The N1 hypothesis and irrelevant sound:Evidence from token set effects

Campbell, Thomas

Elsevier B.V.
2003

Campbell, T, Winkler, I, Kujala, T M & Näätänen, R 2003, 'The N1 hypothesis and irrelevant sound:Evidence from token set effects', Cognitive brain research, vol. 18, no. 1, pp. 39-47. https://doi.org/10.1016/j.c

http://hdl.handle.net/10138/228145
10.1016/j.cogbrainres.2003.09.001

submittedVersion

Available online at www.sciencedirect.com
SCIENCE DIRECT®
Cognitive Brain Research 18 (2003) 39–47
COGNITIVE BRAIN RESEARCH
www.elsevier.com/locate/cogbrainres

Research report

The N1 hypothesis and irrelevant sound: evidence from token set size effects

Tom Campbell[a,b], István Winkler[a,b,c,*], Teija Kujala[a,b,d], Risto Näätänen[a,b]

[a] Cognitive Brain Research Unit, University of Helsinki, Helsinki, Finland
[b] Helsinki Brain Research Centre, University of Helsinki, Helsinki, Finland
[c] Hungarian Academy of Sciences, Budapest, Hungary
[d] Helsinki Collegium for Advanced Studies, University of Helsinki, Helsinki, Finland

Accepted 3 September 2003

Abstract

This study investigated how increases in the number of different types of sound (token set size) within a heard but ignored sequence influence brain activity and performance in a serial recall task (the irrelevant sound effect). We tested the hypothesis that brain processes affected by the refractory state of the neuronal populations involved in generating the auditory N1 play a role in the memory disruption produced by irrelevant sound. Auditory event-related potentials (ERPs) were recorded when volunteers performed a serial recall task that required remembering lists of visually presented numbers that were followed by a distractor-filled retention interval. The results showed that both increments in set size from 1 to 2 and from 2 to 5 elicited an increase of the N1 amplitude. Furthermore, increases in set size from 2 to 5, but not from 1 to 2, caused a significant decrease of the serial recall performance. This result suggested that, if N1 were to play a role in the disruption produced by irrelevant sound, the processes underlying the N1 wave may only serve as a necessary rather than a sufficient condition for disruption.

Theme: Neural basis of behavior
Topic: Cognition

Keywords: Token set size; Irrelevant sound effect; Auditory N1; Immediate memory; Serial recall

1. Introduction

Irrelevant sound can disrupt performance on tasks that require the recollection of recently presented material. This disruption occurs even when people try to ignore that sound. In addition to its implications for approaches to noise abatement and human factors research [3–5,19], laboratory data on this irrelevant sound effect have been used to constrain a number of extant models of the relation of immediate memory to auditory perception [2,6,11,12,21,22,38]. An open question has remained whether this effect is mediated by the processing of changes [48] or, rather, by the processing of the different types of item in the irrelevant sequence [10]. However, neither the neural basis of irrelevant changes nor that of irrelevant tokens has been investigated within this context. Neurobiological data may shed some light upon this open question.

The few electrophysiological studies of the irrelevant sound effect have focused upon the event-related potentials (ERPs) elicited by the to-be-recalled items on an immediate serial recall task, when volunteers were required to remember the order of a list of letters. The attended letters were alternated with irrelevant letters that people were asked to ignore. With auditory to-be-remembered items, volunteers attended a stream of to-be-remembered items spoken by a man, and ignored an interleaved sequence of irrelevant letter sounds spoken by a woman [30,31]. In an analogous study, people attended to visual to-be-remembered items, interleaved with irrelevant spoken letter sounds or tones [29].

In all cases, those sound sequences that included more acoustic dissimilarity between the constituent sounds proved

* Corresponding author. Institute for Psychol., Hungarian Academy of Sciences, H-1394 Budapest, P.O. Box 398, Hungary. Tel.: +36-1-3533-244; fax: +36-1-269-2972.
E-mail address: winkler@cogpsyphy.hu (I. Winkler).

0926-6410/$ - see front matter © 2003 Elsevier B.V. All rights reserved.
doi:10.1016/j.cogbrainres.2003.09.001

more disruptive to immediate memory performance. This disruptive effect coincided with a long-lasting frontal positivity and posterior negativity. The effect was maximal around 300 ms, in the case of auditory items, and 400 ms with visual to-be-remembered items.

This electrophysiological effect thus coincided with significant increases in the irrelevant sound effect whether the to-be-remembered items were auditory or visual. However, this effect could be described a sufficient yet not a necessary condition for disruption by irrelevant sound. Indeed, this ERP effect could not be shown to occur when the irrelevant sound was presented after the list of to-be-remembered items, yet, a disruption has been shown to occur also under such circumstances [10,28,32,41].

With irrelevant sound confined to a retention interval, Campbell et al. [10] showed that the greater the number of different types of irrelevant items—that is, the greater the token set size—the greater the disruption. Not only was there a disruptive advantage shown with an increment in set size from 1 (AAAAAAAAAA...) to 2 (ABABABABAB...), but also an additional disruptive advantage of a five-token (ABCDEABCDE...) over a two-token sequence. Thus, this behavioural disruption was not attributable to the fact of change per se.

This finding with auditory to-be-remembered items was discrepant with those shown elsewhere with visual to-be-remembered items [48], where increments in set size from 1 to 2 were significant, yet, those beyond 2 only generated a modest numerical disruptive advantage that did not prove reliable. The central concern of the present investigation was the electrophysiological basis of this effect.

Clues to the electrophysiological basis of the irrelevant sound effect may reside within the literature on auditory ERPs to sounds that are heard yet ignored. An extensive literature exist upon the auditory mismatch negativity (MMN) is a fronto-centrally maximal auditory ERP component that is elicited task-independently by ‘‘deviant’’ sounds violating some regular aspect of the preceding acoustic stimulation (for reviews, see Refs. [33,34,36]). One theoretical possibility is that MMN serves as a call for attention to be directed to the deviant sound [33,44]. The switch of attention is assumed to produce a fronto-central positivity (P3a) occurring subsequent to the MMN. The elicitation of the P3a coincides with impaired performance and prolonged reaction times on primary tasks [7,14–16,43,45–47,51,52]. However, it is known that tonal sequences similar in structure to those used by Campbell et al.—where no rule is violated—do not elicit a MMN [20] (cf. Ref. [48]).

A more promising explanation of the token set size effect concerns the N1 component of the auditory ERP, which is a long-latency vertex negativity, peaking ca. 100 ms after stimulus onset. It has been shown that when N1 is of a higher amplitude, a subsequent P3a can follow [1]. A possibility is that N1 must exceed a momentary threshold for the occurrence of P3a, which coincides with conscious detection of the onsets of sounds [33]. The N1 component of the auditory ERP has been shown to exhibit refractoriness (for reviews, see Refs. [35,36]). That is, upon repeated presentation of a given sound, the N1 amplitude attenuates in a manner that is subject to recovery following a period of silence. N1 is generated by neuronal populations that are activated by the auditory stimuli. Some of these neuronal populations are tuned to specific attributes of that stimulus. The responsiveness of these populations become attenuated by repeated presentation of a sound in a manner, which is subject to recovery after a period of silence or the absence of the particular feature to which the given population is tuned. Accordingly, the more tokens in a given sequence, the less features subsequent sounds share and, in turn, the less feature-specific refractoriness will occur [20].

Näätänen [33] suggested that when the activity of the N1-generating neuronal populations exceeds a momentary threshold, the conscious detection of onsets, energy changes or transitions within a sequence of sounds takes place, i.e., attention is ‘‘switched’’ to a significant discontinuity in that sequence. However, even when attention is not switched to the irrelevant material, it is assumed that the activity of the N1-generating neuronal populations engages some processing capacity (if nothing else, then the ‘‘call for attention’’ has to be processed before it is denied). This engagement of processing capacities may interfere with the processes that support retention of the to-be-remembered stimuli. In this way, the increased processing demands of sequences within which N1s of a larger amplitude are elicited—such as sequences containing more tokens—may result in an increased disruption of performance of the primary task, even when the sound is ignored successfully such as it does not (often) capture attention.

The objective of the present study was to test this N1 hypothesis that the refractory properties of the neuronal populations underlying the N1 wave play a role in the effect of the token set size on the memory disruption caused by irrelevant sound. Restated explicitly, the N1 hypothesis puts forth the proposition that an increase in auditory N1 amplitude will produce an increase in memory disruption. The approach adopted was to manipulate the size of the set from which the irrelevant sounds were drawn, using visual to-be-remembered items while recording the EEG.

2. Materials and methods

2.1. Volunteers

Twenty-two university students and members of the public volunteered in exchange for a small honorarium. The experiment was undertaken with the understanding and written consent of each volunteer according to the Declaration of Helsinki. Four volunteers’ data, which were contaminated by extensive artefacts, were excluded from the study.

The ages of the remaining volunteers ranged from 18 to 27 with a mean age of 23 years (three males). All reported intact hearing and normal or corrected-to-normal vision. They were right-handed, and Finnish was their first language.

2.2. Stimuli and procedure

The structure of one trial is shown in Fig. 1. Five seconds before the onset of each list, a fixation cross (+) appeared in the centre of the computer screen. After 2 s, a tone was presented to warn volunteers that a list was about to be presented. The screen was blank for 200 ms following the offset of the fixation cross and then a list of to-be-recalled material was presented, which consisted of the numbers 1 to 9 in a random order that contained no easy-to-remember sequences. These digits were presented for 800 ms with an interitem interval of 200 ms when the screen was black. Volunteers were required to attend to these digits.

A 10.5-s retention interval followed list presentation, during which the word ''ODOTA'' (meaning ''wait'') appeared in the centre of the screen. The onset of the first irrelevant sound came 900 ms after the onset of the word ''ODOTA'' and the last irrelevant sound onset came 550 ms after this word's offset. During this period, volunteers were required to silently rehearse the list items and to ignore any sound that they heard.

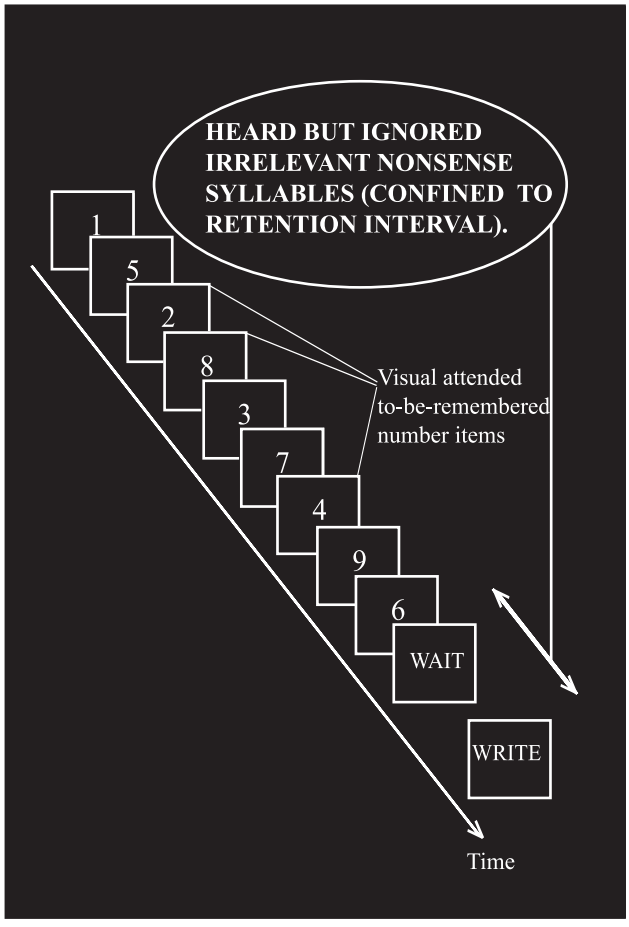

Fig. 1. Schematic diagram of one trial.

The screen was blank for 1 s, and then the word ''KIRJOITA'' (write) appeared for 10 s, followed by the fixation cross that preceded the next list. During this period, participants were required to write down the digits in a strictly left to right fashion, without correction, while attempting to preserve the correct serial order and position of items, leaving a ''/'' when uncertain about an item.

Volunteers initiated the first trial by saying that they were ready; subsequent lists were then presented at regular intervals.

The order of irrelevant speech conditions was randomised, each condition receiving 20 trials (the to-be-remembered list followed by 30 irrelevant speech sounds). For each one-token trial, 30 repetitions of a token followed the list. Each of the five tokens—*jus*, *käs*, *nev*, *tam* or *poi*—were presented after four lists (5 tokens × 4 lists/token = 20 trials). With two-token sequences, the 30 post-list sounds were structured as ABABAB. . ., each one of the 20 possible orderings of two tokens out of the set of five was used once. On five-token trials, six repetitions of a random ordering of the five different syllables followed each list. For each of the 20 lists in the five-token condition, a different random ordering of irrelevant tokens was used. Thus, each token appeared the same number of times in the three conditions.

Digits were presented in the centre of the computer screen, at a size of 45 × 55 mm in white Helvetica font on a black background. Viewing distance was 1.50 m. Irrelevant material was presented at 85 dB SPL. Each irrelevant item had a fundamental frequency of 115 Hz, was digitized at 44.1 kHz to 16-bit resolution and lasted 280 ms with an interitem silence of 70 ms. The irrelevant stimuli were delivered binaurally via headphones. During the procedure, volunteers were seated in an acoustically and electrically shielded room.

2.3. EEG recording and analysis

EEG was recorded with a 30-channel electrode array of electrodes that were evenly distributed across the scalp [49]. The reference electrode was attached to the nose. Horizontal eye movements were monitored with a bipolar set-up, the two electrodes were attached laterally to the outer canthi of the eyes. Vertical eye movements were monitored using the pre-frontal electrodes (Fp1, Fp2, Fpz) on the cap against the common reference. In order to record mouth and tongue movements, an additional electrode was placed on the submandibular surface and differentially amplified relative to an electrode located on the right masseter muscle. The bioelectric potentials were amplified within frequency limits (0–30 Hz) and digitised (500 Hz, NeuroScan SynAmp system) online. EEG was then filtered (0.5–30 Hz) offline and epochs of 420 ms (including 100-ms pre-stimulus period) were averaged after artefact rejection (epochs with

EEG or EOG exceeding ±50 μV in any channel). ERPs were averaged for each set size condition, collapsing across the particular token sounds used.

Behavioural responses were scored with a strict serial position criterion, and mean error probabilities were collapsed across serial position and trial for each token set size condition. The mean error probability data were entered to a one-way repeated-measures analysis of variance (ANOVA), and critical linear contrasts were conducted.

ERPs elicited by the irrelevant items were digitally re-referenced to the average of all scalp electrodes offline [50]. ERP components were measured at the following integration windows (centred on the peak of the corresponding wave in the group-averaged responses): P1 (58–78 ms), N1 (100–140 ms), P2 (184–204 ms), N2 (252–272 ms). Amplitude measurements were referred to the mean voltage during the 100-ms pre-stimulus period with the exception of N1, as explained below.

A visual inspection of the ERP responses revealed that the N1 wave substantially overlapped the positive deflections appearing in the P1 and P2 latency ranges (see Fig. 3; see also Section 3.2.1). In order to remove the confounding influences of the overlapping ERP components, N1 amplitudes were measured as the average amplitude in the above-defined 40-ms integration window with respect to the line connecting the corresponding positive P1 and P2 peaks (see the shaded area at Fz and Cz in Fig. 3). When estimating the N1 amplitudes this way, the P1 and P2 peaks were represented by their corresponding averaged amplitude measurements (see above).

For each ERP component, two ANOVAs were conducted. Amplitudes were computed from a 3 × 3 array of aligned electrodes consisting of C3, Cz, C4, F3, Fz, F4, and the anterior frontal electrode line, AF3, AFz and AF4. The amplitudes were submitted to a 3 (Token Set Size) × 3 (Frontality) × 3 (Laterality) repeated-measures ANOVA and post hoc Neuman–Keuls tests—with critical α set to 0.05—were conducted where appropriate. The average of the amplitudes measured at M1 and M2 (left and right mastoids, respectively) was entered into a separate one-way ANOVA with three levels of token set size, and critical planned comparisons were conducted. For each token set size, isopotential maps were computed for the N1 amplitudes (measured as explained above). Corresponding maps of Hjorth-transforms [17] were calculated to assess the scalp distribution of the density of radially oriented sources.

3. Results

3.1. Behavioural data

The pattern of mean error probabilities, depicted in Fig. 2, showed that while roughly equivalent disruptive effects occurred with one and two tokens, a larger disruption was shown with the five-token sequences. Statistical analysis

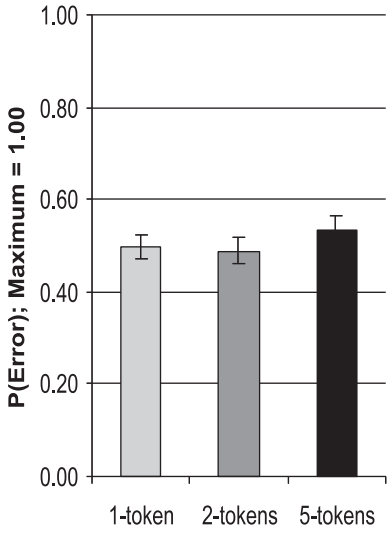

Fig. 2. Mean error probabilities and their standard errors as a function of token set size; $N = 18$.

corroborated these tendencies: The main effect of token set size was significant [$F(2,34) = 4.71$, M.S.E. = 0.002, $p = 0.016$]. Moreover, the disruptive advantage of five over two tokens was significant [$F(1,17) = 7.71$, M.S.E. = 0.005, $p = 0.013$], while the difference between two tokens over one token was not statistically reliable [$F(1,17) = 0.32$, M.S.E. = 0.004, $p = 0.581$].

3.2. ERP data

The primary ERP finding differed from the behavioural disruption. There was an increase in N1 that occurred with each enlargement of set size, as apparent in Fig. 3. There were, however, effects apparent in P1 and P2 that showed substantial overlap with N1.

3.2.1. P1 and P2

The P1 and P2 amplitudes showed a decrease with increases in set size (Fig. 3). Because increases in set size produce increases in the inter-stimulus intervals between identical tokens, an increase in P1 and P2 amplitudes was expected with increased token set size [1]. The opposite pattern of results obtained in the current study suggests that N1 substantially overlapped the P1 and P2 waves. That is, the expected positive increase of the P1 and P2 amplitudes was probably turned to a decrease by the overlap with the N1 wave, which showed a numerically greater negative increase.

If the decrease of the P1 and P2 amplitudes—when an increase could be expected—attained significance, then this would necessitate the form of N1 measurement illustrated in Fig. 3. The main effect of token set size was significant for the fronto-central P1 amplitude [$F(2,34) = 11.09$, M.S.E. = 0.775, $p < 0.001$], as well as at the mastoid leads [$F(2,34) = 10.43$, M.S.E. = 0.391, $p < 0.001$]. Also, the fronto-central P2 amplitudes showed a significant main effect of token set

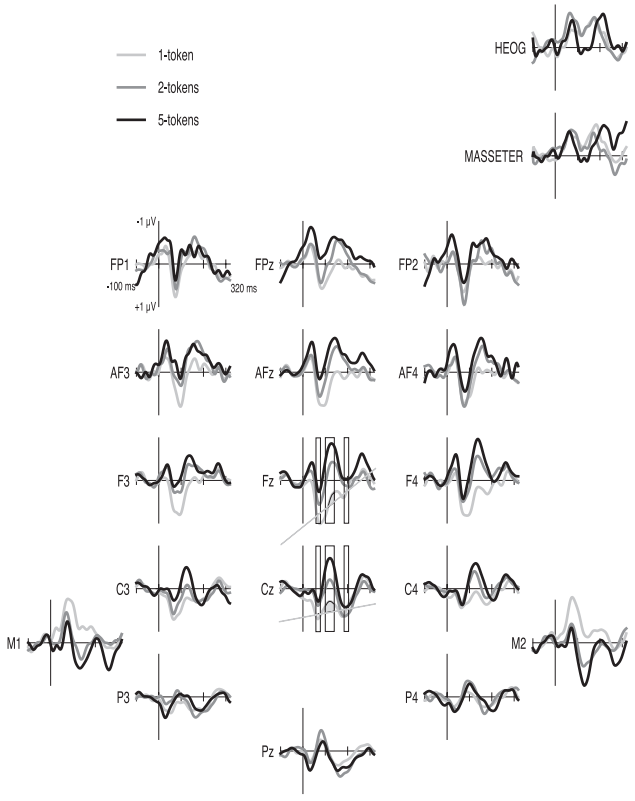

Fig. 3. Grand-averaged ERPs elicited by irrelevant sound items at selected electrodes; $N=18$. Windows around P1, N1 and P2 shown for Fz and Cz represent the windows of integration within which amplitude measurements were taken. A line is drawn between the averaged peaks for P1 and P2 within these windows. The measure of N1 used in the paper is the shaded area between this line and the ERP wave within the window of integration.

size [$F(2,34)=3.70$, M.S.E. $=0.662$, $p=0.035$], although at the mastoids, the effect of token set size missed significance and was marginal [$F(2,34)=3.14$, M.S.E. $=0.462$, $p=0.070$].

3.2.2. N1 and N2

Consistent with the N1 hypothesis, the main effect of token set size was significant for the fronto-central amplitude measures [$F(2,42)=16.17$, M.S.E. $=0.948$, $p<0.001$; see Fig. 3 and Table 1, left side], as was that of frontality [$F(2,42)=3.97$, M.S.E. $=1.413$, $p=0.045$], and laterality [$F(2,42)=9.84$, M.S.E. $=0.617$, $p<0.001$; see also the upper panel of Fig. 4]. The only interaction to reach significance was that of set size by frontality [$F(4,84)=3.82$, M.S.E. $=0.344$, $p=0.020$]. Post hoc Neuman–Keuls tests revealed that increases in N1 amplitude at anterior–frontal sites were apparent with increments in set size from 1 to 2, yet not from 2 to 5. At frontal electrodes, increases in amplitude were shown with each increment in set size, as was also the case at central electrodes. Post hoc Neuman–Keuls tests at Fz and Cz separately revealed a significant increase with each enlargement of set size.

Interactions involving laterality did not attain significance. Thus, while N1 showed a significantly greater amplitude over the right than the left hemisphere (Figs. 3 and 4, top panel), the sources underpinning the influence of set size on the N1 amplitude were bilateral at the three set size levels tested.

At the mastoids, the main effect of set size was also significant [$F(2,34)=10.50$, M.S.E. $=0.666$, $p=0.002$]. Increments in set size from 1 to 2 as well as from 2 to 5 were found to produce significantly increased positivities

Table 1
Grand-averaged N1 amplitudes and Hjorth-transformed values (both in μV units) at selected electrodes; $N=18$

Potential				Hjorth		
Electrode	One token	Two tokens	Five tokens	One token	Two tokens	Five tokens
FP1	−0.416	−0.374	−0.374	−0.454	−0.573	−0.615
Fz	−0.029	−0.153	−0.194	−0.022	−0.088	−0.176
FP2	−0.394	−0.428	−0.516	−0.400	−0.527	−0.625
AF3	0.130	0.009	0.066	−0.011	−0.250	−0.496
Afz	−0.078	−0.427	−0.504	−0.095	−0.466	−0.555
AF4	−0.203	−0.399	−0.424	−0.101	−0.435	−0.412
F3	−0.045	−0.198	−0.409	0.064	−0.028	−0.152
Fz	−0.182	−0.650	−0.891	−0.045	−0.278	−0.372
F4	−0.107	−0.624	−0.839	−0.113	−0.516	−0.902
C3	−0.226	−0.417	−0.770	−0.181	−0.426	−0.620
Cz	−0.280	−0.694	−1.031	−0.042	−0.029	−0.066
C4	−0.227	−0.634	−0.807	−0.217	−0.328	−0.391
P3	−0.085	−0.099	−0.187	−0.005	0.101	0.281
Pz	−0.041	−0.255	−0.599	0.349	0.600	0.873
P4	0.098	0.177	0.484	0.010	0.062	−0.098
M1	0.222	0.688	0.920	0.273	0.829	1.394
M2	0.380	0.671	1.083	0.185	0.390	0.823

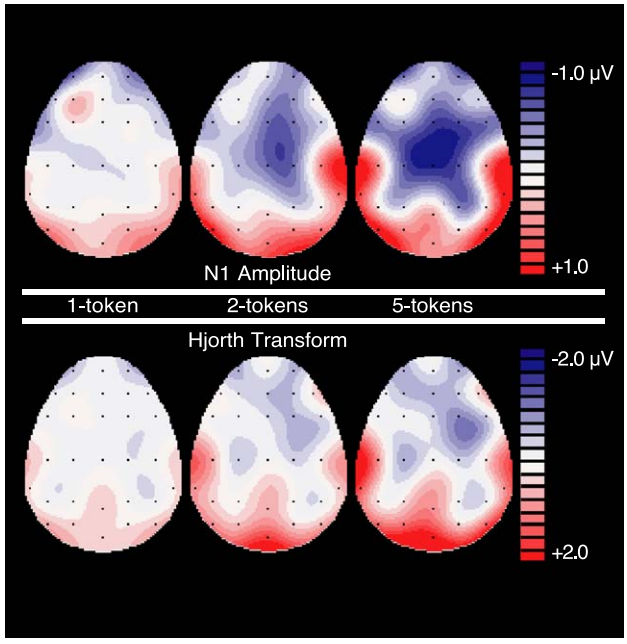

Fig. 4. Isopotential maps of N1 amplitude for each condition (upper panel), together with a measure of the density of radially oriented sources (the Hjorth transform, lower panel). Both maps are calibrated in microvolt units [17].

[$F(1,17) = 9.98$, M.S.E. = 0.258, $p = 0.006$ and $F(1,17) = 7.08$, M.S.E. = 0.263, $p = 0.016$, respectively].

No N2 effects were found to be significant.

4. Discussion

The results showed that an increase in N1 occurs alongside an increase in the memory disruption produced by sound that is heard but ignored. This disruption was produced by increasing the number of different types of irrelevant items (set size) from 2 to 5. The main finding was that an increase in set size from 2 to 5 not only caused an increase in N1, as indexed at Fz, Cz and the mastoids, but also caused an increase in error probability.

The pattern of results were thus consistent with the N1 hypothesis in that the pattern of refractoriness exhibited by N1, in this range, resembled that shown by the disruption of memory produced by irrelevant sound. However, less consistent with the N1 hypothesis was that an increase in set size from 1 to 2, though eliciting increases in N1, did not elicit a significant increase in disruption. It is thus necessary to assume that, if N1 played a role in disruption, an increase in N1 served as a necessary rather than as a sufficient condition for disruption.

While there have been a number of detailed accounts of the generator structure of N1 [18,42,53,27], the current study did not produce results that would delineate how the component structure of N1 might relate to disruption. A possible locus for the source of the significant increases shown at Cz and the mastoids with each increment in set size may be conceptualised as the stimulus-specific N1 Component 3, as elaborated by Näätänen and Picton [35]. Alcaini et al. [1] dissociated also a non-specific N1 component with a frontal topography and a recovery period of less than 8 s. The Hjorth-transforms (Fig. 4, lower panel, Table 1, left side) show signs of a frontal contribution to the observed waveform. If the N1 generator described by Alcaini et al. exhibited some stimulus-specific sensitivity (this has not been ruled out by Alcaini et al.), then it may have contributed to the currently observed token set size effect.

Dissociation of the influences of Alcaini et al.'s [1] and Näätänen and Picton's [35] stimulus-specific components was not possible from the present results. We can be sure that the supratemporal N1 component was affected by the current manipulation in a manner consistent with the conclusion that the supratemporal N1 generators may play a role in memory disruption, but the elicitation of this component is not a sufficient condition for memory disruption. Corroborative evidence for this view stems from the measures reflecting the density of sources on the scalp at the N1 peak latency. Fig. 4 (lower panel) shows qualitatively similar source distributions regardless of set size as well as monotonous quantitative change in the source density with each increment in set size. Signs of increasing frontal activity in the N1 latency range with increased token set size suggest that the frontal N1 generator (perhaps the one described by Alcaini et al.) may show some stimulus specificity and, thus, may have contributed to the observed N1 effects as measured over the central and frontal scalp.

Our results do not fully corroborate those of Campbell et al. [10], who found a significant increase in memory disruption both when increasing the token set size from 1 to 2 and from 2 to 5. However, for distinguishing the change-based and token set size explanations of the irrelevant sound effect, it is important to note that we replicated Campbell et al.'s finding of the disruptive advantage of five over two tokens. In Campbell et al.'s investigation [10], enlargements in set size increased the time interval between repetitions of a particular token in the irrelevant sequence. Although Campbell et al. [10] did not measure the amplitude of N1, based on the present results and on other studies of N1, we can expect that in their study, the N1 amplitude to the irrelevant items increased with increasing token set sizes. In turn, these increases may have caused a greater interference with the brain activity supporting memory for the to-be-remembered items. It still remains puzzling as to why our results did not replicate the reliable disruptive advantage of two tokens over one token shown by Campbell et al. [10]. This was, however, not the first instance of observing a non-linearity in disruption as a function of set size in the irrelevant sound literature [9,25,48].

It is worth noting that Campbell et al. [10] used auditory to-be-remembered items. Inspection of mean error probabilities from Campbell et al.'s [10] Experiment 3a showed that the disruption shown here with auditory to-be-remembered items was overall larger than that seen with visual items here. It might seem that some additional disruption—such as the compound suffix effect [26]—was in operation and that this disruption was reliant upon a modality-specific mechanism confined to the processing of auditory to-be-remembered items by heard irrelevant material. Such an account may have explained why no significant increase in disruption was found with increments in set size from 1 to 2 in the present study, in which to-be-remembered items were presented visually, whereas Campbell et al.'s Experiment 3a demonstrated an increase in disruption with auditory to-be-remembered items. However, this account contrasts the finding of Campbell et al.'s Experiment 1, which showed that identical sequences of irrelevant sound had no additional disruptive potency whether to-be-remembered items were seen or heard by the same group of participants during one experiment session. Campbell et al.'s Experiment 1, taken together with the substantial evidence that individual differences in the magnitude of disruption produced by irrelevant speech replicates across experimental sessions [13], suggests that such informal between-groups comparisons merit cautious interpretation worthy of further scrutiny. At this stage, the evidence could not conclusively support the view that the modality of the to-be-remembered

items was the crucial determinant of the difference in the magnitude of disruption.

An alternative explanation, the one we think has more merit, is that the use of visual rather than auditory to-be-remembered items here may have resulted in a less refractory N1 to the irrelevant sounds in the current procedure than in that of Campbell et al.'s Experiment 3a [10]. A possible interpretation is that the N1 in the present study was, therefore, less susceptible to the release from refractoriness associated with enlargements in token set size from 1 to 2, resulting in a less marked behavioural disruption.

Problematic for the token set size based explanation of the irrelevant sound effect is that the greater the acoustic mismatch between successive irrelevant items in a sequence, the greater the disruption. This finding is apparent from studies that have shown that the parametric degradation of sound tokens with noise decreases the extent of disruption, as do decreases in the pitch separation of irrelevant tokens [23,24]. However, the dissimilarity of successive items has also been shown to increase the amplitude of N1 [8,37,40]. In the current study, it is not the acoustic mismatch between successive tokens that was thought to be responsible for the variation in N1 across set size conditions, but the increase in the interval between identical tokens with increases in set size. Whether acoustic mismatch or set size is increased, the outcome is similar: an increase in the amplitude of N1.

A tenable view is that the extent of disruption is determined by the overall sum of N1 amplitudes in response to irrelevant items, which are presented when the brain has a concurrent memory load of to-be-remembered items. This view contrasts with the possibilities that either the detection of change or the detection of tokens determined the extent of disruption produced by irrelevant sound. Rather, it is that the processes generating N1 interfere with the brain activity that supports the memory for the to-be-remembered items. The amplitude of N1 is reliant upon the dynamics of brain mechanisms that are influenced by the preceding acoustic context. The influence that N1 has on memory may accumulate over the period of time when irrelevant sound is presented during concurrent memory load. Accordingly, it is the N1-generating processes, rather than either the cognitive construct of change or token, that mediates the disruption of memory produced by irrelevant sound.

A possibility is that the N1-generating process engaged processes calling for switching of attention to onsets, transitions and energy changes in the acoustic sequence. Although most irrelevant sounds did not elicit a switch of attention, as no P3a was observed, nevertheless, the N1 amplitude may be correlated with the amount of additional processing received by the irrelevant sounds. Serial order memory is assumed to be subject to time-based decay and susceptible to interference by multiple factors in a manner that is apparently probabilistic. Under quiet conditions, recall is thus error prone, as activity generated by multiple processes interfere with this serial order information. The N1 hypothesis suggests that the processes engaged by the output of the N1-generating neuronal populations may share some limited resource with short-term retention of serial order or lists. The discrepancy between the increased N1 from set size 1 to 2 and the lack of a parallel increase in memory disruption may suggest that this factor only plays a role in memory disruption when the N1-related processes engage a substantial part of the shared resource.

One explicit stance of this argument has roots within the primacy model of serial recall [39]. Assume an activation-based system where a node represents a single list item, and that item's position is coded during presentation of items, as a linear function of list position, such that nodes corresponding to earlier items receive more activation than later items. This difference in activation between nodes is the system's memory for serial order. This activation is subject to exponential decay that may be prevented by covert rehearsal. In quiet conditions, zero-mean Gaussian random jitter—which is apparently random but is, in fact, assumed to be influenced by multiple factors deterministically—is added to all nodes' activations. Recall operates by an iterative winner-take-all process that selects the node with the highest activation, which is then suppressed to zero following report of that item. Gaussian jitter may increase or decrease the activity of a node such that an item may be recalled out of turn or incorrectly, because the Gaussian jitter can make the incorrect item have most active node. Recall of serial order may thus be error prone.

Under conditions of irrelevant sound, it is assumed that the operation of the preattentive allocation process can cause a broadening of this Gaussian distribution of jitter, in a manner directly proportional to the magnitude of activity of the N1 generator processes (assuming that the processes engaged by the output of the N1 generator are amongst the factors influencing the activity of the nodes). The difference between node activations may be high enough that broadening the distribution of Gaussian jitter is of little or no disruptive consequence. It is assumed that this is what can often occur under irrelevant conditions when a one-token sequence AAAAAA... is presented and may have also occurred in the case of the two-token sequences of the current experiment. However, with the five-token sequence of the current experiment, it is argued that the distribution of Gaussian jitter became sufficiently broad to disrupt memory for serial order. This description could thus offer an account for the pattern of disruption shown here.

In summary, the present data provided initial consistent evidence with the N1 hypothesis that refractoriness of components of the auditory N1 may be involved in the disruption produced by irrelevant sound. However, even a full correlation between the N1 amplitude and serial recall performance could not have proven a causal relationship and, as was shown by the results, an increase of the N1 amplitude is not always paralleled by a corresponding increase of memory disruption. We have offered admittedly speculative explanations to account for this discrepancy. Thus, the working hypothesis offered here is that if N1

played a role in memory disruption, an overall increase in the N1 amplitude must serve as a necessary rather than a sufficient condition for an increase in disruption.

Acknowledgements

This research has been supported by a Marie Curie Fellowship of the European Community programme "Improving the Human Research Potential and the Socio-Economic Knowledge Base" under contract number HPMF-CT-2000-00902, the Hungarian National Research Fund (OTKA T034112) and the Academy of Finland (200522). Thanks are due to Kati Haataja for the translation of experimental instructions into Finnish, Teemu Peltonen for letting his voice to be recorded and Markus Klaske for his technical assistance.

References

[1] M. Alcaini, M.H. Giard, M. Thévenet, J. Pernier, Two separate frontal components in the N1 wave of the human auditory-evoked response, Psychophysiology 31 (1994) 611–615.
[2] A.D. Baddeley, Working Memory, Oxford Psychol. Series, vol. 11, Oxford University Press, Oxford, 1986.
[3] S.P. Banbury, D.C. Berry, Habituation and dishabituation to speech and office noise, J. Exp. Psychol., Appl. 3 (1997) 1–16.
[4] S.P. Banbury, D.C. Berry, The disruption of office-related tasks by speech and office noise, Br. J. Psychol. 89 (1998) 285–295.
[5] S.P. Banbury, W.J. Macken, S. Tremblay, D.M. Jones, Auditory distraction short-term memory: phenomena and practical implications, Hum. Factors 43 (2001) 12–29.
[6] C.P. Beaman, Computational explorations of the irrelevant sound effect in serial short-term memory, in: L.R. Gleitman, A.K. Joshi (Eds.), Proceedings of the Twenty-Second Annual Conference of the Cognitive Science Society, Erlbaum, Mahwah, NJ, 2000, pp. 37–41.
[7] S. Berti, E. Schröger, A comparison of auditory and visual distraction effects: behavioral and event-related indices, Cogn. Brain Res. 10 (2001) 265–273.
[8] R.A. Butler, Effects of changes in stimulus frequency and intensity on habituation of the human vertex potential, J. Acoust. Soc. Am. 44 (1968) 945–950.
[9] R. Campbell, B. Dodd, Aspects of hearing by eye, in: H. Bouma, D.G. Bounhuis (Eds.), Attention and Performance X, Lawrence Erlbaum Associates, Hove, 1984, pp. 300–311.
[10] T.A. Campbell, C.P. Beaman, D.C. Berry, Auditory memory and the irrelevant sound effect: further evidence for changing-state disruption, Memory 10 (2002) 199–214.
[11] N. Cowan, Converging evidence about information-processing, Behav. Brain Sci. 13 (1990) 237.
[12] N. Cowan, Attention and Memory. An Integrated Framework, Oxford Univ. Press, Oxford, 1995.
[13] W. Ellermeier, K. Zimmer, Individual differences in susceptibility to the "irrelevant speech effect", J. Acoust. Soc. Am. 102 (1997) 2191–2199.
[14] C. Escera, K. Alho, I. Winkler, R. Näätänen, Neural mechanisms of involuntary attention switching to novelty and change in the acoustic environment, J. Cogn. Neurosci. 10 (1998) 590–604.
[15] C. Escera, E. Yago, K. Alho, Electrical responses reveal the temporal dynamics of brain events during involuntary attention switching, Eur. J. Neurosci. 14 (2001) 877–883.
[16] C. Escera, M.J. Corral, E. Yago, An electrophysiological and behavioral investigation of involuntary attention towards auditory frequency, duration and intensity changes, Cogn. Brain Res. 14 (2002) 325–332.
[17] B. Hjorth, Source derivation simplifies topographical EEG interpretation, Am. J. E.E.G. Technol. 20 (1980) 121–132.
[18] M.A. Howard, I.O. Volkov, R. Mirsky, P.C. Garell, M.D. Noh, M. Granner, H. Damasio, M. Steinschneider, R.A. Reale, J.E. Hind, J.F. Brugge, Auditory cortex on the human posterior superior temporal gyrus, J. Comp. Neurol. 416 (2000) 79–92.
[19] R. Hughes, D.M. Jones, The intrusiveness of sound: laboratory findings and their implications for noise abatement, Noise and Health 13 (2001) 51–70.
[20] T. Jacobsen, T.E. Schroger, Is there pre-attentive memory-based comparison of pitch? Psychophysiology 38 (2001) 723–727.
[21] D.M. Jones, Objects, streams and threads of auditory attention, in: A.D. Baddeley, L. Weizkrantz (Eds.), Attention: Selection, Awareness and Control, Oxford Univ. Press, Oxford, England, 1993, pp. 87–104.
[22] D.M. Jones, C.P. Beaman, W.J. Macken, The object-oriented episodic record model, in: S.E. Gathercole (Ed.), Models of Short-Term Memory, Psychol. Press, Hove, 1996, pp. 209–238.
[23] D.M. Jones, D. Alford, A. Bridges, S. Tremblay, W.J. Macken, Organizational factors in selective attention: the interplay of acoustic distinctiveness and auditory streaming in the irrelevant sound effect, J. Exp. Psychol. Learn. 25 (1999) 464–473.
[24] D.M. Jones, D. Alford, W.J. Macken, S.P. Banbury, S. Tremblay, Interference from degraded auditory stimuli: linear effects of changing state in the irrelevant sequence, J. Acoust. Soc. Am. 108 (2000) 1082–1088.
[25] D.C. LeCompte, Irrelevant speech, serial rehearsal, and temporal distinctiveness: a new approach to the irrelevant speech effect, J. Exp. Psychol. Learn. 22 (1996) 1154–1165.
[26] D.C. LeCompte, M.J. Watkins Grouping in primary memory—the case of the compound suffix, J. Exp. Psychol. Learn. 21 (1995) 96–102.
[27] B. Lütkenhöner, O. Steinstrater, High-precision neuromagnetic study of the functional organization of the human auditory cortex, Aud. Neuro-Otol. 3 (1998) 191–213.
[28] W.J. Macken, N. Mosdell, D.M. Jones, Explaining the irrelevant sound effect: temporal distinctiveness or changing state? J. Exp. Psychol. Learn. 25 (1999) 810–814.
[29] M. Martín-Loeches, W. Sommer, Testing models of the irrelevant speech effect on working memory with event-related potentials, Z. Psychol. 206 (1998) 1–22.
[30] M. Martín-Loeches, S.R. Schweinberger, W. Sommer, ERP correlates of irrelevant speech and phonological similarity in auditory–verbal working memory, Hum. Brain Mapp. 1 (1995) 275.
[31] M. Martín-Loeches, S.R. Schweinberger, W. Sommer, The phonological loop model of working memory: an ERP study of irrelevant speech and phonological similarity effects, Mem. Cogn. 25 (1997) 471–483.
[32] C. Miles, D.M. Jones, C.A. Madden, Locus of the irrelevant speech effect in short-term memory, J. Exp. Psychol. Learn. 17 (1991) 578–584.
[33] R. Näätänen, The role of attention in auditory information processing as revealed by event-related potentials and other brain measures of cognitive function, Behav. Brain Sci. 13 (1990) 201–288.
[34] R. Näätänen, Attention and Brain Function, Erlbaum, Hillsdale, NJ, 1992.
[35] R. Näätänen, T.W. Picton, The N1 wave of the human electric and magnetic responses to sound: a review and analysis of the component structure, Psychophysiology 24 (1987) 375–425.
[36] R. Näätänen, I. Winkler, The concept of auditory stimulus representation in cognitive neuroscience, Psychol. Bull. 125 (1999) 826–859.
[37] R. Näätänen, M. Sams, K. Alho, P. Paavilainen, K. Reinikainen, E.N. Sokolov, Frequency and location specificity of the human vertex N1 wave, Electroencephalogr. Clin. Neurophysiol. 69 (1988) 523–553.

[38] I. Neath, Modeling the effects of irrelevant speech on memory, Psychon. Bull. Rev. 7 (2000) 403–423.
[39] M.P.A. Page, D. Norris, The primacy model: a new model of immediate serial recall, Psychol. Rev. 105 (1998) 761–781.
[40] T. Picton, D.L. Woods, G.B. Proulx, Human auditory sustained potentials: I. The nature of the auditory response, Electroencephalogr. Clin. Neurophysiol. 45 (1978) 186–197.
[41] P. Salamé, A.D. Baddeley, Disruptions of short-term memory by unattended speech: implications for the structure of working memory, J. Verbal Learn. Verbal Behav. 21 (1982) 150–164.
[42] M. Scherg, J. Vajsar, T.W. Picton, A source analysis of the late human auditory evoked potentials, J. Cogn. Neurosci. 1 (1989) 336–355.
[43] E. Schröger, A neural mechanism for involuntary attention shifts to changes in auditory stimulation, J. Cogn. Neurosci. 8 (1996) 527–539.
[44] E. Schröger, On the detection of auditory deviations: a pre-attentive activation model, Psychophysiology 34 (1997) 245–257.
[45] E. Schröger, C. Wolff, Attentional orienting and reorienting is indicated by human event-related brain potentials, Neuroreport 9 (1998) 3355–3358.
[46] E. Schröger, C. Wolff, Behavioral and electrophysiological effects of task-irrelevant sound change: a new distraction paradigm, Cogn. Brain Res. 7 (1998) 71–87.
[47] E. Schröger, M.-H. Giard, C. Wolff, Auditory distraction: event-related potential and behavioral indices, Clin. Neurophysiol. 111 (2000) 1450–1460.
[48] S. Tremblay, D.M. Jones, Habituation versus changing state explanations of the irrelevant sound effect: evidence from the effects of token set size, J. Exp. Psychol. Learn. 24 (1998) 659–671.
[49] J. Virtanen, T. Rinne, R.J. Ilmoniemi, R. Näätänen, MEG-compatible multichannel EEG electrode array, Electroencephalogr. Clin. Neurophysiol. 99 (1996) 568–570.
[50] J. Virtanen, J. Ahveninen, R.J. Ilmoniemi, R. Naatanen, E. Pekkonen, Replicability of MEG and EEG measures of the auditory N1/N1m-response, Electroencephalogr. Clin. Neurophysiol. 108 (1998) 291–298.
[51] E. Yago, M.J. Corral, C. Escera, Activation of the brain network of involuntary attention as a function of auditory frequency change, NeuroReport 12 (2001) 4093–4097.
[52] E. Yago, C. Escera, K. Alho, M.-H. Giard, Cerebral mechanisms underlying orienting of attention towards auditory frequency changes, NeuroReport 12 (2001) 2583–2587.
[53] G. Zouridakis, P.G. Simos, A.C. Papanicolaou, Multiple bilaterally asymmetric cortical sources account for the auditory N1m component, Brain Topogr. 10 (1998) 183–189.