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Brain electrical correlates of dimensional weighting: An ERP study

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Abstract

In visual search, there is a reaction time (RT) cost for targets on a given trial if the previous target was defined in a different dimension. According to the "dimension-weighting" account (Müller, Heller, & Ziegler, 1995), limited attentional weight needs to be shifted to the new dimension, resulting in slower RTs. The present study aimed at identifying brain electrical correlates associated with the weight shift. Analyses of ERPs revealed several components to reflect dimension changes whether the task was to detect the target or to identify its defining dimension. N2 amplitudes were more negative whenever the dimension changed. The P3 exhibited latency differences that mirrored RTs in both tasks, but the amplitudes showed no direct relation to stimulus- or response-related processes. Finally, slow-wave amplitudes were enhanced for dimension changes. Taken together, the results provide support for relatively early, perceptual processes underlying dimension change costs.

Descriptors: Attention, Visual search, ERP, Dimension weighting account, N2, P3, SW

One prime function of visual attention is to select relevant information from the huge variety of structures present in the visual field at any one time. Selective attention may be guided bottom-up by salient features in the field, or top-down by the intention to seek particular information relevant to the task at hand. Selective-attention mechanisms can also be differentiated according to the type of information that forms the basis for selection: space-based, object-based, and dimension- (or feature-) based. Space-based theories of attention (e.g., Eriksen & St. James, 1986; Posner, 1980) propose that observers direct (a "spotlight" of) attention to particular locations in space. However, observers can also attend to a particular task-relevant object even if this object shares the same location with another, irrelevant object—which has led to the notion of attentional selection being object based (e.g., Baylis & Driver, 1993; Duncan, 1984). Finally, dimension-based theories of attention (e.g., Allport, 1971; Müller, Heller, & Ziegler, 1995) propose that selection is based on dimensional properties of the objects in the visual field. The latter notion is of special relevance to visual search tasks in which observers have to find a target embedded in an array of irrelevant distractors, with the target being singled out by a unique feature in one dimension or a conjunction of features in separable dimensions. Because dimension-based selection is of special interest for the present investigation, it is considered in more detail below.

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Dimension-Based Visual Selection

Dimension-based theories of visual selection assume that selection is limited by the dimensional nature of the discrimination required to discern response-relevant (target) attributes. A well-supported account has recently been developed by Müller and colleagues (e.g., Found & Müller, 1996; Müller et al., 1995; Müller, Reimann, & Krummenacher, 2003), based on cross-dimensional singleton feature search. In this task, observers have to discern the presence (versus the absence) of an odd-one-out feature target within a field of homogeneous distractors, with the target-defining dimension varying unpredictably across trials (e.g., green vertical distractor bars and target variably defined by color, e.g., *red* vertical bar, or orientation, e.g., green *right-tilted* bar). Performance of this task indicates that the target does not automatically "pop out" of the field of homogeneous distractors based on the operation of some early, saliency-based detection mechanism. Rather, target detection is influenced by an "attentional" mechanism that modulates the processing system by allocating limited "selection weight" to the various dimensions that potentially define the target. Dimensions are assigned weight largely passively, in bottom-up manner: The dimension defining the target on the current trial is allocated a larger weight than alternative dimensions (that may define the target on other trials). However, this weight set may be modified, to some extent, in top-down manner, based on advance information

as to the target-defining dimension on a given trial (Müller et al., 2003).

Two important pieces of evidence for this account are: (1) the observation of a *cross-dimension search cost*, that is, slowed search reaction times (RTs) when the target-defining dimension is variable across trials (e.g., color, orientation) compared to when the target dimension is fixed, but the critical feature is variable within this dimension (for color, e.g., red, blue); (2) the observation of a *dimension-specific intertrial effect* in cross-dimension search, that is, slowed RTs when the target-defining dimension changes on consecutive trials (e.g., when an orientation-defined target follows a color-defined target) compared to when it is repeated. Found and Müller (1996; see also Müller, Krummenacher, & Heller, 2004) showed that this intertrial effect is indeed dimension specific, rather than feature specific, in nature: There is a RT cost only when the target-defining dimension is changed, but not when the critical feature is changed within a constant dimension.

Müller and his colleagues (Found & Müller, 1996; Müller et al., 1995, 2003) took these cross-dimension search cost and dimension-specific intertrial effects as evidence for what they refer to as a "dimension-weighting" account. Similar to visual-search theories such as Guided Search (Cave & Wolfe, 1990; Wolfe, 1994, 1998), it is assumed that attention operates on a master map of integrated saliency signals derived separately in dimension-specific input modules. In contrast to earlier versions of Guided Search, intradimensional saliency processing is "weighted" prior to signal integration by the master map units. The greater the weight assigned to the target-defining dimension, the faster the rate at which evidence for a feature difference within this dimension accumulates at the master map level. When the target-defining dimension on a given trial n is the same as that on the previous trial $n-1$, the weight is already set to the correct dimension, permitting rapid search. In contrast, when the target-defining dimension is changed, a time-consuming "reweighting" process is involved, possibly to determine the dimension defining the target and render it salient at the master map level. This assumes that the target dimension must be weighted to permit target detection (as originally proposed by Müller et al., 1995). Alternatively, a target might also be detected, albeit slower, in a nonweighted dimension and the reweighting follows target detection. Ultimately, the dimension-weighting account is neutral with respect to this issue.

Neural Correlates of Transition Effects

The neural correlates of dimension weighting have been investigated in a set of studies by Pollmann and his colleagues, using event-related functional magnetic resonance imaging (fMRI) (Pollmann, 2004; Pollmann, Weidner, Müller, & von Cramon, 2000, 2006; Weidner, Pollmann, Müller, & von Cramon, 2002). Pollmann and his colleagues identified a fronto-posterior network consisting of a variety of areas that have been reported to be involved in visual search and shifts of visuo-spatial attention. They interpreted the specific activation pattern revealed in prefrontal cortex, increased activation on dimension change relative to no-change trials, as reflecting processes critical for dimensional weight shifting (Pollmann, 2004; Pollmann et al., 2000). Extending the search task from singleton feature to singleton conjunction search, Weidner et al. (2002) found a double dissociation. There was a dimension change-related increase of activation in frontopolar cortex in singleton feature, but not singleton conjunction search. In contrast, there was a dimension change-related activation in pregenual frontomedian cortex in singleton conjunction, but not singleton feature search. This pattern of activations gave rise to the assumption that frontal areas are involved in the *control* of dimensional weight shifting—"automatic" in singleton feature search, "voluntary" in singleton conjunction search—whereas higher level visual areas in superior parietal and temporal cortex mediate the weight shifts via feedback to the dimension-specific input areas in occipital cortex (Pollmann et al., 2006).

Aims and Overview of the Present Experiments

The present study was designed to identify electro-cortical correlates of dimension weighting in cross-dimensional singleton feature search by means of ERP analysis. The fMRI studies reported above provided evidence that anterior brain structures are involved in the attentional weighting of target-defining dimensions. These findings make it likely that ERP correlates of dimensional weighting can be discovered as well, providing insight into the time course of the weighting processes. This was the aim of the present study, which examined ERP components time-locked to the onset of a search display on a given trial n containing a target defined in a particular dimension, contingent on the target-defining dimension on the preceding trial $n-1$. That is, the present study looked for ERP components that systematically vary with changes versus repetitions, across trials, in the target-defining dimension and thus presumably reflect the (re)allocation of attentional weight to relevant dimensions.

According to the dimension-weighting account, a change of the target-defining dimension on consecutive trials would lead to a shifting of attentional weight from the old to the new dimension. Thus, before a weight shift is initiated, a change in the target-defining dimension has to be detected. This process may be associated with systematic variations in the anterior N2 component, which has been shown to reflect the detection of pop-out targets in visual search (Luck & Hillyard, 1994). In a series of experiments, Luck and Hillyard demonstrated that this component was elicited by task-relevant singleton feature "targets" as well as nonrelevant singletons, which they took to "suggest that it may be related to the auditory mismatch negativity (Näätänen, Simpson, & Loveless, 1982)" (p. 305), although it appeared to be modulated by top-down task set. However, Luck and Hillyard did not directly examine repetitions versus changes in the target-defining dimension on consecutive trials, making it difficult to compare their findings with the intertrial effects that were the focus of the present study. A more direct comparison can be made with other investigations that have revealed the N2 to reflect perceptual mismatch or cognitive conflict (Pritchard, Shappell, & Brandt, 1991; Wang, Cui, Wang, Tian, & Zhang, 2004) and the inhibition of overt or covert responses (Kiefer, Marzinzik, Weisbrod, Scherg, & Spitzer, 1998; Pfefferbaum, Ford, Weller, & Koppel, 1985). Thus, the anterior N2 might be a possible indicator of dimension changes in visual search for pop-out targets. Following detection of a change in the target-defining dimension, weight is shifted to the new dimension. This process may be associated with variations in later ERP components such as the P3 or slow wave (SW), though the weight shifting may not have to be completed prior to response execution. In contrast, repetition of the target-defining dimension on consecutive trials might be linked to ERP components preceding the N2, such as the P1-N1 complex, which is thought to reflect early attentional processes (e.g., Luck, Woodman, & Vogel, 2000).

According to the dimension-weighting account, the weight shifting should be reflected in an ERP component *prior* to the initiation of the response. Failure to identify such a component prior to response would support theories that account for dimension change costs in terms of response-related processes (e.g., Cohen & Magen, 1999; Mortier, Theeuwes, & Starreveld, 2005). Thus, in addition to identifying ERP components associated with attentional weight shifting, the time course of the ERP can provide new insights into the controversial issue of the point in time, and stage of processing, at which the weight adjustment occurs.

These questions were examined in two experiments that adapted the two singleton feature search tasks used by Found and Müller (1996) for EEG recording. In both experiments, the target on a given trial differed from the distractors in either color or orientation. In Experiment 1 (with 30% target-absent trials), observers were required to simply respond "target present" or "absent" (target present/absent discrimination); in Experiment 2 (with target-present trials only), observers had to explicitly indicate the target-defining dimension (color/orientation-target discrimination). These tasks were compared to examine the relation of dimensional-weight shifting to target detection and (dimensional) identification, respectively. Müller et al. (1995; see also Müller et al., 2004) argued that target detection requires at least implicit knowledge, that is, attentional weighting, of its defining dimension, whereas explicit identification of this dimension involves an extra, time-consuming process, that is, focal-attentional analysis of the type of feature contrast generated by the target (according to Müller et al., simple detection responses can be initiated prior to target analysis). If this is correct, then no differences in ERP components reflecting weight shifting should be observed between the simple target detection (Experiment 1) and the explicit identification task (Experiment 2). In contrast, if processing differed fundamentally between the two tasks, systematic differences in ERP effects should be observed.

EXPERIMENT 1

Method

Participants

Eleven observers (7 female) took part in Experiment 1. One observer had to be excluded from the analyses of ERPs, due to excessive artifacts. The ages of the resulting 10 observers ranged from 20 to 28 years ($X = 25.7$, $SD = 2.5$ years). Observers were either paid or received course credit for participating. All participants were right-handed, had normal or corrected-to-normal vision, and reported no history of neurological disorder.

Stimuli and Procedure

The experiment was conducted in a dimly illuminated, sound-attenuated, and electrically shielded chamber. A 21-in. display monitor was placed 110 cm in front of the observer, with the central fixation cross aligned with the observer's horizontal straight-ahead line of sight. Each trial started with a central asterisk presented for 500 ms. This was followed by the search display, which consisted of 18 elements presented below the fixation marker and remained in view until the observer reacted. Distractor elements in the search display were green vertical bars, the singleton target element was either a red or a blue vertical bar (color-defined targets) or a 45° left- or right-tilted green bar (orientation-defined targets). Targets could appear, unpredictably on a trial, at one of four possible locations (two to the left and two to the right) of the fixation marker. Search displays contained a target on 70% (and no target on 30%) of the trials, with targets positioned equally likely to the left and right of the fixation. Observers were instructed to press a button with the index finger of one hand to respond "target present" and with the index finger of the other hand to respond "target absent." Responses were to be made as fast and accurately as possible. After an intertrial interval of 1000 ms, the next trial was initiated. After half of the experiment, the response assignment was reversed.

The order of target-defining dimensions (and features) on consecutive trials was pseudorandomized, to ensure comparable numbers of trials with dimension (and feature) repetitions and changes across trials. There was a total of 360 trials with repeated color targets (Color same Dimension, CsD), 178 trials with a repetition of target's color feature (e.g., red–red; Color: same Dimension same Feature, CsDsF), and 182 trials with a color feature change (e.g., red–blue; Color: same Dimension different Feature, CsDdF). Similarly, there was a total of 358 trials with repeated orientation targets (Orientation same Dimension, OsD), 182 trials with a repetition of the target's orientation feature (e.g., left-tilted–left-tilted; Orientation: same Dimension same Feature, OsDsF), and 176 trials with an orientation feature change (e.g., left-tilted–right-tilted; Orientation: same Dimension different Feature, OsDdF). Further, on 194 trials, the dimension changed from orientation to color on consecutive trials (Color: different Dimension, CdD), and on 194 trials, it changed from color to orientation (Orientation: different Dimension, OdD).

EEG Recordings

The electroencephalogram (EEG) was recorded continuously, at a sampling rate of 500 Hz, using 64 Ag/AgCl electrodes including those corresponding to the 10-10 system (American Electroencephalographic Society, 1994). The electrodes were mounted on an elastic cap (Easy Cap, Falk Minow Services). Vertical and horizontal eye movements were monitored by means of electrodes placed at the outer canthi of the eyes and the superior and inferior orbits. Electrophysiological signals were amplified using a 0.1–100-Hz bandpass filter via BrainAmps (BrainProducts, Munich). All electrodes were referenced to Cz and re-referenced off-line to linked mastoids. ERPs were averaged off-line over a 1000-ms epoch relative to a 200-ms prestimulus baseline. Eye movements were corrected by means of independent component analyses (ICA) implemented in the Brain Vision Analyzer software (Brain Products, Munich). Epochs with artifacts, that is, excessive peak-to-peak deflections (> 100 μV or < -100 μV), bursts of electromyographic activity (permitted maximal voltage step/sampling points 50 μV), and activity lower than 0.5 μV within intervals of 500 ms (indicating "dead channels" in the montage), were excluded from averaging on an individual-channel basis.

Following the elimination of artifacts, latencies of the P1, N1, N2, P3, and SW components were determined as the maximum deflection within the time windows derived by visual inspection of the grand average potentials (see Table 1). After identification of component latencies, mean amplitudes were calculated using the time windows specified in Table 1. Note that only trials n with a correct response, following trials $n-1$ with a correct response, were included in the analyses.

Amplitudes and latencies were analyzed by repeated-measures ANOVAS with the factors target Dimension (color vs.

Table 1. *Experiment 1: Time Windows for Calculating Mean Amplitudes of ERP Components at Various Recording Sites and Latency Windows for Determining Peak Latency of ERP Components at the Corresponding Sites*

Component	Mean time window	Latency window	Recording site (left, midline, right)
P1	50–90 ms	40–100 ms	frontal, central, parietal, occipital
N1	115–155 ms	100–170 ms	frontal, central, parietal, occipital
N2	250–00 ms	220–330 ms	frontal, central, parietal, occipital
P3	340–380 ms	320–420 ms	frontal, central, parietal, occipital
Slow wave	420–600 ms	—	frontal, central, parietal, occipital

orientation), Intertrial Transition (same feature, different feature, different dimension), Electrode Position (electrode sites over the left, the midline, and the right hemisphere), and the topographical distribution of the electrode indicated by the factor Anterior/Posterior (frontal, central, parietal, and occipital). Whenever required, significant main effects and interactions were further examined using Tukey HSD post hoc contrasts.

Results

Behavioral Data

Overall, 1.2% of all trials resulted in misses and 1.8% in false alarms indicative of no speed accuracy trade-off. Figure 1 presents the correct detection (target present) RTs dependent on the cross-trial transition (same Dimension same Feature sDsF, same dimension different feature sDdF, different dimension dD), separately for color- and orientation-defined targets. A repeated-measures ANOVA with the factors Dimension (color vs. orientation) and Transition (sDsF, sDdF, dD) revealed both main effects to be significant, $F(1,9) = 42.06$, $p < .0001$, and, respectively, $F(2,18) = 65.89$, $p < .0001$. The interaction was not significant, $F(2,18) = .832$, $p < .45$. Color-defined targets were responded to overall faster than orientation targets (382.5 vs. 414.2 ms). More importantly, the pattern of intertrial transition

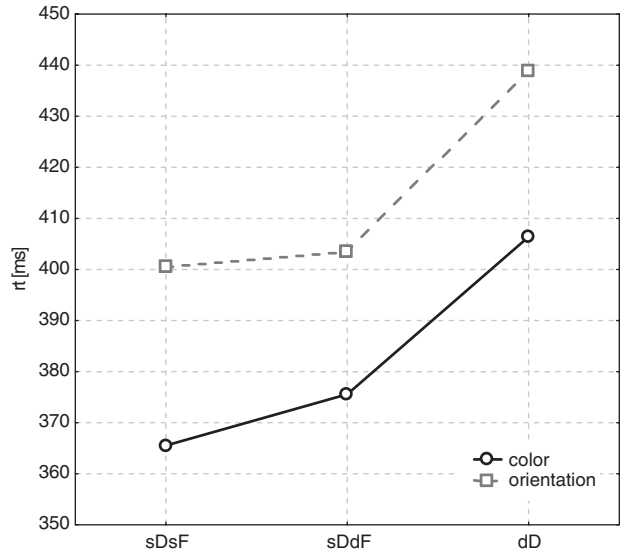

Figure 1. Detection task: Mean reaction times to color and, respectively, orientation targets on trial n dependent on the identity of the target on trial $n-1$: same-dimension same-feature (sDsF), same-dimension different-feature (sDdF), and different-dimension (dD). The black solid line indicates reaction times to color targets, the gray dashed line reaction times to orientation targets.

effects replicated the pattern observed by Found and Müller (1996): There was a significant RT cost for changes, relative to repetitions, of the target-defining dimension across trials (39.6-ms cost for dD vs. sDsF; $p < .0002$), whereas there was no significant cost for feature changes, relative to repetitions, within a repeated dimension (6.5-ms cost for sDdF vs. sDsF; $p < .22$).

Electrophysiology

Figure 2 displays the grand average waveforms (collapsed over color and orientation targets) with the onset of same- and different-dimension targets on trial n, dependent on the target-defining dimension on trial $n-1$, for selected electrode locations.

As can be seen from Figure 2, target display onset was associated with a pronounced negative shift in the time range of the N2 at frontal and, less marked, central leads. A late positive complex revealed differences between same- and different-dimension targets dependent on the target-defining dimension on the previous ($n-1$) trial at posterior electrodes. Analyses of the various components showed the factor Intertrial Transition to have a significant effect on the N2, P3, and SW components. For all analyses, only main effects and significant interactions involving the factor Transition will be reported.

P1. Mean amplitudes and latencies of the P1 component were examined by repeated-measures ANOVAs with the factors Dimension (color, orientation), Transition (sDsF, sDdF, dD), Electrode Position (left, midline, right), and Anterior/Posterior (frontal, central, parietal, occipital). The ANOVA of the P1 amplitudes failed to reveal any significant effects of Transition (main effect: $F[2,18] = 2.48$, $p > .112$; interactions involving Transition, all $p > .12$), as did the ANOVA of the P1 latencies.

N1. Analogous ANOVAs of the mean amplitudes and latencies of the N1 component also failed to reveal repetition versus change of the target-defining dimension (or respectively, feature) to have a significant effect on either the amplitudes (main effect: $F[2,18] = 0.67$, $p > .936$; interactions involving Transition: all $p > .152$) or the latencies. The latency ANOVA revealed only a marginal Transition × Dimension interaction, $F(2,18) = 3.12$, $p > .068$, with shorter onset latencies following a repetition rather than a change in the target-defining dimension for color targets, but not for orientation targets (though post hoc contrasts revealed none of the comparisons to be significant, all $p > .26$).

N2. Similar to the above analyses, the mean amplitudes of the N2 component were examined by an ANOVA with the factors Dimension, Transition, Electrode Position, and Anterior/Posterior. This ANOVA revealed the main effect of Transition to be significant, $F(2,18) = 6.96$, $p < .021$, with changes in the target-defining dimension giving rise to a more negative-going deflection of the N2 (with 2.2 μV, 1.9 μV, and 1.5 μV for same feature, different feature, and different dimension trials averaged

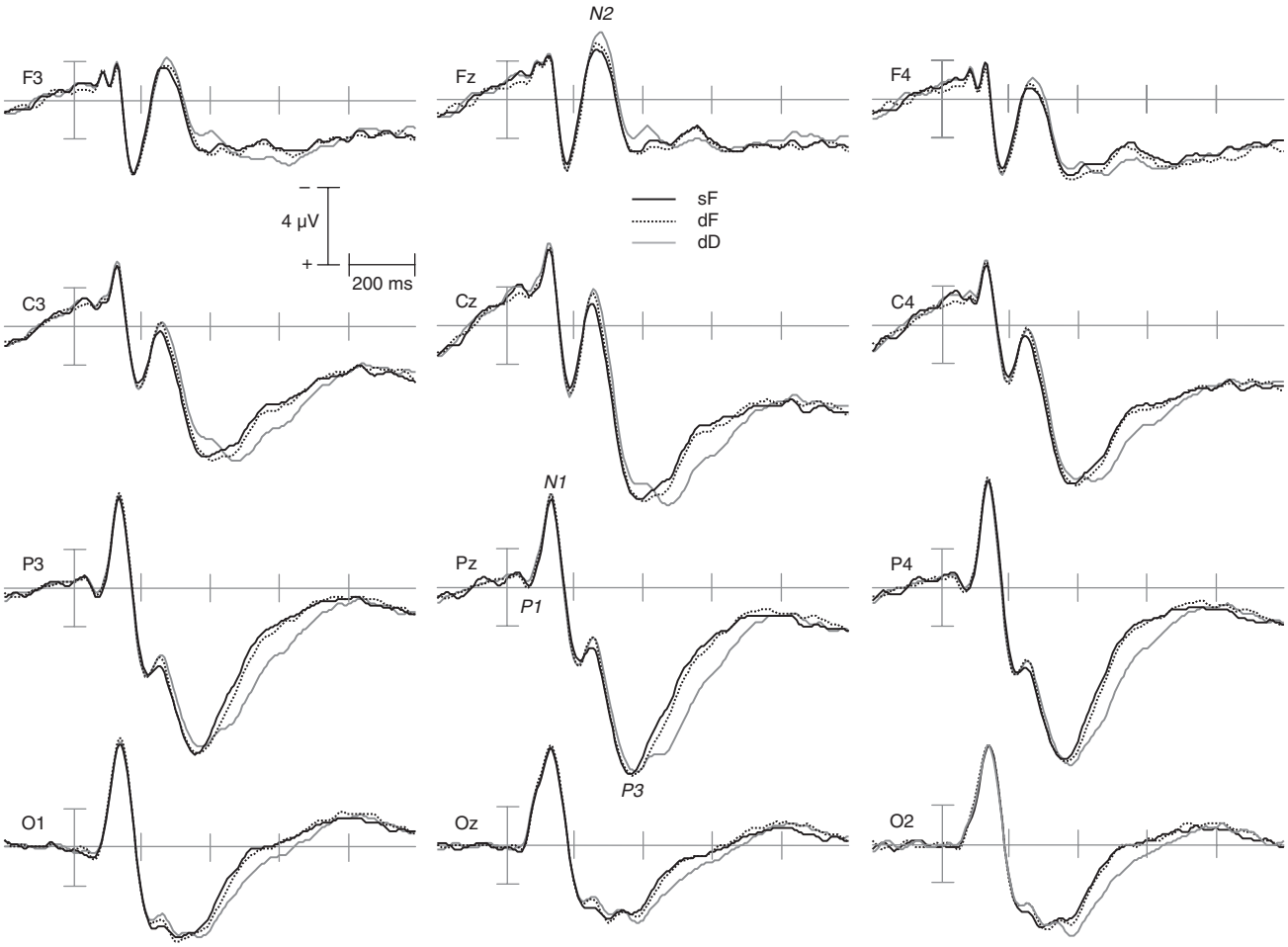

Figure 2. Detection task: Grand average waveforms elicited with onset of the target display on trial *n* dependent on the identity of the target on trial $n-1$, for selected electrode positions. Black solid lines indicate same-dimension same-feature trials (sDsF), black dotted lines same-dimension different-feature trials (sDdF), and gray solid lines different-dimension trials (dD). Averages were collapsed across color and orientation targets, as the Dimension × Transition interaction was nonsignificant. Negativity is plotted upward, and the data are presented relative to a 200-ms prestimulus baseline. Components labeled in italics are the N2 at Fz and the P1, N1, and P3 at Pz.

over all electrode sites, respectively). Furthermore, the main effect of Electrode Position was significant, $F(2,18) = 4.84$, $p < .021$, with strongest negativities at midline electrodes (1.17 μV) as compared to left- and right-lateral recording sites (1.97 μV and 2.50 μV, respectively). The main effect of Anterior/Posterior was also significant, $F(3,27) = 6.38$, $p < .019$, with the strongest negative deflection over frontal sites (− 1.63 μV) and decreasing negativity for central and parietal recording sites (with 0.81 μV and 4.64 μV, respectively). As compared to parietal leads, a relative stronger negative deflection was again recorded over occipital leads (3.70 μV). These main effects were qualified by significant interactions of Transition × Electrode Position, $F(4,36) = 2.73$, $p < .044$, and Transition × Electrode Position × Anterior/Posterior, $F(12,108) = 3.15$, $p < .021$. The strongest negative deflections were observed at frontal electrodes, with a maximum over the frontal midline (Fz) recording site (− 2.69 μV). The difference between same- and different-dimension trials was still pronounced at central midline electrodes and decreased toward posterior sites. The three-way interaction was due to decreasing differences between same- and different-dimension trials from left-occipital leads to midline and right-occipital recording sites.

An analogous ANOVA of the N2 latencies revealed a marginally significant Dimension × Transition × Anterior/Posterior interaction, $F(6,54) = 3.03$, $p < .052$, with increasing latency differences between color and orientation targets from frontal toward occipital leads. Orientation targets elicited an earlier N2 onset than color targets, irrespective of whether or not there was a dimension change, at all electrode locations—except for frontal sites. Here, at the maximum of the N2, earlier onset latencies for color compared to orientation targets were exhibited for same feature trials, but the inverse amplitude pattern was found for different feature and dimension change trials.

Topography of N2 Effect

To further explore the topography of the dimension change effect, difference waves were computed by subtracting same-dimension from different-dimension trial waveforms.[1] Figure 3

[1]Note that, because there were no significant differences in N2 amplitudes between color- and orientation-defined targets, the time course of activity was aggregated across the two dimensions; similarly, because there were no differences between same- and different-feature trials in the absence of a dimension change, both types of trial were aggregated in the condition "same dimension."

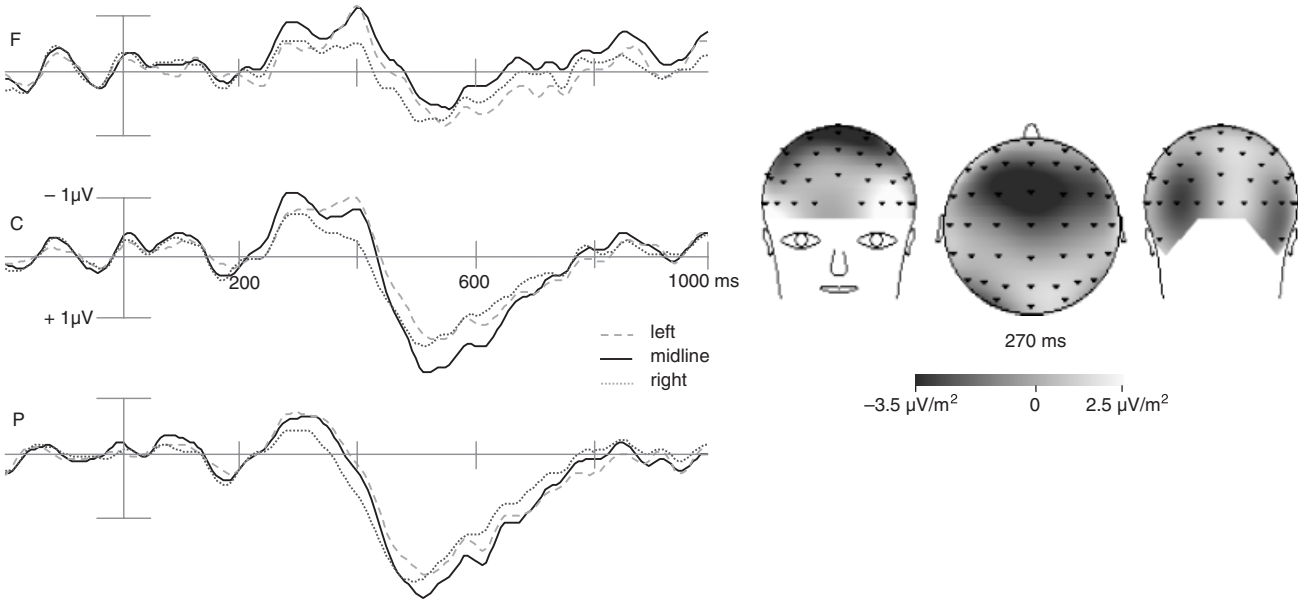

Figure 3. Detection task—Left side: Different-dimension minus same-dimension difference wave forms for frontal (F), central (C), and parietal (P) electrode positions. Gray broken lines depict left-lateral, black solid lines midline, and gray dotted lines right-lateral electrode positions. Right side: Current density distribution of the dimension change effect computed for the difference wave forms at 270 ms.

presents the resulting difference waves and the current source density map for the difference wave at 270 ms after target display onset.

To examine whether the change effect was lateralized, difference wave amplitudes (mean amplitudes for the time range 270 ± 30 ms) were examined by a repeated-measures ANOVA with the factors Dimension, Electrode Position (left, midline, right), and Anterior/Posterior (frontal, central, parietal, occipital). The results revealed the main effect of Electrode Position to be significant, $F(2,18) = 5.35$, $p < .033$, with the strongest effect of dimensional repetition versus change at midline electrodes. Furthermore, the Electrode Position × Anterior/Posterior interaction was significant, $F(6,54) = 3.30$, $p < .008$. At frontal and central sites, left- and right-lateral amplitudes did not differ (post hoc contrasts, all $p > .99$). Difference wave amplitudes at frontal midline electrodes were significantly more negative than left- and right-lateral amplitudes ($p < .01$), but amplitudes at central midline sites did not differ significantly from central left- and right-lateral recording sites ($p > .92$). There were no differences among any electrode positions at parietal and occipital electrode locations (all $p > .36$). This pattern is consistent with a frontal maximum, without lateralization of the N2 component in the detection task.

P300. The mean amplitude of the P3 over the time window 340–380 ms after target display onset was examined by a repeated-measures ANOVA with the factors Dimension, Transition, Electrode Position (left, midline, right), and Anterior/Posterior (frontal, central, parietal, occipital). This ANOVA revealed significant main effects of Electrode Position, $F(2,18) = 3.676$, $p < .046$, and Anterior/Posterior, $F(3,27) = 8.865$, $p < .006$, as well as significant interactions of Dimension × Transition, $F(2,18) = 5.593$, $p < .013$, Transition × Electrode Position, $F(4,36) = 4.109$, $p < .006$, and Dimension × Transition × Anterior/Posterior, $F(6,54) = 3.328$, $p < .051$. Maximum amplitudes of the P3 were located over parietal midline electrodes and revealed more positive-going deflections over the right as compared to the left hemisphere.

The influence of the factors Dimension and Transition at the parietal maximum of the P3 deflection was examined further by an ANOVA with the factors Dimension, Transition, and Electrode Position (left, midline, right). This ANOVA revealed the interaction of Transition × Electrode Position to be significant, $F(4,36) = 4.927$, $p < .014$. Post hoc contrasts revealed a significant difference between feature repetitions and changes of the target-defining dimension ($p < .017$) with more positive-going P3 amplitudes for dimension change trials (8.94 μV) as compared to feature repetition trials (8.58 μV). No difference between feature repetitions and changes ($p < .37$) or feature repetitions and dimension changes ($p < .88$) were observed at right-parietal electrode sites. While the strongest positive deflections were observed over parietal midline electrodes, no significant effects of dimension repetitions versus changes were found for left- and midline-parietal sites (all $p > .56$).

An analogous ANOVA of the P3 latencies revealed only the main effects of Transition, $F(2,18) = 25.79$, $p < .001$, and Anterior/Posterior, $F(3,27) = 5.55$, $p < .026$, to be significant. The P3 had an earlier onset for same-dimension (i.e., same- and different-feature) trials (365 and 369 ms, respectively) compared to different-dimension trials (393 ms). Repetition of the target-defining dimension led to comparable onset latencies of the P3, whether or not the target feature was repeated ($p < .56$). In contrast, changes of the target-defining dimension were associated with significantly longer P3 latencies (all $p < .001$). P3 latencies did not differ significantly at frontal, central, and parietal recording sites (with 391, 397, 372 ms, respectively; all $p > .56$). But the P3 showed a significantly earlier onset at occipital (342 ms) as compared to all other recording positions ($p < .014$).

Slow wave. Amplitudes in the slow-wave window were examined by a repeated-measures ANOVA with the factors Dimension, Transition, Electrode Position (left, midline, right), and

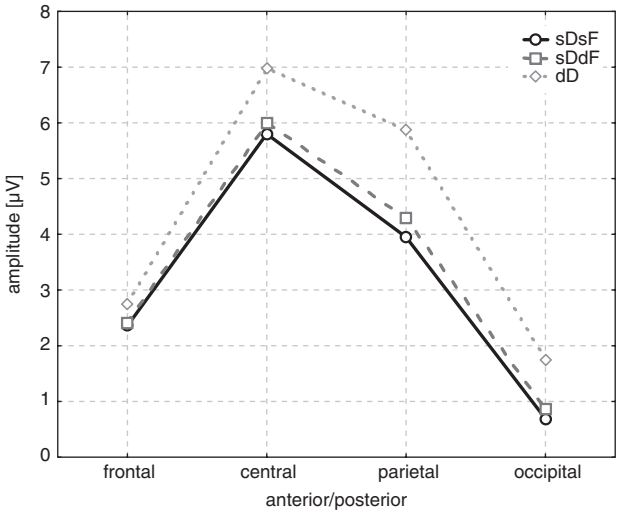

Figure 4. Detection task: Mean slow wave amplitudes from 420 to 600 ms after display onset as a function of Anterior/Posterior electrode position (frontal, central, parietal, occipital), separately for the three intertrial Transition conditions: same-dimension same-feature (sDsF), same-dimension different-feature (sDdF), and different-dimension (dD).

Anterior/Posterior (frontal, central, parietal, occipital). This analysis revealed the main effects of Transition, $F(2,18) = 12.398$, $p < .004$, and Anterior/Posterior, $F(3,27) = 7.689$, $p < .011$, as well as the interaction of Transition × Anterior/Posterior, $F(6,54) = 9.37$, $p < .001$ (see Figure 4) to be significant.

Slow-wave amplitudes were enhanced for different-dimension as compared to same-dimension (i.e., same- and different-feature) trials. Post hoc contrasts revealed significant differences between same-dimension trials (irrespective of a repetition/change of the target feature) and different-dimension trials at central, parietal, and occipital sites (all $p < .003$). For same-dimension trials, there were no significant differences between feature changes and repetitions at these locations ($p < .78$). In contrast to the central, parietal, and occipital sites, there were no differences between same- and different-dimension trials at frontal electrodes (all $p > .34$). The maximum absolute slow-wave deflection was located over central sites, with a nonsignificant decrease toward parietal locations ($p < .55$) and significantly less pronounced deflections over frontal and occipital leads (all $p < .03$). The largest amplitude difference between same- and different-dimension trials was observed over parietal leads.

The largest Transition effect at parietal electrodes was examined further by an ANOVA with the factors Dimension, Transition, and Electrode Position (left, midline, right). This ANOVA revealed all three factors to have a significant influence on slow-wave amplitude (all $p < .032$), including a significant interaction of Transition × Electrode Position, $F(4,36) = 2.908$, $p < .035$. There were more positive-going deflections for orientation targets, with the strongest amplitude overall recorded at the parietal midline. Same- and different-feature trials did not differ significantly in slow-wave amplitude ($p < .59$), whereas both differed compared to different-dimension trials ($p < .001$). There were no differences in slow-wave amplitude for same- and different-feature trials at left- and right-parietal electrode locations (all $p < .97$), but significant differences between both lateral recording sites and the midline position (all $p < .001$). The interaction was due to a decreasing effect of dimension changes from left- to right-parietal recording sites for different-dimension trials.

Discussion

The RT data replicated the findings of Found and Müller (1996). There were general RT advantages for targets defined in the color dimension. However, for both color and orientation targets, RTs were markedly slower when the target-defining dimension changed across trials, whereas there were no RT differences between same-dimension trials with and without a change in the target feature. This pattern of effects is consistent with the notion that attentional weights are assigned to target dimensions rather than features and that a dimension change requires (or is associated with) the shifting of attentional weight from the old to the new target-defining dimension.

The missing influence of dimension repetition versus change on event-related P1 and N1 is consistent with the assumption (e.g., Hillyard & Anllo-Vento, 1998) that these early components are associated with perceptual processing within the focus of attention, in particular, when focal attention is allocated in advance to a circumscribed display region where a target appears later. In contrast, these components are not significantly modulated when the display is processed in parallel to discern the presence of a feature contrast, that is, prior to the allocation of focal attention to a selected location.

The systematic pattern of RT effects was mirrored by effects in the fronto-centrally distributed N2 component of the visually evoked potential. Changes in the target-defining dimension were associated with stronger negative-going deflections in the time range 250–300 ms. Conversely, the negativities were less pronounced with repetitions of the target-defining dimension, whether or not the target feature changed (although there appeared to be some differences for feature changes within dimensions, these failed to reach significance—as with the RT data).

The systematic pattern of N2 amplitude effects might be taken as evidence of an additional process that comes into play only when the target-defining dimension changes on consecutive trials. This pattern is consistent with the dimension-weighting account, which assumes that, when the target-defining dimension changes from trial $n-1$ to trial n, limited attentional weight has to be shifted to the new dimension. Increased negativities of the N2 therefore might be interpreted as being associated with the detection of a change in the relevant dimension, which signals that a new dimensional weight set (assigning greater weight to the new dimension for upcoming trials) is required. The change effect, as reflected in the difference waves (same-dimension trials subtracted from different-dimension trials), revealed a frontal distribution. This is in line with several studies that have reported a frontally distributed effect of "difference detection" (e.g., Näätänen, 1990; Wang et al., 2004) or a prefrontal effect reflecting response-independent inhibition-related executive functions (Kiefer et al., 1998).

The latency of another component of the ERP, the P3, showed a systematic relation to the RT pattern of effects. However, the P3 falls within a time window that involves several processes, some of which are associated with response requirements. Thus, any interpretation of the P3 effects must consider several underlying processes. One tentative interpretation might be that, after the detection of a change of the target-defining dimension, as reflected by increased negativities of the N2

component, attentional weights have to be shifted. The time-consuming redistribution of the dimensional weights might contribute to the P3 pattern in the present investigation, in line with the observed latency pattern for the P3 over parietal recording sites: prolonged onset latencies for a change of the target-defining dimension compared to a repetition, irrespective of target feature changes/repetitions within the repeated dimension.

Finally, the slow wave (SW) exhibited a systematic variation that mirrored the RT pattern. The strongest effect of dimension change was observed over parietal leads, with a midline maximum. However, dimension changes significantly influenced slow-wave amplitudes at all posterior recording sites. This pattern started over central sites and continued over parietal to occipital sites, revealing a widespread effect of changes in the target-defining dimension.

The topography of the N2 modulations on dimension change trials is consistent with the results of Pollmann et al. (2000), who used fMRI to study the neural correlates of dimension weighting. Pollmann et al. interpreted the specific activation pattern revealed in frontal cortex as reflecting a critical process in dimensional weight shifting: the detection of environmental change that requires the reallocation of dimension-specific processing resources (see also Pollmann, 2004). In line with these findings, the topography of the N2 modulation revealed in the present study points to a generator in frontal cortex. This is also consistent with a study by Kiefer et al. (1998), reporting an enhanced N2 component in a go/no-go task that was largely independent of motor-related processes and taken to reflect higher level executive functions. Dipole reconstruction pointed to bilateral generators within the inferior prefrontal area. However, without reconstructing the sources of the present data, the assumption of frontal generators underlying the observed N2 pattern remains tentative.

In addition to the study of Kiefer and colleagues reported above, the present N2 modulation occurred within the time range of other negative components that reflect perceptual mismatch or cognitive conflict (Error Related Negativity, ERN, e.g., Falkenstein, Hohnsbein, Hoormann, & Blanke, 1990; Mismatch Negativity, MMN, e.g., Näätänen, 1990; Mismatch N2, e.g., Pritchard et al., 1991; Wang et al., 2004). In the present task, this might be the detection of a change in the target-defining dimension, signaling the need to redistribute the attentional weight to the new dimension.

If this is correct, the same pattern of N2 and P3 amplitude effects should be observed in Experiment 2, in which observers were required to explicitly discriminate the target-defining dimension, giving a "color" versus an "orientation" response. Experiment 2 was expected to confirm the pattern of N2 modulations, as an indicator for the detection of changes in the target-defining dimension. Furthermore, the pattern of N2, P3, and SW effects were expected to shed light on the question of whether (implicit) knowledge of the dimensional identity of the target is required to detect its presence. If so, the patterns of ERP components were expected to be comparable in the two experiments.

EXPERIMENT 2

Method

Participants

Twelve subjects (7 female) took part in Experiment 2; 3 of the 12 observers had already taken part in Experiment 1. One observer had to be excluded from the ERP analyses due to excessive artifacts. The resultant 12 observers ranged in age from 22 to 32 years ($X = 27.08$ years, $SD = 2.54$). All subjects were right-handed, had normal or corrected-to-normal vision, and reported no history of neurological disorder.

Stimuli and Procedure

The procedure was the same as that of Experiment 1, except that a target was present on all trials. Observers had to respond to color-defined targets (whether red or blue) with the index finger of one hand and to orientation targets (whether left- or right-tilted) with the index finger of the other hand, with hand counterbalanced across observers. After half the experiment, the response assignment was reversed.

The order of target dimensions on consecutive trials was pseudorandomized to assure approximately comparable number of dimension repetition and change trials. There were 506 trials in total with repeated color-defined targets (Color same Dimension, CsD), with a feature repetition (e.g., red–red) on 248 trials and a feature change (e.g., red–blue) on 258 trials. And there were 500 trials with repeated orientation-defined targets (Orientation same Dimension, OsD), with a feature repetition (e.g., left-tilted–left-tilted) on 248 trials and a feature change (e.g., left-tilted–right-tilted) on 252 trials. On 488 and 486 trials, the target-defining dimension changed from orientation to color (Color different Dimension, CdD) and, respectively, from color to orientation (Orientation different Dimension, OdD).

Data Processing

Manual response and EEG data recording was the same as in Experiment 1. For Experiment 2, amplitudes and latencies of the P1, N1, N2, and P3 components were derived from visual inspection of the grand average waveforms as maximum deflection within the time windows specified in Table 2. The maximum deflection within the defined time ranges was defined as the component's latency. Only trials with correct reaction, following a trial with a correct reaction, were included in the analyses.

Table 2. *Experiment 2: Time Windows for Calculating Mean Amplitudes of ERP Components at Various Recording Sites and Latency Windows for Determining Peak Latency of ERP Components at the Corresponding Sites*

Component	Mean time window	Latency window	Recording site (left, midline, right)
P1	50–90 ms	40–100 ms	frontal, central, parietal, occipital
N1	115–155 ms	100–170 ms	frontal, central, parietal, occipital
N2	250–300 ms	220–330 ms	frontal, central, parietal, occipital
P3	340–380 ms	320–420 ms	frontal, central, parietal, occipital
Slow wave	420–600 ms	—	frontal, central, parietal, occipital

Results

Behavioral Data

Overall, 3.9% incorrect reactions were recorded (4.1% and 3.5% for color and orientation targets, respectively). The RT results were again consistent with the general pattern of effects reported by Found and Müller (1996): costs for changes, relative to repetitions, of the target-defining dimension, but little costs for changes, relative to repetitions, of the target-defining feature within a constant dimension. In contrast to Experiment 1 (detection task), a repeated-measures ANOVA with the factors Dimension (color, orientation) and Transition (same feature, different feature, different dimension) failed to reveal a main effect of Dimension, $F(1,10) = .623$, $p < .448$. However, as in Experiment 1, the main effect of Transition was significant, $F(2,20) = 16.84$, $p < .0001$, though there was also a significant Dimension × Transition interaction, $F(2,20) = 21.56$, $p < .0001$.

This interaction, which is illustrated in Figure 5, was due to orientation targets showing only a dimension-specific effect (i.e., increased RTs for different-dimension targets relative to different-feature targets), but no feature-specific change effect (i.e., no increased RTs for different-feature relative to same-feature targets; $p < .75$). In contrast, color targets showed both a dimension-specific (dD vs. sDdF, $p < .0001$) and a feature-specific change effect (sDdF vs. sDsF, $p < .0001$).

Electrophysiology

Figure 6 presents the ERPs with onset of the search display, collapsed over orientation and color targets. As in Experiment 1, there were no effects of the factor Transition for the early P1 and N1 components; however, the N2, P3, and SW components exhibited systematic variations with changes versus repetitions of the target-defining dimension across trials. For all analyses, only main effects and significant interactions involving the factor Transition are reported.

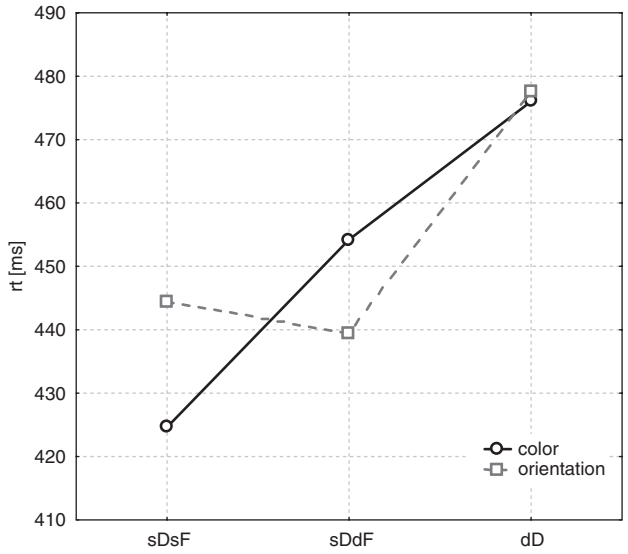

Figure 5. Discrimination task: Mean reaction times to color and, respectively, orientation targets on trial n dependent on the dimensional identity of the target on trial $n-1$: same-dimension same-feature (sDsF), same-dimension different-feature (sDdF), and different-dimension (dD). The black solid line indicates reaction times to color targets, the gray dashed line reaction times to orientation targets.

P1 and N1. The mean amplitudes of the P1 and N1 components were examined by repeated-measures ANOVAs with the factors Dimension (color, orientation), Transition (sDsF, sDdF, dD), Electrode Position (left, midline, right), and Anterior/Posterior (frontal, central, parietal, occipital). There were no significant effects of Transition for either P1 amplitudes (main effect: $F[2,20] = 1.29$, $p > .297$; interactions involving Transition: all $p > .20$) or N1 amplitudes (main effect: $F[2,20] = 1.61$, $p > .225$; interactions involving Transition all: $p > .10$). Analogous ANOVAs of the onset latencies of the P1 and N1 also failed to reveal any significant effects involving Transition.

N2. The mean amplitude of the N2 component was examined in a repeated-measures ANOVA with the factors Dimension, Transition, Electrode position (left, midline, right), and Anterior/Posterior (frontal, central, parietal, occipital). This ANOVA revealed the main effects of Transition, $F(2,20) = 3.88$, $p < .038$, Electrode Position, $F(2,20) = 10.81$, $p < .005$, and Anterior/Posterior, $F(3,30) = 9.78$, $p < .003$, to be significant. These were qualified by interactions of Dimension × Transition, $F(2,20) = 3.98$, $p < .035$, Transition × Electrode Position, $F(4,40) = 2.72$, $p < .043$, and Transition × Electrode Position × Anterior/Posterior, $F(12,120) = 3.32$, $p < .023$. Similar to Experiment 1, a change in the target-defining dimension resulted in a more negative-going deflection in the N2 range at frontal sites, compared to a repetition of the target dimension (main effect of Transition). This effect was strongest over frontal midline sites and decreased toward posterior sites. At frontal midline recordings, different-dimension trials exhibited significantly larger negative deflections compared to same-dimension trials, that is, relative to both same- and different feature trials (both $p < .001$), which did not differ between themselves ($p > .1$). The same pattern of effects was observed for right- and left-frontal electrode locations. Post hoc contrasts revealed N2 amplitudes for different-dimension trials at frontal sites to be significantly different relative to same-dimension, that is, both same- and different-feature trials ($p < .007$ and $p < .026$, respectively), without the latter showing a difference ($p < .082$). The additional main effect of Electrode Site confirmed a fronto-central topography of this N2 effect, $F(2,20) = 13.111$, $p < .001$.

Furthermore, there was a marginally significant interaction among all four factors, $F(12,120) = 2.41$, $p < .060$, reflecting differential activation patterns between color and orientation targets defined by the same and, respectively, different features within a repeated dimension at central, parietal, and occipital, but not frontal sites for the three electrode locations (see Figure 7).

For color targets, same- and different-feature trials did not differ at frontal sites, but both types of trial differed compared to dimension changes. The same pattern of frontal-site effects was evident for orientation targets: no differences between same- and different-feature trials, but significantly enlarged N2 amplitudes for different-dimension trials. At central, parietal, and occipital sites, the two dimensions showed slightly diverging patterns of effects: Color targets gave rise to stronger negative deflections at these electrode locations on different-dimension relative to same-dimension trials, and at parietal sites, there were significant differences between same-feature trials and the two other conditions (different feature and different dimension). In contrast, there were no differences between different-feature and different-dimension trials for orientation targets, neither at central, parietal, nor occipital sites. For color targets no differences between same- and different-feature trials at occipital leads were evident.

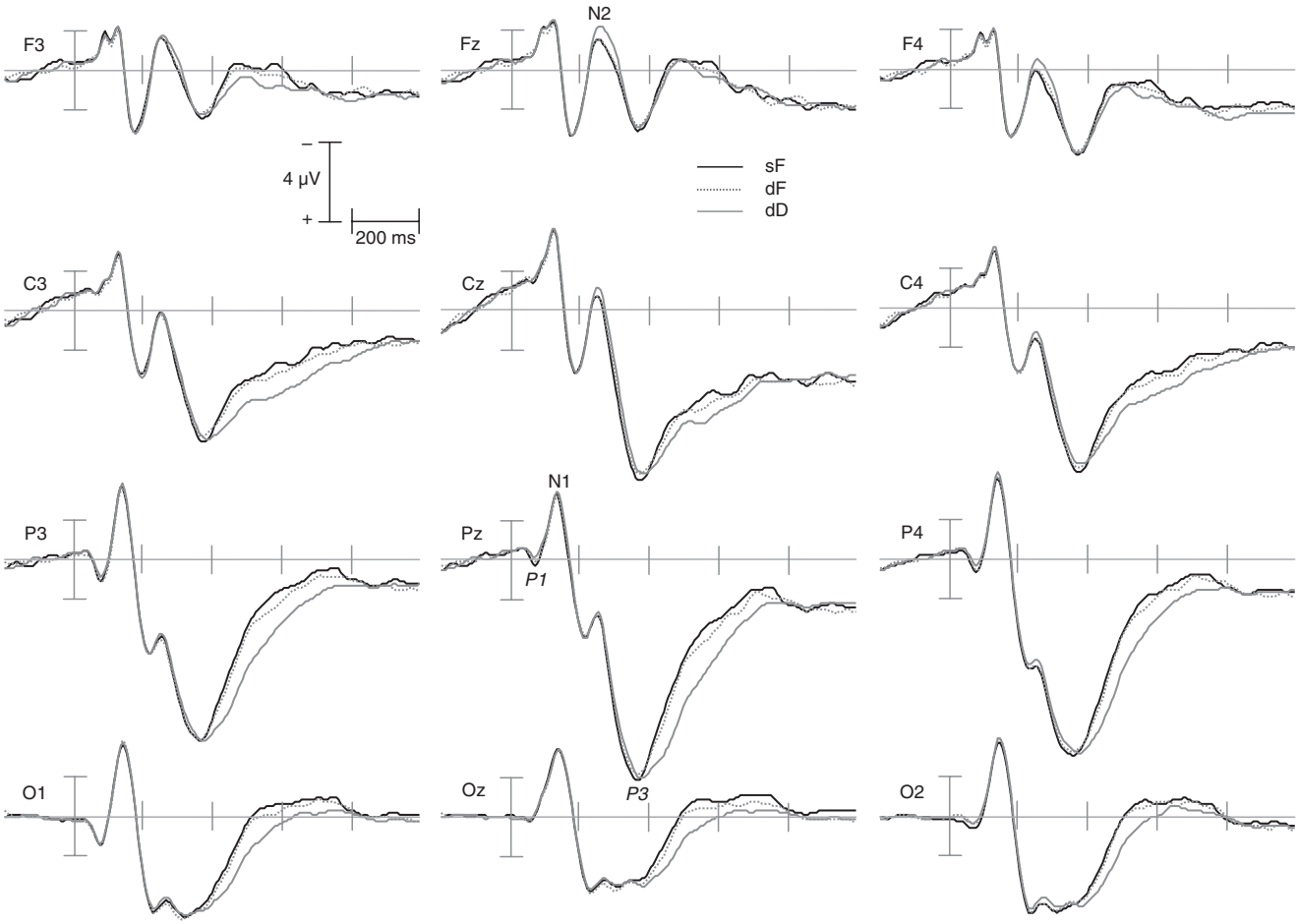

Figure 6. Discrimination task: Grand average waveforms elicited with onset of the target display on trial *n* dependent on the identity of the target on trial $n-1$, for selected electrode positions. Black solid lines indicate same-dimension same-feature trials (sDsF), black dotted lines same-dimension different-feature trials (sDdF), and gray solid lines different-dimension trials (dD). Averages were collapsed across color and orientation targets, as the Dimension × Transition interaction was nonsignificant. Negativity is plotted upward, and the data are presented relative to a 200-ms prestimulus baseline. Components labeled in italics are the N2 at Fz and the P1, N1, and P3 at Pz.

An analysis of N2 latencies with the factors described above revealed no significant effects of the factor Transition.

Topography of N2 effect. To map the N2 dimension change effect topographically, difference waves were computed by subtracting same-dimension trials (combined across same- and different-feature trials and color and orientation dimensions) from different-dimension trials (combined across color and orientation dimensions). Figure 8 presents the resulting difference wave forms and the current source density distribution at 274 ms after stimulus onset.

A repeated-measures ANOVA of the mean difference wave (274 ± 30 ms) with the factors Dimension, Electrode Position (left, midline, right), and Anterior/Posterior (frontal, central, parietal, occipital) revealed the main effect of Electrode Position to be marginally significant, $F(2,20) = 3.29$, $p < .058$, with the strongest activations at midline electrodes. In addition, the interaction Electrode Position × Anterior/Posterior, $F(6,60) = 5.81$, $p < .005$, and the three-way interaction was significant, $F(6,60) = 2.37$, $p < .041$. As in Experiment 1, there were no significant differences between left-lateral, midline, and right-lateral electrodes at parietal and occipital recordings (all $p > .99$). In contrast to Experiment 1, frontal midline amplitudes did differ from left-, but not right-lateral electrodes ($p < .001$ and $p > .95$, respectively). This pattern suggests a slight right-lateralization of the frontal N2 component in the discrimination task.

P300. To examine for influences of dimension changes versus repetitions on the P3 component, the mean amplitudes over the time window 340–380 ms after display onset were subjected to a repeated-measures ANOVA with the factors Dimension, Transition, Electrode Position (left, midline, right), and Anterior/Posterior (frontal, central, parietal, occipital). This analysis revealed the main effects of Electrode Position and Anterior/Posterior to be significant, $F(2,20) = 45.21$, $p < .024$, and $F(3,30) = 16.69$, $p < .001$, respectively. Larger positive deflections were observed over midline and right-hemisphere electrodes compared to left-hemisphere electrodes, with a strong parieto-central maximum.

An analogous ANOVA of the P3 latencies revealed the main effects of Transition, $F(2,20) = 4.84$, $p < .040$, and Anterior/Posterior, $F(3,30) = 5.01$, $p < .037$, to be significant, as in Experiment 1. As before, P3 onset latencies did not differ between same- and different-feature trials (359 vs. 360 ms), but, for both types of

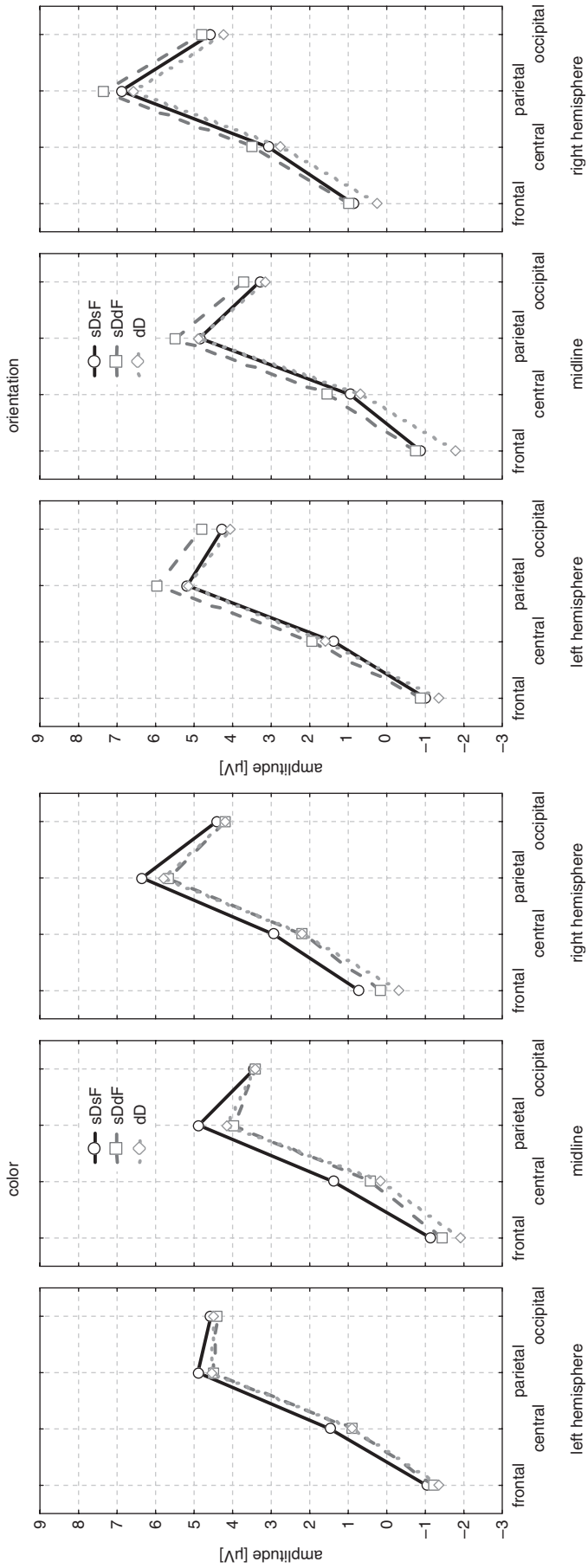

Figure 7. Discrimination task: N2 amplitudes as a function of target Dimension (left side: color; right side: orientation), Intertrial Transition (black solid lines: same-dimension same-feature, sDsF; gray dashed lines: same-dimension different-feature, sDdF; gray dotted lines: different-dimension, dD), Anterior/Posterior (frontal, central, parietal, occipital), and Electrode position (left hemisphere: F3, C3, P3, O1; midline: Fz, Cz, Pz, Oz; right hemisphere: F4, C4, P4, O2).

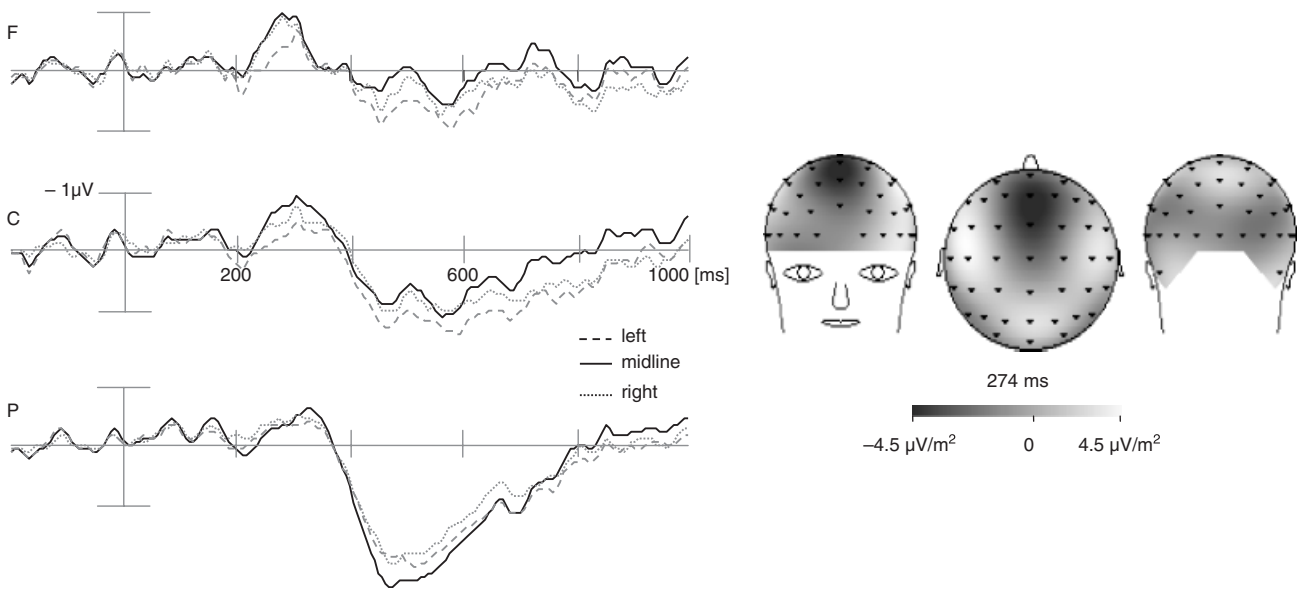

Figure 8. Discrimination task—Left side: Different-dimension minus same-dimension difference wave forms for frontal (F), central (C), and parietal (P) electrode positions. Gray broken lines depict left-lateral, black solid lines midline, and gray dotted lines right-lateral electrode positions. Right side: Current density distribution of the dimension change effect computed for the difference wave forms in the detection task at 274 ms.

trial, latencies were shorter compared to different-dimension trials (371 ms; both $p < .001$). P3 latencies were comparable at frontal, central, and parietal leads (all $p > .91$) and differed significantly for frontal and central relative to occipital electrodes ($p < .02$). In contrast to Experiment 1, the interaction Dimension × Transition was significant, $F(2,20) = 8.00$, $p < .003$ (see Figure 9).

For orientation targets, the latencies were comparable for same- and different-feature trials ($p > .23$), but significantly longer for different-dimension trials (all $p < .001$). Color targets, by contrast, were associated with monotonically increasing onset latencies: same-feature < different-feature < different dimension. Post hoc contrasts revealed the P3 onset latency to be significantly shorter for same-feature as compared to both different-feature and different-dimension trials ($p < .043$ and $p < .001$, respectively); there was no difference between different-feature and different-dimension trials ($p > .35$).

Slow wave. Amplitudes in the slow-wave window were examined by a repeated-measures ANOVA with the factors Dimension, Transition, Electrode Position (left, midline, right), and Anterior/Posterior (frontal, central, parietal, occipital). This analysis revealed the main effects of Dimension, $F(1,10) = 6.83$, $p < .026$, Transition, $F(2,20) = 16.31$, $p < .001$, and Anterior/Posterior, $F(3,30) = 12.47$, $p < .001$, to be significant. Orientation targets elicited a stronger positive-going slow wave compared to color targets, and the strongest positive amplitudes were observed for dimension change trials. A post hoc contrast revealed no significant differences between same- and different-feature trials ($p > .43$), whereas both types of trial differed significantly from different-dimension trials (all $p < .001$). The main effect of Anterior/Posterior was due to the strongest slow-wave amplitudes at central and parietal leads, and significantly less positive-going deflections at frontal and occipital sites. These main effects were qualified by interactions of Dimension × Electrode Position, $F(2,20) = 7.52$, $p < .004$, and Transition × Anterior/Posterior, $F(6,60) = 6.87$, $p < .002$ (see Figure 10). For both dimensions, slow-wave modulations were significantly stronger for midline and right-hemisphere compared to left-hemisphere electrode sites. However, there were significant differences between right and midline electrodes only for color, but not for orientation, targets ($p < .001$ and $p < .06$, respectively). The Transition × Anterior/Posterior interaction was due to the strongest effect of dimension change being located over parietal sites.

Although there was no dimension change effect at frontal electrodes (all $p > .31$), there were significant differences between same- and different-dimension trials at all posterior locations (sDsF and, respectively, sDdF vs. dD, all $p < .007$; sDsF vs. sDdF, all $p > .68$). As can be seen from Figure 10, the dimension change effect was most prominent at parietal sites, followed by central and occipital electrode positions.

Discussion

In Experiment 2, observers had to explicitly identify the dimensional identity of the target in order to respond. As in Experiment 1, performance measures exhibited the general pattern of slowed RTs on trials with a change, compared to a repetition, of the target-defining dimension. However, in contrast to Experiment 1, for color-defined targets, there was a feature-specific as well as a dimension-specific change effect, whereas orientation-defined targets only showed the latter effect. The feature-change effect (i.e., prolonged RTs for different- compared to same-feature targets in the absence of a dimensional change) replicates the findings of Found and Müller (1996), who reported such an effect only with color, but not with orientation targets (see also Müller et al., 2003). To explain this effect, Found and Müller suggested that, in the color dimension, feature contrast may be computed in a number of "subdimensions" or channels coding the inputs from separable populations of color analyzers (see also Wolfe, Chun, & Friedman-Hill, 1995). Thus, a change in the

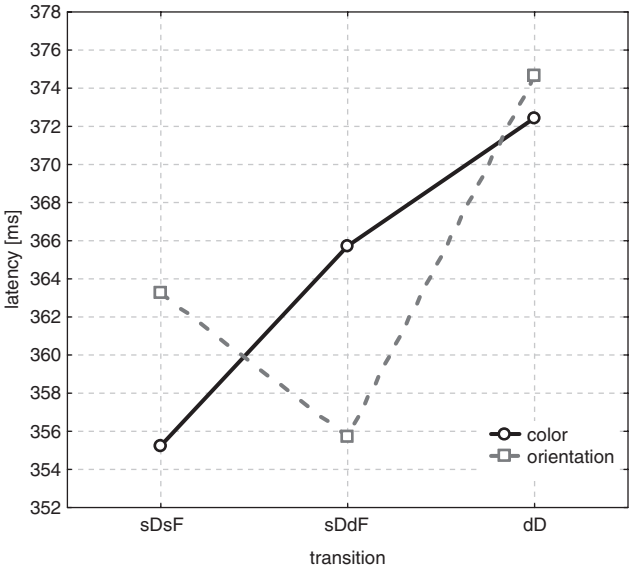

Figure 9. Discrimination task: Mean P3 latencies for color and, respectively, orientation targets on trial n dependent on the dimensional identity of the target on trial $n-1$: same-dimension same-feature (sDsF), same-dimension different-feature (sDdF), and different-dimension (dD). The black solid line indicates P3 latencies for color targets, the gray dashed line P3 latencies for orientation targets.

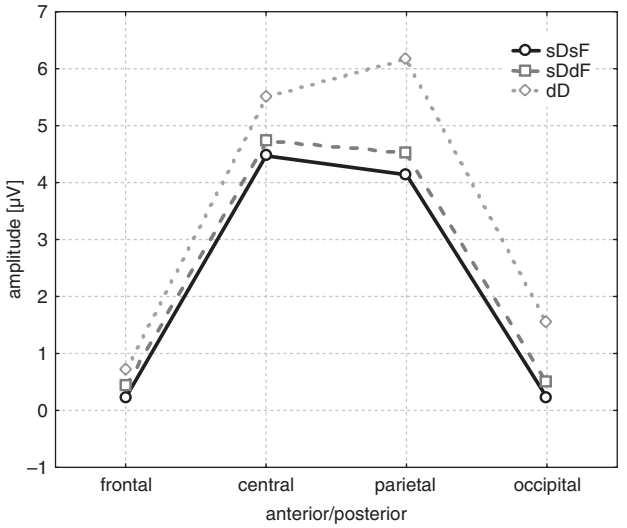

Figure 10. Discrimination task: Mean slow-wave amplitudes from 420 to 600 ms after display onset as a function of Anterior/Posterior electrode position (frontal, central, parietal, occipital), separately for the three Intertrial Transition conditions: same-dimension same-feature (sDsF), same-dimension different-feature (sDdF), and different-dimension (dD).

target-defining color across trials would lead to similar, albeit less marked, costs as a change in the target-defining dimension.

As in Experiment 1, there was no influence of dimension repetition versus change on early visual evoked components. This is consistent with the P1 and N1 reflecting the processing of nonspatial features within the (allocated) focus of attention, rather than parallel processes coding feature contrast prior to the allocation of focal attention.

Importantly in the present context, the differences in RT performance between the two dimensions were not associated with differential N2 amplitude effects at frontal sites. For both dimensions, identical patterns of enhanced N2 amplitudes were observed. As in Experiment 1, the strongest N2 enhancement was found at frontal sites with changes in the target-defining dimension, whereas there were no significant differences between same- and different-feature trials at frontal leads. Note that, although the change effect—reflected in the N2 enhancement—was located fronto-centrally without any lateralization in Experiment 1, a slight right-lateralization was evident in Experiment 2. Further work is necessary to replicate and account for this change in topography.

This general pattern of N2 amplitude modulations is consistent with the dimension-weighting account of Müller and his colleagues (e.g., 1995; Found & Müller, 1996) arguing that these modulations reflect processes of detecting that a new dimensional weight set must be established. Importantly, the N2 enhancements (associated with changes in the target-defining dimension) were similar, both in terms of latency and topography, whether observers had to simply discern the presence of an odd-one-out target (Experiment 1) or explicitly identify its defining dimension (Experiment 2). The similar topography in the two tasks (experiments) supports the assumption of one-and-the-same generator being active during a cognitive process shared by the two tasks.

The P3 component exhibited a different pattern in the discrimination, compared to the detection, task: There was no effect of the factor Transition on P3 amplitudes. However, there were Transition effects on P3 latencies: For orientation targets, there was an effect of dimension change (vs. repetition), in the absence of an effect of feature change (vs. repetition) when the dimension was repeated; in contrast, for color targets, there was both a dimension change effect (sDsF vs. dD) and a feature change effect (sDsF vs. sDdF). This differential pattern of P3 effects is in line with dimension change, but not feature change, effects in the RTs to orientation-defined targets and monotonically increasing RTs (sDsF < sDdF < dD) for color-defined targets. Thus, the pattern of P3 latencies exactly matches that of the RTs in the discrimination task, further supporting the assumption that processes of attentional weight shifting might contribute to this component.

Finally, the pattern of slow-wave amplitudes observed in the discrimination task replicated that in the detection task. In particular, there was a systematic SW variation that mirrored the RT pattern, with the strongest effect of dimension change (vs. repetition) observed over parietal leads with a midline maximum. Again, all posterior recordings showed dimension changes to provoke significantly more positive-going deflections from central over parietal to occipital recordings, implicating a widespread effect of dimension changes on consecutive trials.

In summary, the N2, P3, and SW amplitude and latency effects in the "discrimination" Experiment 2 were comparable to the effects in the "detection" Experiment 1. Thus, the systematic and similar variations of both components support the assumption that the detection of an odd-one-out feature target requires (at least implicit) knowledge of its dimensional identity. If processing differed fundamentally between the two tasks, then systematic differences in ERP effects should have been observed. However, the N2 latencies were virtually equivalent (252 and 257 ms sD and, respectively, dD trials in the detection, as compared to 258 ms and 259 ms in the discrimination task), and, if anything, the P3 latencies were shorter for the discrimination than

the detection task (367 and 393 ms for sD and, respectively, dD trials in the detection task, as compared to 360 and 374 ms for the discrimination task). The latter difference may be taken to suggest that weight shifting is expedited when the task requires explicit knowledge of the target-defining dimension (Müller et al., 2004). However, because different observers participated in the two experiments, any direct comparison must be interpreted with caution.

General Discussion

Two experiments examining visual search for singleton feature targets across dimensions replicated the pattern of RT effects described by Found and Müller (1996): Repetitions of the target-defining dimension on consecutive trials led to faster RTs, whether or not the target-defining feature changed within the repeated dimension, compared to changes in the target-defining dimension. This pattern is consistent with the dimension-weighting account proposed by Müller and his colleagues (e.g., Müller et al., 1995, 2003; Found & Müller, 1996). The aim of the present study was to identify parameters of the EEG associated with the pattern of RT effects described above—predicated on the idea (1) that components of the ERP that display the same systematic variation with changes versus repetitions of the target-defining dimension can help to trace the time course of the dimension-weighting process, and (2) that the topography of possible indicators would provide tentatively information about the brain areas involved in the dimension-based modulation of visual search.

Analyses of ERPs with onset of the target display, dependent on the dimensional identity of the target on the previous trial, revealed three components to exhibit such a systematic variation: the N2, the P3 (with respect to its onset latency), and the SW. Whether the task required simple target detection (Experiment 1) or discrimination of the target-defining dimension (Experiment 2), the three components showed the same pattern: changes (vs. repetitions) of the target-defining dimension led to an increased negativity of the N2, longer latencies of the P3, and an increased positive deflection within the SW time range. Besides minor differences between color- and orientation-defined targets, these amplitude and latency effects mirror the RT patterns typically observed in cross-dimension search for singleton feature targets. This also extends to the amplitude modulations for same-dimension trials, which were unaffected by whether or not the target-defining feature changed within the repeated dimension. This pattern of effects reinforces the proposal that the attentional weighting is dimension-, rather than feature-, specific in nature.

The identification of ERP parameters likely reflecting attentional (re)weighting at the level of electrocortical activity pertains to an important issue controversially discussed in the literature: the question as to the point in time, and stage of processing, of the weight adjustment. The present findings favor an account that assumes that attentional weight is (re)assigned at a relatively early point in time, and is associated with the generation of dimension-based (saliency) representations. That is, limited "weight" resources need to be (re)allocated to the mechanisms establishing the presence of a target or, respectively, its dimensional identity. Accordingly, the (re)allocation of attentional weight is a prerequisite for the selection and execution of a manual response (Müller et al., 1995; Found & Müller, 1996). The dimension-based account, which associates weight shifting with perceptual processes, has recently been challenged by models in which the (re)allocation of attentional resources is assumed to occur after visual encoding mechanisms have completed processing and the relevant response is selected. For example, Cohen and Magen (1999) argued that dimension-based intertrial effects arise at a (dimensions-specific) response selection stage. A similar, response-based stance was advocated by Mortier et al. (2005). They failed to find dimension-based intertrial effects in a "compound" search task, in which observers' responses are based not on the search-relevant feature of the target (e.g., its unique outline shape, such as a circle among squares), but on some additional attribute associated with the target (e.g., the orientation of a line presented within the circular target). In compound tasks, perceptual (search-related) and response-related effects of the task are assumed to be dissociable—so that intertrial effects, if they were indeed perceptual in nature, should be observed in compound as well as detection tasks. It is important to note, however, that the above "nonfindings" are not unequivocal. For example, dimension-based intertrial RT effects in a compound search tasks were reported by both Krummenacher, Müller, and Heller (2002) and Wolfe, Butcher, Lee, and Hyle (2003), and doubt has been cast on the simple dissociability of search- and response-related processes in compound tasks (e.g., Müller & Krummenacher, 2006; Pollmann et al., 2006).

The results of the present study support the assumption that the requirement for a (re)allocation of attentional resources is detected before visual encoding mechanisms have completed processing and the relevant response is selected. In Experiment 1, observers were required to respond to a target with the index finger of one-and-the-same hand irrespective of its defining dimension. Despite this, there was an amplitude modulation of the N2, arguing that this modulation is unrelated to changes in manual response processes (selection, preparation, or execution). In Experiment 2, changes in the target-defining dimension were coupled to changes in response selection and execution. Yet, the N2 showed a pattern of effects similar to that in Experiment 1. Thus, the N2 modulation is selectively associated with (perceptual) changes in the target-defining dimension, while being unrelated with response times. Thus, a redistribution of attentional weight is initiated prior to response selection taking place. Taken together, the present results argue that the detection of dimensional change and the initiation of weight shifting is independent of and occurs prior to response selection.

Further support for the assumption of weight shifting processes being initiated and carried out before response selection is initiated stems from the observed P3 modulations. In Experiment 1, observers had to respond to odd-one-out targets with the index finger of one-and-the-same hand. Therefore, dimension changes were not associated with changes in response selection. Thus, purely response-driven effects cannot explain the differential P3 effects found in the present study. To further examine whether P3 latency modulations induced by dimension change across trials were primarily associated with stimulus- or response-related processing, additional analyses were carried out on stimulus- and response-locked P3s. These revealed no systematic differences in P3 amplitudes dependent on the reference event (stimulus-locked vs. response-locked) in the detection or the discrimination task—arguing against the P3 modulations observed in the present tasks being driven by response processes and instead supporting the assumption that the P3 is mediating between perceptual (search-related) and response-related processes (Verleger, Jaskowski, & Wascher, 2005). In particular, dimensional weight-shifting processes might contribute to the P3

"complex" observed in both the detection and the discrimination experiment of the present study.

Finally, the pattern of SW amplitudes mirrored that of the RTs in both experiments, with increased positive deflections for dimension change, compared to repetition (i.e., both same- and different feature), trials. These effects cannot simply be attributed to response-related processes, because the required (target-present) response in Experiment 1 was the same for all targets, irrespective of the target-defining dimension. In Experiment 2, the two dimensions were associated with different responses—nevertheless, the pattern of SW amplitudes was comparable to that in the detection task. This implies that the weight shifting process, initiated with the N2 component, influences the ERP beyond the P3.

The topography of the N2 effect indicates that frontal brain areas are likely involved in the dimension weighting process. A frontally distributed negativity was also found in several studies that have used EEG to identify change-related activity in matching tasks, revealing enhanced N270 amplitudes for changes between the S1 and S2 stimuli (Cui, Wang, Wang, Tian, & Kong, 2000; Tian, Wang, Wang, & Cui, 2001; Wang, Tang, Kong, Zhuang, & Li, 1998; Wang et al., 2003; Zhang et al., 2001). Such enhanced negativities have been taken to reflect the detection of change or the processing of conflict. Further analyses aimed at reconstructing the source of the measured surface potentials are needed to identify the neural generators underlying the N2. However, the results are in line with the work of Pollmann and his colleagues (Pollmann, 2004; Pollmann et al., 2000, 2006; Weidner et al., 2002), who used fMRI to identify a fronto-posterior network of brain areas playing a critical role in dimensional weight shifting. The pattern of frontal activations was interpreted as reflecting the control of dimensional weight shifting, whereas higher level visual areas in superior parietal and temporal cortex were assumed to mediate the weight shifts via feedback to the dimension-specific input areas in occipital cortex (Pollmann et al., 2006).

In the present experiments, the N2 modulation occurred about 250 ms after search display onset with a frontal distribution. The systematic variation of the N2 with changes in the target-defining dimension is a novel finding, likely reflecting the detection of dimension change and the initiation of the resetting of dimensional weights. The redistribution of the attentional weights might contribute to the subsequent P3 and SW effects, revealing systematic variations with changes in the target-defining dimension (but not feature changes within a repeated dimension). Because the N2 modulation in the present study was revealed by analyses of ERP components dependent on the intertrial history of target "events," it is proposed to term this modulation "*transition N2*" (tN2) in visual search. Further work is required to investigate these findings in more detail and to examine whether early indicators of dimensional change may be found dependent on dimensional intertrial transitions in singleton feature search.

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