In the main experimental condition (Figure 1), the Auditory-Visual condition (AV), a response was required to each visual stimulus of an auditory-visual stimulus pair. Subjects were instructed to ignore the auditory stimuli, which were a repetitive, "standard" tone randomly replaced by a slightly higher "deviant" tone (p = 0.1) or by a natural, "novel" sound (p = 0.1) and to press a response button to even numbers and another to odd numbers. Two additional conditions were used as controls: In the Visual-alone condition (Va), the auditory stimuli were omitted, and subjects performed the forced-choice visual RT task. In the Auditory-alone (Aa) condition, the visual stimuli were omitted, and subjects were instructed to concentrate on reading a book.

RESULTS

Performance

In the Va condition, the mean hit rate was 91.5% (SEM \pm 1.7%) and the mean RT was 461 \pm 11 msec. Subjects missed the target on an average 4.5% \pm 1.6% of the trials and pressed the wrong button in 4.0% \pm 0.8% of the trials.

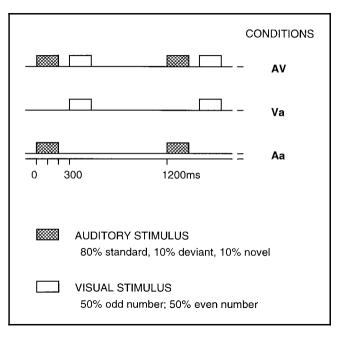


Figure 1. Experimental conditions. In the main condition [Auditory-Visual (AV), top], pairs of stimuli, consisting of an auditory stimulus followed at 300 msec (onset-to-onset) by a visual stimulus (an arabic number between 1 and 8), were presented with an interpair interval of 1200 msec. The auditory stimulus was either a standard tone (80%), a deviant tone (10%), or a novel sound (10%). Subjects were instructed to press one response button to odd (50%) and the other to even (50%) numbers and to ignore auditory stimulation. In the Visual-alone (Va) condition, the auditory stimuli were omitted. In the Auditory-alone (Aa) condition, the visual stimuli were omitted, the subject being instructed to read a self-selected book and to ignore the auditory stimuli.

Performance data in the Va and AV conditions are shown in Figure 2. In the AV condition, subjects tended to respond faster to visual stimuli preceded by a standard tone than to those preceded by no sound (Va condition) $(444 \pm 9 \text{ and } 461 \pm 11 \text{ msec}, \text{ respectively})$, although this effect did not quite reach statistical significance (F(1,9) = 3.8, p < 0.09). The hit, error, and miss rates were similar in the Va condition and in the AV condition after a standard tone. In the AV condition, the occurrence of a deviant tone before the visual stimulus caused a significant hit rate decrease of 2.3% (F(1, 9) = 6.50, p <0.04), which was probably due to an increased number of wrong responses to target stimuli (F(1, 9) = 4.78, p < 1000.06; see Figure 2), the number of missing responses being identical after standard and deviant tones. A novel sound occurring before a visual stimulus caused an average increase of 17 msec in the RT to visual stimuli compared with the RT to these stimuli that were preceded by standard tones (F(1, 9) = 22.19, p < 0.002). The RT to visual stimuli after novel sounds was also significantly longer than that to visual stimuli following deviant tones (F(1,9) = 11.07, p < 0.009).

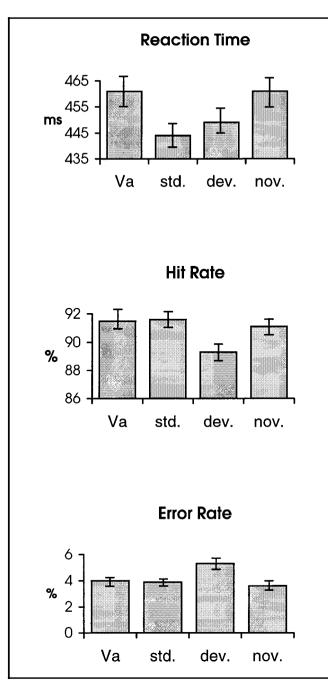


Figure 2. Mean RT (top panel), hit rate (middle panel), and error rate (bottom panel) in the Visual-alone (Va) condition and in the Auditory-Visual (AV) condition to the visual stimulus occurring after the standard tone (std.), the deviant tone (dev.), or the novel sound (nov.). The bars indicate the standard error of mean.

Auditory ERPs

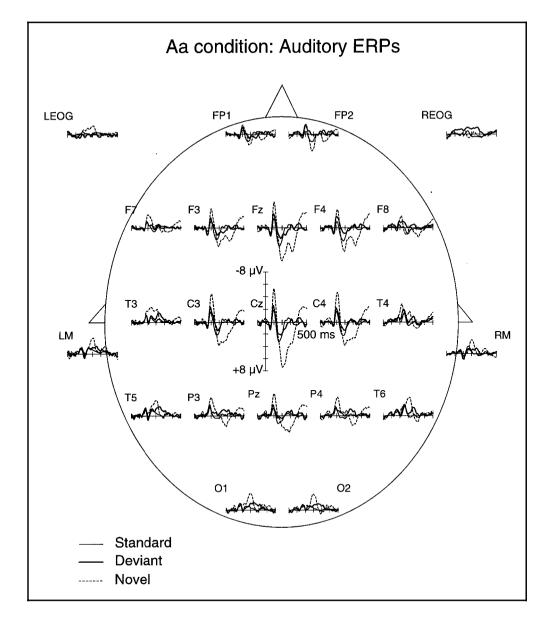
In the Aa condition, standard tones elicited N1 (mean peak amplitude $-2.0 \ \mu\text{V}$ at Cz, with a mean peak latency of 93 msec) and P2 deflections (3.4 μ V, 158 msec; at Cz), which were largest over the fronto-central scalp locations (Figure 3a). Deviant tones elicited N1 and P2 waves at latencies similar to those in response to standard tones and the mismatch negativity (MMN). The MMN

was largest over the frontal electrodes (mean peak amplitude $-2.2 \ \mu$ V, mean peak latency 153 msec at Fz) and inverted in polarity at the mastoid electrodes (Figure 3b), being significantly different from zero at F3, Fz, and F4 in the intervals 100 to 150 msec (F(1, 9) = 10.31, p < 0.02) and 150 to 200 msec (F(1, 9) = 5.63, p < 0.05). The MMN was followed by a smaller, fronto-centrally distributed negative wave, peaking at around 400 msec (Figure 3b). Mean amplitude comparisons between ERPs to deviant and standard tones at Fz and Cz revealed that this second negativity was significant in the 350- to 400-msec (F(1, 9) = 7.56, p < 0.02) intervals. In the AV condition, the MMN was similar to that obtained in the Aa condition (Figure 4).

In the Aa condition, novel sounds elicited an ERP characterized by a prominent N1 deflection (mean peak amplitude at Cz -5.6μ V; peak latency 96 msec) and by a long-duration, broadly distributed P3a wave (Figure 3; as seen in Figure 3b, even the deviant tones elicited some P3a). The novel-sound N1 was larger than the standard-tone N1 (*F*(1, 9) = 10.99, *p* < 0.01) and was probably composed of overlapping N1 and MMN components. The difference between the standard-tone and novel-sound ERPs extended beyond the N1 latency range, there being a significant difference at Fz between the standard and novel ERP amplitudes measured as mean voltages over 120 to 160 msec latency window (*F*(1, 9) = 5.23, *p* < 0.05; Figure 5).

The present P3a to novel sounds had a double peak over the frontal scalp locations (Figure 3b), suggesting two different subcomponents. The early part of the P3a (peak latency 230 msec in the grand-mean ERP at Fz) was largest over the central scalp areas, appearing with inverted polarity at lateral and posterior scalp sites (Figures 3b and 5). The late part of the P3a (peak latency 315 msec in the grand-mean ERP at Fz) in turn was largest over frontal areas and did not show polarity inversion at any recording site (Figures 3b and 5). The different scalp distributions of the two P3a subcomponents in the Aa condition are shown in Figure 6 (top).

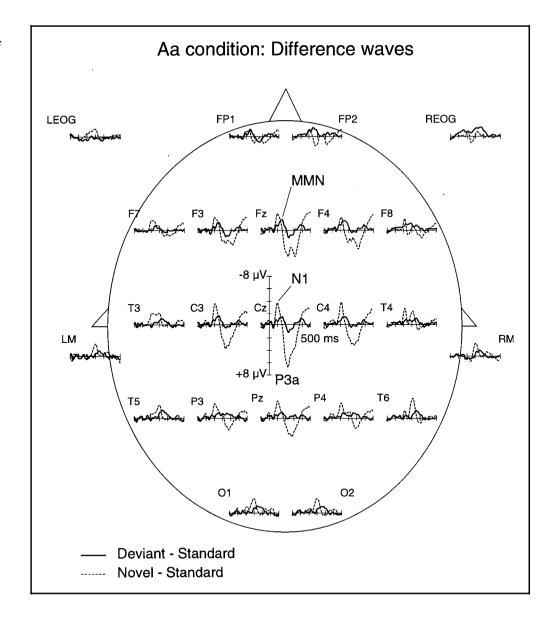
The component structure of the P3a deflection was evaluated by means of two different analyses of variance (ANOVA) for normalized ERP amplitudes (McCarthy & Wood, 1985). First, an ANOVA was performed to prove a possible scalp-distribution difference between the P2 to standard tones and the early P3a to novel sounds. Then, another ANOVA was performed to confirm the different scalp distributions of the two subcomponents of the P3a. The P2 amplitude was identified as the mean voltage between 150 and 200 msec from stimulus onset in the standard-tone ERP. The early P3a was measured as the mean voltage between 175 and 275 msec in the difference wave obtained by subtracting the ERP to the standard tone from that to the novel sound. The late P3a was measured as the mean voltage between 275 and 375 msec of the same difference wave. A three-factor design **Figure 3a.** Grand-mean ERPs averaged across subjects to standard tones (600 Hz), deviant tones (700 Hz), and novel sounds in the Auditory-alone (Aa) condition.



was used for both analyses: Wave (P2 versus early P3a for the first analysis and early P3a versus late P3a for the second analysis) × Frontality (F7, F3, Fz, F4, F8 versus T3, C3, Cz, C4, T4 versus T5, P3, Pz, P4, T6) × Laterality (F7, T3, T5 versus F3, C3, P3 versus Fz, Cz, Pz versus F4, C4, P4 versus F8, T4, T6). The results of the first analysis revealed a significant Wave \times Frontality interaction (F(2, 18) = 5.09, p < 0.03, $\varepsilon = 0.7547$), due to the early P3a being posterior to the P2, and a significant Wave \times Laterality interaction (F(4, 36) = 4.06, p < 0.03, $\varepsilon =$ 0.5469), due to the more predominantly right-hemispheric scalp distribution of the P2 than that of the early P3a. The results of the second analysis revealed that the late P3a was anterior in distribution to the early P3a (significant Wave × Frontality interaction, F(2, 18) = 9.39, p < 0.006, $\varepsilon = 0.7187$) and, further, that the late P3a was more preponderant over the right hemisphere than the

early P3a (significant Wave × Laterality interaction, F(4, 36) = 12.07, p < 0.0002, $\varepsilon = 0.5740$). The second analysis also revealed a significant third-order interaction (Wave × Frontality × Laterality, F(8, 72) = 8.98, p < 0.0004, $\varepsilon = 0.3441$) that was due to the predominantly right-frontal distribution of the late phase of the P3a deflection (Figure 6, top).

In the AV condition, novel sounds elicited ERPs similar to those elicited by them in the Aa condition (large N1 and P3a deflections), although the early peak of the P3a seemed to be partly abolished at frontal electrodes (Figure 7). However, difference waves (ERP to novel sounds minus ERP to standard tones) revealed that the early phase of the P3a was similar in the Aa and AV conditions, whereas the late P3a was enhanced in the AV condition (Figure 7). The statistical scalp-distribution analyses of the two P3a phases across the conditions [ANOVA, with Figure 3b. Grand-average difference waves obtained in the Aa condition by subtracting the ERP to standard tones from that to deviant tones (continuous line) and to novel sounds (dashed line).



four factors: Condition (Aa versus AV) × Wave (early P3a versus late P3a) × Frontality × Laterality] confirmed the scalp-distribution difference between the two P3a phases (Wave × Frontality interaction, F(2, 18) = 9.96, p < 0.003, $\varepsilon = 0.8361$; Wave × Laterality, F(4, 36) = 6.88, p < 0.02, $\varepsilon = 0.3977$; Wave × Frontality × Laterality, F(8,72) = 9.81, p < 0.0001, x = 0.4381) and also revealed a significant increase of the late P3a amplitude over the right hemisphere in the AV condition (Wave × Condition × Laterality, F(4, 36) = 3.84, p < 0.04, $\varepsilon = 0.5367$).

Visual ERPs

Visual stimuli in the Va condition and those after standard tones in the AV condition elicited almost identical ERPs characterized by P1, N1, P2, N2, and P3 deflections (Figure 8). ANOVA for these visual ERP components with Condition (after standard in AV versus Va) as a function revealed no significant effects of standard tones on visual ERPs.

Figure 9 shows grand-average ERPs to auditory-visual stimulus pairs in the AV condition. The AV stimulus pairs elicited an auditory ERP, lasting up to 400 msec post-stimulus for novel sounds and up to 300 msec post-stimulus for deviant tones, followed by a visual ERP similar to that obtained in the Va condition. All three types of AV stimulus pairs (standard-visual, deviant-visual, novel-visual) were also characterized by a long-lasting fronto-polar negativity that was larger to visual stimuli following novel sounds than to those following standard or deviant tones (Figure 9). ANOVA for mean amplitudes at Fp1 and Fp2 in consecutive 100-msec latency windows yielded significant effects for the novel-standard comparison (F(1, 9), ranging from 7.79 to 11.96; p < 0.03

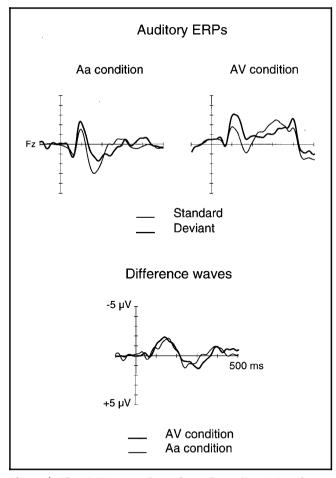


Figure 4. The MMN was similar in the Auditory-alone (Aa) and Auditory-Visual (AV) conditions, as revealed by nonsignificant differences of the mean amplitude of five consecutive 50-msec intervals starting at MMN onset (50 msec) [two-way ANOVAs: Condition (Aa versus AV) \times Electrode (F3, Fz, F4, C3, Cz, C4)]. Top: Grand-average ERPs to standard and deviant tones at Fz in the two conditions. Bottom: Deviant-standard difference waves.

in all cases) at latency windows 200 to 300 msec, 300 to 400 msec, and 400 to 500 msec from visual-stimulus onset.

Novel sounds and deviant tones enhanced the frontal N2 deflection elicited by the subsequent visual stimulus in comparison with the visual N2 elicited after standard tones (Figure 9). This was revealed by two-way ANOVAs using the Electrode (F3, Fz, F4) and the auditory Stimulus (standard versus either deviant or novel) as factors. The mean amplitude in the 200- to 300-msec range from visual-stimulus onset at Fz was $-1.9 \ \mu$ V after standard tones, $-3.3 \ \mu$ V after deviant tones (F(1, 9) = 6.41, p < 0.04), and $-3.6 \ \mu$ V after novel sounds (F(1, 9) = 8.99, p < 0.02). The mean amplitude in the 300- to 400-msec interval was also significantly larger for visual stimuli following deviant tones: F(1, 9) = 8.15, p < 0.02 for

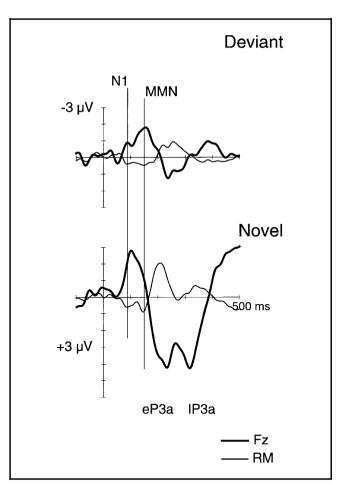
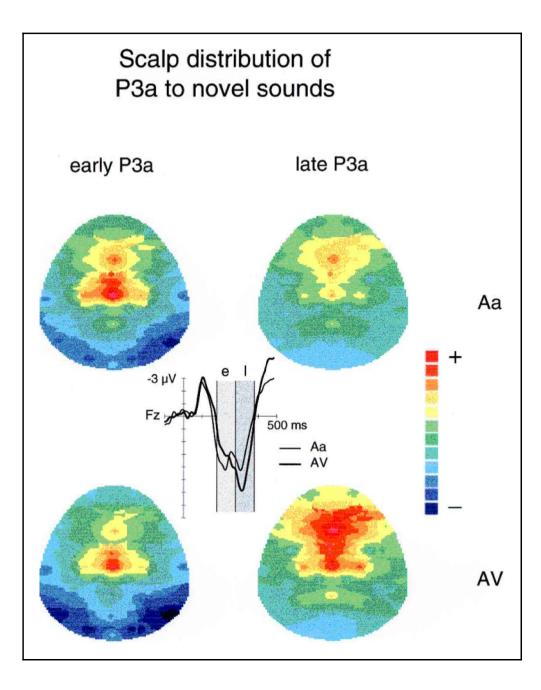


Figure 5. The component structure of the ERPs to deviant tones and novel sounds, as revealed by the deviant-standard and novel-standard difference waves at Fz and right-mastoid (RM) recordings. Deviant tones elicited the MMN with a peak latency of 150 msec at Fz and with an inverted polarity at RM (top). Novel sounds elicited a large N1 deflection at Fz that was presumably composed of contributions from two different processes, for which the significant differences between the standard-tone and novel-sound ERPs extended beyond the N1 latency range (bottom). The novel sounds also elicited a P3a wave with two peaks at Fz, suggesting two different subcomponents contributing to the recorded P3a: an early P3a (peak latency 230 msec), which inverted polarity at RM and other posterior and lateral sites, and a late P3a (peak latency, 315 msec), which inverted polarity at no recording site.

deviant versus standard; F(1, 9) = 7.95, p < 0.01 for novel versus standard.

Preceding novel sounds and deviant tones also enhanced the terminal slope of the visual P3b (Figure 9); this was revealed by the significantly larger mean amplitude of the 500- to 600-msec latency window (at P3, Pz, and P4) in the ERP to visual stimuli following deviant tones than in the ERP to visual stimuli following standard tones (F(1,9) = 15.56, p < 0.04). The mean amplitude in the 400- to 500-msec (F(1,9) = 6.94, p < 0.03) and 500-to 600-msec (F(1,9) = 11.99, p < 0.008) intervals was

Figure 6. The scalp distribution of the P3a to novel sounds. Left: early P3a; right: late P3a; top: Auditory-alone (Aa) condition; bottom: Auditory-Visual (AV) condition. The central plot shows the novelstandard difference waves at Fz in the Aa (thin) and AV (thick) conditions, as well as the 100-msec latency windows used to measure the two phases of the P3a (e = early P3a; 1 = late P3a).



also significantly larger for visual stimuli following novel sounds than after those following the standard tones.

DISCUSSION

Engagement of Attention During Visual Performance

The present results provide evidence that attention during visual performance can be engaged by shortly preceding unexpected novel sounds or small changes in the task-irrelevant acoustic environment. These results also revealed that this phenomenon depends on the intrinsic nature of the eliciting acoustic events. In addition, the observed effects on visual performance were associated with different patterns of neural events activated by deviant tones and novel sounds, as reflected by the component structure of the auditory ERPs elicited.

As expected, novel sounds prolonged the RT to successive visual stimuli. This result is in agreement with the extensive literature reporting delayed RTs to target stimuli caused by preceding irrelevant sound changes or novels sounds (Alho et al., 1992; Cherry, 1953; Dawson et al., 1989; Fillion et al., 1991; Grillon et al., 1990; Schröger, 1996; Woods, 1992; Woodward et al., 1991), and it provides experimental support for the everyday experience that attention can be engaged by irrelevant acoustic events.

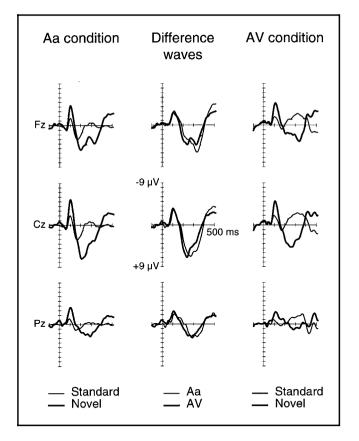


Figure 7. Grand-average ERPs to standard tones and novel sounds in the Auditory-alone (Aa, left column) and Auditory-Visual (AV, right column) conditions. The novel-standard difference waves are shown in the middle column. The late P3a was larger in the AV condition than in the Aa condition, whereas the early P3a was of similar amplitude in the two conditions.

A surprising finding of this study was, however, that the slightly deviant tones degraded the visual performance accuracy by decreasing the rate of hit responses, whereas the widely deviant novel sounds did not. It was expected that the amount of distraction caused by deviant tones and novel sounds would be proportional to the magnitude of their deviance from the standard tone, and indeed such an effect was found for the RT (17-msec increase after novel sounds), confirming other recent findings (Jääskeläinen et al., 1996; Schröger, 1996). However, this RT effect was not paralleled by a similar effect on the hit rate. Indeed, deviant tones were associated with a lower hit rate, whereas the hit rate after novel sounds was similar to that after standard tones. Similar results were obtained in a related study by Jääskeläinen et al. (1996) and recently replicated for deviant tones (Alho, Escera, Díaz, Yago, & Serra, 1997). These findings suggest that the distracting effects of an irrelevant acoustic event on visual performance may depend on the nature of the eliciting sound. That is, obtrusive novel sounds appear to cause a stronger effect on the RT to successive visual target stimuli than minor changes in the acoustic stream, but in contrast, these minor changes

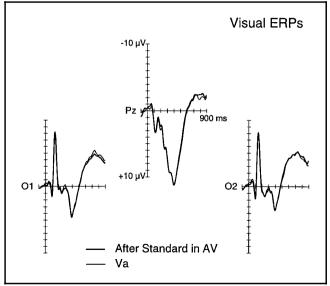
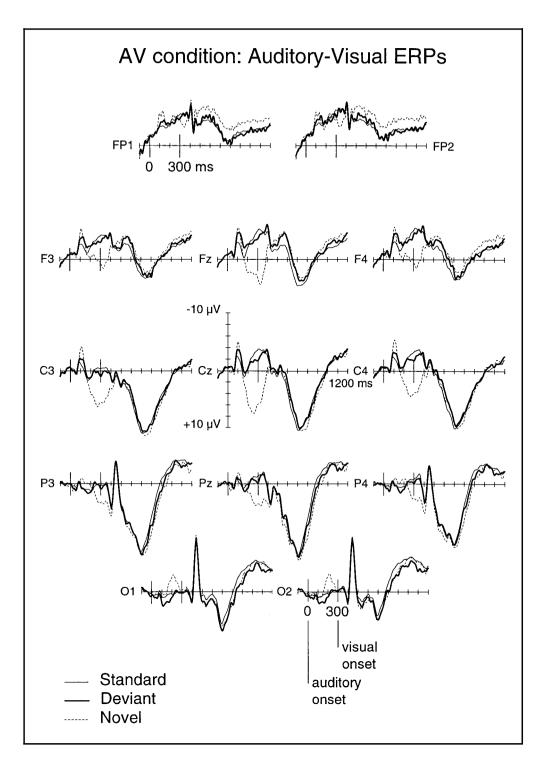


Figure 8. Grand-average visual ERPs at Pz, O1, and O2 in the Visual alone (Va) condition and in the Auditory-Visual (AV) condition after standard tones.

seem to cause a significant decrement in the number of hit responses to visual targets not observed after the novel sounds.

The differential effects of distracting novel and deviant sounds on visual performance may be explained by tracing these behavioral effects back to the associated neural events, as reflected by ERPs. Novel sounds enhanced the N1 amplitude and elicited the MMN, as suggested by significant differences between the standard-tone and novel-sound ERP that extended over the N1 latency range. The present MMN to novel sounds peaked and terminated (returned to baseline) earlier than the MMN to deviant tones, in concordance with previous studies showing that with larger deviances MMN becomes earlier (Tiitinen, May, Reinikainen, & Näätänen, 1994), overlapping the N1 peak for wider deviances (Tiitinen et al., 1994; Scherg et al., 1989). The present MMN to novel sounds is also supported by recent magnetoencephalograph (MEG) data showing that novel sounds activated an MMN source in the human auditory cortex (Alho et al., 1998). Thus, for novel sounds, the attention-switching signal was probably triggered by a combined response of the transient-detector system reflected in the supratemporal N1 (Näätänen, 1990, 1992; Näätänen & Picton, 1987), or some other N1 component, such as the frontal N1 reported by Giard et al. (1994), and the stimulus-change detector system reflected in the MMN (Näätänen, 1990, 1992). This combined signal for attention capture started with the N1 generation and was overlapped by the MMN process, resulting in an effective attentional reorientation as reflected by the subsequent large P3a wave and the clearly delayed RT to the following visual targets.

Figure 9. Grand-average ERPs in the Auditory-Visual (AV) condition to standardvisual (thin line), deviantvisual (thick line), and novel-visual (dashed line) stimulus pairs. Only the most relevant electrodes are shown. The epoch starts 100 msec before auditory-stimulus onset, which was used as the baseline period, to avoid the large amplitude variability due to the auditory response in the 100 msec preceding the visual stimulus. Novel sounds enhanced the fronto-polar negativity, and the frontal N2 to visual stimuli was enhanced after novel sounds and deviant tones, which also affected the offset slope of P3b. Notice that whereas the traces for standard-visual, deviant-visual, and novel-visual stimulus pairs perfectly match each other during the onset slope of the P3b, they start to differ from the P3b peak for the novel-visual stimulus pair and about 100 msec later for the deviant-visual stimulus pair (see P3, Pz, P4).



Deviant tones, in turn, elicited a distinct MMN, which was followed by a small P3a wave and by a second negative peak (Figure 5). Thus, the neural mechanism responsible for triggering the attention-switching signal was based, for deviant tones, on the stimulus-change detector system reflected in the MMN (Näätänen, 1990; 1992; Schröger, 1996). This attention capture signal associated with the MMN was sporadically followed by an orienting of attention, as suggested by the small (averaged) P3a and also by the 2.3% hit rate decrease to visual targets that followed deviant tones.

Although the subjects were instructed to ignore the auditory stimuli in the AV condition, the RT to visual targets following standard tones was slightly (insignificantly) shorter than the RT in the Va condition with no auditory stimuli. This suggests that the auditory stimuli were used as warning signals for the following visual targets, and thus, attention may have been bimodal in the AV condition. However, because the occurrence of an auditory stimulus change or a novel sound was task-irrelevant and unpredictable to the subjects, attention was involuntarily engaged by these events. In a more controlled experiment, attention was more effectively withdrawn from the auditory stimuli, and their occurrence was made noninformative by the simultaneous presentation of a visual cue stimulus informing about the successive appearance of a visual target (Alho et al., 1997). Like in the present study, a decreased hit rate for visual stimuli that followed MMN- and P3a-eliciting deviant tones was found. This confirms the involuntary nature of the attentionswitching mechanism indicated by the MMN, P3a, and deterioration of the visual-task performance.

Component Structure of the P3a to Novel Sounds

In both the Aa and AV conditions, novel sounds elicited a large, long-lasting, and broadly distributed P3a wave, with a double peak at Fz (in the Aa condition), suggesting a contribution of two different subcomponents. The early part of this P3a had a peak latency of 230 msec and showed a centrally dominant scalp distribution that was similar in the Aa and AV conditions (Figure 6). The early P3a inverted in polarity at posterior and lateral electrodes (Figures 3b and 5), suggesting bilateral generators located in the vicinity of the temporal and parietal lobes (cf. Scherg & von Cramon, 1986). The late part of the P3a, in turn, had a peak latency of 315 msec and did not invert in polarity at posterior electrodes, and its scalp distribution was centered over the right frontal areas. In contrast to the early P3a, the late P3a was enhanced in the AV condition, particularly over the right hemisphere (Figure 6), when subjects apparently used auditory stimuli as warning signals to prepare for visual targets.

The component structure of the present P3a, and the scalp distribution of the two subcomponents, is in agreement with previous results in patients with brain lesions, suggesting a critical role of the temporal-parietal junction in the generation of the auditory (Halgren, Baudena, Clarke, Heit, Marinkovic, et al., 1995; Halgren, Baudena, Clarke, Heit, Liégeois, et al. 1995; Knight, Scabini, Woods, & Clayworth, 1989), somatosensory (Yamaguchi & Knight, 1991, 1992), and visual P3a (Knight, 1991, 1997). It is also compatible with results suggesting a frontal contribution to P3a such as those obtained in patients with frontal lesions (Knight, 1984), in topographical analysis (Friedman & Simpson, 1994; Friedman, Simpson, & Hamberger, 1993), in dipole-source modeling (Mecklinger & Ullsperger, 1995), and in intracerebral recordings in humans (Baudena, Halgren, Heit, & Clarke, 1995).

The late P3a amplitude in the AV condition was enhanced with regard to its amplitude in the Aa condition. This amplitude enhancement of the late P3a reflects an

attentional monitoring of auditory stimuli in the AV condition, these stimuli being used as warning signals for the following visual targets. The attentional modulation of the P3a amplitude is in agreement with that reported in previous studies (Holdstock & Rugg, 1995; Woods, 1992) and suggests that the late P3a may more closely reflect an actual orienting of attention than the involuntary N1 and MMN processes triggering this attentional orienting. Also, several other findings support the association of the late phase of the P3a with the actual reorientation of attention or orienting response. First, the right-frontal dominance in the late P3a scalp distribution observed in the present experiment agrees with findings suggesting the involvement of right-frontal areas in the reorientation of attention (Fuster, 1989; Mesulam, 1981). Second, it has been shown that the P3a amplitude is attenuated with the repetition of the eliciting novel event (Courchesne, 1978; Friedman & Simpson, 1994; Knight, 1984). This habituating behavior is a characteristic feature of the orienting response (Öhman, 1979, 1992; Sokolov, 1963).

The early P3a was insensitive to attentional manipulations, as it was to similar amplitude and scalp distribution in the Aa and AV conditions. Its peak latency was rather short (230 msec), and its scalp distribution suggests neural generators probably located in the temporalparietal cortex (Knight, 1991, 1997; Knight et al., 1989; Yamaguchi & Knight, 1991). Thus, the early P3a might reflect a neural process other than attentional reorientation, such as the violation of a polisensorial model of the external world maintained in the temporal-parietal association cortex (Yamaguchi & Knight, 1991).

Conclusions

The results reported in the present paper support the notion of two different brain mechanisms of involuntary attention, differentially activated by different acoustic events. The ERP component structure to novel sounds suggests that unexpected novelty in the acoustic environment involuntary captures attention by simultaneously activating two different neural mechanisms: a transient-detector mechanism associated with the supratemporal N1 component of the auditory ERPs and a change-detector mechanism reflected in the MMN. Presumably, the attention-switching signal generated by the combined activation of these two mechanisms resulted in an effective engagement of attention, as indicated by the large P3a component elicited by the novel sounds and the increased RT to subsequent visual targets. Deviant tones, in turn, resulted in different behavioral effects and ERP component structure, which was characterized by the MMN. This supports the notion that small changes in the acoustic environment capture attention involuntarily by activating the stimulus-change detector mechanism reflected in the MMN.

METHODS

Subjects, Stimuli, and Procedure

Ten right-handed paid students (21 to 40 years, 3 males) with normal hearing and normal or corrected-to-normal visual acuity participated in the experiment. Two further subjects were discarded due to their inability to reach the required 90% hit rate in a practice session.

Subjects were presented with four blocks of 400 stimulus pairs (AV condition) in a visual forced-choice RT task. Each pair consisted of an auditory stimulus followed after 300 msec (onset-to-onset) by a visual stimulus. The interpair interval (onset-to-onset) was 1.2 sec (Figure 1). The auditory stimuli were either standard or deviant tones, and novel sounds, presented in random order with probabilities of 0.8, 0.1, and 0.1, respectively. The standard and deviant tones were sinusoidal tone bursts of 200-msec duration including 10-msec rise and fall times, presented binaurally at 75 dB SPL through headphones, with respective frequencies of 600 and 700 Hz. Sixty different environmental sounds, such as those produced by a drill, hammer, rain, door, telephone ringing, etc., were used as novel sounds. They were digitally recorded, then low-pass filtered at 10,000 Hz, and edited to have a duration of 200 msec, including rise and fall times of 10 msec, and an intensity maximum of 70 to 80 dB SPL. Each different novel sound occurred only once within a stimulus block and was presented twice or three times during the whole experiment. Both deviant tones and novel sounds were always preceded by at least one AV pair containing a standard tone. The visual stimuli were the digits 1 to 8 presented one at a time in random order on a computer screen for 200 msec. They subtended a vertical angle of 1.7° and a horizontal angle of 1.1° (30 mm × 20 mm; 100 cm from the subject's eyes).

Subjects were comfortably seated in a reclining chair in a dimly lit, electrically and acoustically shielded room. They were instructed to focus on the small fixation cross appearing in the middle of the screen and to press one response button with the right index finger for odd numbers and another button with the right middle finger for even numbers. Subjects were also instructed to ignore the auditory stimulation. Both speed and accuracy were emphasized for the primary visual task.

In two further conditions, visual stimuli were omitted (Va condition; Figure 1) or auditory stimuli were omitted (Aa condition; Figure 1). In the Va condition, subjects received three blocks of 200 visual stimulus each, the instruction being identical to that in the AV condition. In the Aa condition, subjects were instructed to concentrate on reading a self-selected book and to ignore the auditory stimulation.

The Aa and AV blocks were presented in an alternating order, with half of the subjects starting with the AV condition. The Va blocks were presented one at the beginning, one at the middle, and one at the end of the experiment. Before the experimental blocks, subjects participated in a practice session consisting of two Va blocks. They were required to reach a 90% hit rate before the experiment could start. Two out of 12 subjects failed to reach this level even after several additional blocks and were finally discarded.

EEG Recording and Averaging

The electroencephalogram (EEG) (bandpass 0–100 Hz) was continuously digitized at a rate of 500 Hz by a SynAmps amplifier (NeuroScan, Inc.) from 19 scalp electrodes positioned according to the 10–20 system: Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, and O2. One additional electrode was placed at the right mastoid (RM) and another at the left mastoid (LM). The horizontal EOG was recorded with electrodes attached to the left and right canthi. The vertical electroculogram (EOG) was assessed using recordings from Fp1 and Fp2 electrodes. The common reference electrode was placed on the tip of the nose.

ERPs were averaged off-line for each auditory stimulus class, for an epoch of 1300 msec including a preauditory stimulus period of 100 msec, in the AV and Aa conditions. In the Va condition, an ERP was also obtained for an epoch of 1300 msec, including a previsual stimulus period of 400 msec. Epochs in which the EEG or EOG exceeded $\pm 100 \ \mu$ V, as well as the five first epochs of each block, were automatically excluded from averaging. In the Aa and AV conditions, standard-tone trials immediately following deviant-tone or novel-sound trials were also excluded from the averages. Individual ERPs were band-pass filtered between 0.01 and 30 Hz.

Data Analysis

In the AV and Va conditions, a correct button press within 800 msec after visual-stimulus onset was regarded as a hit, the mean RT being computed only for the hit trials. An incorrect button press during this period was classified as an error, and trials with no response within this time window were considered misses. Hits, errors, misses, and RTs were computed across odd and even numbers.

Auditory ERP amplitudes (N1, P2, MMN, and P3a) were referred to the mean amplitude in the 100-msec baseline period preceding the auditory stimulus in both the Aa and AV conditions. For auditory ERP analysis, each individual waveform in the Aa and AV conditions was linearly detrended over an epoch of 600 msec (including baseline) to remove any possible asymmetrical contribution to EEG caused by slow EOG movements due to reading in the Aa condition. The effects of the standard tones on the visual ERPs were analyzed at O1 and O2 for peaks as follows (identified in the individual waveforms within the specified latency ranges): P1: 75 to 140 msec; N1: 100 to 200 msec; P2: 180 to 280 msec; N2: 225 to 350 msec. The visual P3b was identified at Pz between 300 and 500 msec from visual-stimulus onset. For this analysis, visual ERP amplitudes were measured against the mean amplitude of the 100-msec baseline preceding visual-stimulus onset. The fronto-polar negativity was analyzed at Fp1 and Fp2, the visual frontal N2 at F3, Fz, and F4, and the visual P3b at P3, Pz, and P4. The amplitude measurements for visual ERP components in the AV condition were referred to the 100-msec previsual stimulus baseline, which was differentially affected by standard tones, deviant tones, and novel sounds (see Figure 9).

The ERP and performance data were statistically analyzed by ANOVA with repeated measures. Where appropriate, nominal degrees of freedom and epsilon values after the Greenhouse-Geisser correction are reported. Scalp-distribution analyses were performed after normalizing ERP amplitudes to prevent amplitude differences between different components from washing out the genuine scalp-distribution differences. This normalization was done by dividing the amplitude at each electrode by the sum of the squared amplitudes at all electrodes (McCarthy & Wood, 1985).

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Note

1. The MMN amplitude in the unattended channel can be, however, attenuated under some conditions by strongly focusing attention on other sources of auditory stimulation (Näätänen, 1991; Näätänen, Paavilainen, Tiitinen, Jiang, & Alho, 1993; Oades & Dittmannbalcar, 1995; Trejo, Ryan-Jones, & Kramer, 1995; Woldorff, Hackley, & Hillyard, 1991).

REFERENCES

- Alho, K. (1995). Cerebral generators of mismatch negativity (MMN) and its magnetic counterpart (MMNn) elicited by sound changes. *Ear and Hearing*, *16*, 38-51.
- Alho, K., Escera, C., Díaz, R., Yago, E., & Serra, J. M. (1997). Effects of involuntary auditory attention on visual task performance and brain activity. *NeuroReport*, *8*, 3233– 3237.
- Alho, K., Winkler, I., Escera, C., Huotilainen, M., Virtanen, J., Jääskeläinen, I., Pekkonen, E., & Ilmoniemi, R. (1998). Proc-

essing of novel sounds and frequency changes in the human auditory cortex: Magnetoencephalographic recordings. *Psychophysiology*, *35*, 211–224.

- 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.
- Baudena, P., Halgren, E., Heit, G., & Clarke, J. M. (1995). Intracerebral potentials to rare target and distractor auditory and visual stimuli. III. Frontal cortex. *Electroencephalography and Clinical Neurophysiology*, 94, 251–264.
- Cherry, E. C. (1953). Some experiments on the recognition of speech with one and with two ears. *Journal of the Acoustical Society of America*, *29*, 975–979.
- Courchesne, E. (1978). Changes in P3 waves with event repetition: Long-term effects on scalp distribution and amplitudes. *Electroencephalography and Clinical Neurophysiology*, 45, 468–482.
- Courchesne, E., Hillyard, S. A., & Galambos, R. (1975). Stimulus novelty task relevance and the visual evoked potentials in man. *Electroencephalography and Clinical Neurophysiology*, 39, 131–143.
- Cowan, N., Winkler, I., Teder, W., & Näätänen, R. (1993). Memory prerequisites of the mismatch negativity in the auditory event-related potential (ERP). *Journal of Experimental Psychology: Learning, Memory and Cognition*, 19, 909–921.
- Csépe, V., Karmos, G., & Molnár, M. (1987). Evoked potential correlates of stimulus deviance during wakefulness and sleep in cat—animal model of mismatch negativity. *Electroencephalography and Clinical Neurophysiology*, 66, 571-578.
- Davis, H., Mast, T., Yoshie, N., & Zerlin, S. (1966). The slow response of the human cortex to auditory stimulus: Recovery process. *Electroencephalography and Clinical Neurophysiology*, 21, 105–113.
- Davis, H., & Zerlin, S. (1966). Acoustic relations of the human vertex potentials. *Journal of the Acoustical Society of America*, 39, 109-116.
- Dawson, M. E. (1990). Psychophysiology at the interface of clinical science, cognitive science, and neuroscience. *Psychophysiology*, 27, 243–255.
- Dawson, M. E., Fillion, D. L., & Schell, A. M. (1989). Is elicitation of the autonomic orienting response associated with allocation of processing resources. *Psychophysiology*, 26, 560–572.
- Duncan, C. C. & Kaye, W. H. (1987). Effects of chlonidine on event-related potential measures of information processing. In R. Johnson, J. W. Rohrbaugh, & R. Parasuraman (Eds.), *Current trends in event-related potential research (EEG supplement 40)* (pp. 527–531). Amsterdam: Elsevier.
- Fillion, D. J., Dawson, M. E., Schell, A. M., & Hazlett, E. A. (1991). The relationship between skin conductance orienting and the allocation of processing resources. *Psychophysiology*, 28, 410-424.
- Folk, C. L., Remington, R. W., & Johnston, J. C. (1992). Involuntary covert orienting is contingent on attentional control settings. *Journal of Experimental Psychology: Human Perception and Performance, 18*, 1030-1044.
- Ford, J. M., Roth, W. T., & Kopell, B. S. (1976). Auditory eventrelated potentials to unpredictable shifts in pitch. *Psychophysiology*, 13, 32–39.
- Friedman, D., & Simpson, G. (1994). ERP amplitude and scalp distribution to target and novel events: Effects of temporal order in young, middle-aged and older adults. *Cognitive Brain Research*, 2, 49–63.

Friedman, D., Simpson, G., & Hamberger, M. (1993). Age-related changes in scalp topography to novel and target stimuli. *Psychophysiology*, 30, 383–396.

Fuster, J. (1989). The prefrontal cortex: Anatomy, physiology, and neuropsychology of the frontal lobe. New York: Raven.

Gati, I., & Ben-Shakhar, G. (1990). Novelty and significance in orientation and habituation: A feature-matching approach. *Journal of Experimental Psychology: General*, 119, 251– 263.

Giard, M. H., Lavikainen, J., Reinikainen, K., Perrin, F., Bertrand, O., Pernier, J., & Näätänen, R. (1995). Separate representation of stimulus frequency, intensity, and duration in auditory sensory memory: An event-related potential and dipole-model analysis. *Journal of Cognitive Neuroscience*, 7, 133–143.

Giard, M. H., Perrin, F., Echallier, J. F., Thévenet, M., Froment, J. C., & Pernier, J. (1994). Dissociation of temporal and frontal components in the auditory N1 wave: A scalp current density and dipole model analysis. *Electroencephalography and Clinical Neurophysiology*, 92, 238–252.

Giard, M. H., Perrin, F., Pernier, J., & Bouchet, P. (1990). Brain generators implicated in processing of auditory stimulus deviance. A topographic ERP study. *Psychophysiology*, 27, 627-640.

Grillon, C., Courchesne, E., Ameli, R., Geyer, M., & Braff, D. L. (1990). Increased distractibility in schizophrenic patients. *Archives of General Psychiatry*, *47*, 171–179.

Halgren, E., Baudena, P., Clarke, J. M., Heit, G., Liégeois, C., Chauvel, P., & Musolino, A. (1995). Intracerebral potentials to rare target and distractor auditory and visual stimuli. I. Superior temporal plane and parietal lobe. *Electroencephalography and Clinical Neurophysiology*, 94, 191-220.

Halgren, E., Baudena, P., Clarke, J. M., Heit, G., Marinkovic, K., Devaux, B., Vignal, J. P., & Biraben, A. (1995). Intracerebral potentials to rare target and distractor auditory and visual stimuli. II. Medial, lateral, and posterior temporal lobe. *Electroencephalography and Clinical Neurophysiology*, 94, 229-250.

Hari, R., Hämäläinen, M., Ilmoniemi, R., Kaurokanta, E., Reinikainen, K., Salminen, J., Alho, K., Näätänen, R., & Sams, M. (1984). Responses of the primary auditory cortex to pitch changes in a sequence of tone pips: Neuromagnetic recordings in man. *Neuroscience Letters*, 50, 127–132.

Hari, R., Kaila, K., Katila, T., Tuomisto, T., & Varpula, T. (1982). Interstimulus interval dependence of the auditory vertex response and its magnetic counterpart: Implications for their neural generation. *Electroencephalography and Clinical Neurophysiology*, 54, 561–569.

Hikosaka, O., Miyauchi, S., & Shimojo, S. (1993). Voluntary and stimulus-induced attention detected as motion sensation. *Perception*, 22, 517–526.

Holdstock, J. S., & Rugg, M. D. (1995). The effect of attention on the P300 deflection elicited by novel sounds. *Journal* of *Psychophysiology*, 9, 18–31.

Jääskeläinen, I., Alho, K., Escera, C., Winkler, I., Sillanaukee, P., & Näätänen, R. (1996). Effects of ethanol and auditory distraction on forced choice reaction time. *Alcohol*, *13*, 153– 156.

Javitt, D. C., Schroeder, C. E., Steinschneider, M., Arezzo, J. C., & Vaughan, H. G. (1992). Demonstration of mismatch negativity in the monkey. *Electroencephalography and Clinical Neurophysiology*, 83, 87–90.

Knight, R. T. (1984). Decreased response to novel stimuli after prefrontal lesions in man. *Electroencephalography* and Clinical Neurophysiology, 59, 9–20.

Knight, R. T. (1991). Evoked potential studies of attention capacity in human frontal lobe lesions. In H. S. Levin, H. M. Eisenberg, & A. L. Benton (Eds.), *Frontal lobe function and dysfunction* (pp. 139-153). New York: Oxford University Press.

Knight, R. T. (1996). Contribution of human hippocampal region to novelty detection. *Nature*, 383, 256–259.

Knight, R. T. (1997). Distributed cortical network for visual attention. *Journal of Cognitive Neuroscience*, 9, 75-91.

Knight, R. T., Scabini, D., Woods, D. L., & Clayworth, C. C. (1989). Contributions of temporal-parietal junction to the human auditory P3. *Brain Research*, *502*, 109–116.

Kropotov, J. D., Näätänen, R., Sevostianov, A. V., Alho, K., Reinikainen, K., & Kropotova, O. V. (1995). Mismatch negativity to auditory stimulus change recorded directly from the human temporal cortex. *Psychophysiology*, *32*, 418-422.

Lehtonen, J. B. (1973). Functional differentiation between late components of visual evoked potentials recorded at occiput and vertex: Effects of stimulus interval and contour. *Electroencephalography and Clinical Neurophysiol*ogy, 35, 75-82.

Lyytinen, H., Blomberg, A. P., & Näätänen, R. (1992). Event-related potentials and autonomic responses to a change in unattended auditory stimuli. *Psychophysiology*, 29, 523– 534.

Mäkelä, J. P., Hari, R., & Leinonen, L. (1988). Magnetic responses of the human auditory cortex to noise/square wave transitions. *Electroencephalography and Clinical Neurophysiology*, 69, 423–430.

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.

McMillan, N. A. (1973). Detection and recognition of intensity changes in tone and noise: The detection-recognition disparity. *Perception & Psychophysics, 13,* 65-75.

Mecklinger, A., & Ullsperger, P. (1995). The P300 to novel and target events: A spatio-temporal dipole model analyses. *NeuroReport*, *7*, 241-245.

Mesulam, M. M. (1981). A cortical network for directed attention and unilateral neglect. *Annals of Neurology*, 10, 309– 325.

Molnár, M., Skinner, J. E., Csépe, V., Winkler, I., & Karmos, G. (1995). Correlation dimension changes accompanying the occurrence of mismatch negativity and the P3a eventrelated potential component. *Electroencephalography and Clinical Neurophysiology*, 95, 118–126.

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. (1991). Mismatch negativity outside strong attentional focus: A commentary on Woldorff et al. (1991). *Psychophysiology*, 28, 478–484.

Näätänen, R. (1992). Attention and brain function. Hillsdale, NJ: Erlbaum.

Näätänen, R., Paavilainen, P., Alho, K., Reinikainen, K., & Sams, M. (1989). Do event-related potentials reveal the mechanism of the auditory sensory memory in the human brain? *Neuroscience Letters*, *98*, 217-221.

Näätänen, R., Paavilainen, P., Tiitinen, H., 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 a analysis of the component structure. *Psychophysiology*, 24, 375-425.

Oades, R. D., & Dittmannbalcar, A. (1995). Mismatch negativity (MMN) is altered by directing attention. *NeuroReport*, *6*, 1187–1190. Öhman, A. (1979). The orienting response, attention and learning: An information-processing perspective. In H. D.
Kimmel, E. H. van Olst, & J. F. Orlebeke (Eds.), *The orienting reflex in humans* (pp. 443–471). Hillsdale, NJ: Erlbaum.

Öhman, A. (1992). Preattentive processing and orienting. In B. A. Campbell, H. Hayne, & R. Richardson (Eds.), Attention and information processing in infants and adults: Perspectives from human and animal research (pp. 263– 295). Hillsdale, NJ: Erlbaum.

Pfefferbaum, A., Buchsbaum, M., & Gips, J. (1971). Enhancement of the average evoked response to onset and cessation. *Psychophysiology*, 8, 332–339.

Posner, M. I. (1978). *Chronometric explorations of mind.* Hillsdale, NJ: Erlbaum.

Ritter, W., Vaughan, H. G., Jr., & Costa, L. D. (1968). Orienting and habituation to auditory stimuli: A study of short term changes in average evoked responses. *Electroencephalography and Clinical Neurophysiology*, 25, 550-556.

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.

Scherg, M., & von Cramon, D. (1986). Evoked dipole source potentials of the human auditory cortex. *Electroencephalography and Clinical Neurophysiology*, 65, 334–360.

Schröger, E. (1996). A neural mechanism for involuntary attention shifts to changes in auditory stimulation. *Journal of Cognitive Neuroscience*, 8, 527–539.

Siddle, D. A. T. (1991). Orienting, habituation, and resource allocation: An associative analysis. *Psychophysiology*, 28, 245-259.

Sokolov, E. N. (1963). *Perception and the conditioned reflex.* New York: Macmillan.

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). Hillsdale, NJ: Erlbaum.

Squires, N. K., Squires, K. C., & Hillyard, S. A. (1975). Two varieties of long-latency positive waves evoked by impredictable auditory stimuli. *Electroencephalography and Clinical Neurophysiology*, 38, 387–401.

Theeuwes, J. (1994). Stimulus-driven capture and attentional set: Selective search for color and visual abrupt onsets. *Journal of Experimental Psychology: Human Perception and Performance*, 20, 799–806.

Tiitinen, H., May, P., Reinikainen, K., & Näätänen, R. (1994). At-

tentive novelty detection in humans is governed by preattentive sensory memory. *Nature*, *372*, 90-92.

Trejo, L. J., Ryan-Jones, D. L., & Kramer, A. F. (1995). Attentional modulation of the mismatch negativity elicited by frequency differences between binaurally presented tones. *Psychophysiology*, 32, 319–328.

Vaughan, H. J., Jr., & Ritter, W. (1970). The sources of the auditory evoked responses recorded from the scalp. *Electroencephalography and Clinical Neurophysiology*, 28, 360–367.

Velasco, M., & Velasco, F. (1986). Subcortical correlates of the somatic, auditory and visual vertex activities. II. Referential EEG responses. *Electroencephalography and Clinical Neurophysiology*, 63, 62-67.

Velasco, M., Velasco, F., & Olvera, A. (1985). Subcortical correlates of the somatic, auditory and visual vertex activities. I. Bipolar EEG responses and electrical stimulation. *Electroencephalography and Clinical Neurophysiology*, 61, 519–529.

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.

Woods, D. L. (1992). Auditory selective attention in middleaged and elderly subjects: An event-related brain potential study. *Electroencephalography and Clinical Neurophysiol*ogy, 84, 456-468.

Woodward, S. H., Brown, W. S., March, J. T., & Dawson, M. E. (1991). Probing the time-course of the auditory oddball P3 with secondary reaction time task. *Psychophysiology, 28*, 609–616.

Yamaguchi, S., & Knight, R. T. (1991). Anterior and posterior association cortex contributions to the somatosensory P300. Journal of Neuroscience, 11, 2039–2054.

Yamaguchi, S., & Knight, R. T. (1992). Effects of temporalparietal lesions on the somatosensory P3 to lower limb stimulation. *Electroencephalography and Clinical Neurophysiology*, 84, 139–148.

Yantis, S. (1993). Stimulus-driven attentional capture and attentional control settings. *Journal of Experimental Psychology: Human Perception and Performance*, 19, 676-681.

Yantis, S., & Hillstrom, A. P. (1994). Stimulus-driven attentional capture: Evidence from equiluminant visual objects. *Journal of Experimental Psychology: Human Perception* and Performance, 20, 95–107.