ABSTRACT

Misbinding is a perceptual phenomenon where subject perceives the features of different objects correctly but does not bind them correctly into integrated objects: the features of two stimuli are combined in the perceived object. Although a lot of the feature misbinding research has studied this phenomenon in space domain only a limited set of studies have investigated feature misbinding in time domain. We conducted three experiments with a simple masking task with stimuli presented in the same location to study temporal dynamics and electrophysiological correlates of misbinding. Comparing the temporal dynamics of misbinding and error trials we found that misbinding is a different phenomenon from simple error trials. However, we also observed asymmetries between misbinding types. Our results suggest that only misbinding where the shape of the first stimulus is bound to orientation of the second stimulus is a real perceptual phenomenon whereas misbinding where shape of the second stimulus is bound to orientation of the first stimulus is indistinguishable from error trials. These results were supported by electrophysiological evidence. Comparing the event-related potentials of misbinding types and errors, we found that misbinding with shape of the first stimulus and orientation of the second stimulus differs from simple errors by having higher P3 amplitude. Our results show that the masking paradigm can be a fruitful experimental setup for studying feature binding and misbinding.
KOKKUVÕTE

Väärsõlmimise ajaline dünaamika ja EEG korrelaadid.


Märksõnad: tunnuste sõlmimine, sündmuspotentsiaal, visuaalne maskeerimine.
1. INTRODUCTION

Perception seems effortless: even from a moving tram one can easily perceive a clown on the street with his red nose and awkward-shaped clothing. However, perception becomes quite complex once one starts thinking about how it is actually accomplished in the brain. For example, it is known that different features of the object (the clowns face, the color of his nose, the shape of his clothes, the movement of the whole scene) are processed in different parts of the brain and that there is no brain area, where all this information is put together (e.g. Koch, 2004). By which miracle do these different features become bound together to one integrated perceptual object (the clown)? This problem, called the “binding problem”, is somehow easily solved by the brain, but the scientists still ponder how feature binding is achieved. One must note that the binding problem indeed is a real problem for the brain and not just a hypothetical construct: for example, Friedman-Hill et al (1995) have examined a neurological patient R.M. with bilateral parietal-occipital lesions who miscombined color and shape even under free viewing conditions.

There have been many hypotheses to explain neural mechanisms of feature binding. For example, binding could be accomplished by the visual cortical hierarchy by combining simple feature combinations step-by-step into higher order cells selective for specific feature combinations (Barlow, 1972). Although the idea has been criticised (e.g. Singer & Gray, 1995), there indeed are neurons in the human medial temporal lobe that are selective for specific persons regardless of their clothing, viewpoint or figure background (Quiroga et al., 2005). However, there are no such neurons for novel feature combinations, thus feature binding has to be achieved differently when one perceives an object that he or she has never encountered before. In this situation binding of features could be achieved by neural synchrony (Fries et al., 2001; Singer & Gray, 1995). Especially gamma synchronization is often considered as a potential mechanism of perceptual binding (Gray et al., 1989; Tallon-Baudry & Bertrand, 1999). However, gamma synchrony hypothesis has also received some critique and there have been several studies where gamma activity does not predict binding (Palanca & DeAngelis, 2005; Thiele & Stoner, 2003). The inconsistent results are sometimes considered as a result of properties of stimuli, the structure of task, data type and attention (Fries et al., 2002; Henrie & Shapley, 2005; Tallon-Baudry et al., 1997). The ongoing discussions around the binding problem (e.g., a special issue in Frontiers in
Psychology published in spring 2013) show that this research topic is still important and presents many unanswered research questions.

Although there are a lot of studies that analyze neural basis of binding, there is also a variety of research of psychophysical studies that try to explain what are the precise perceptual mechanisms of binding (e.g. Treisman and Gelade, 1980). Beneficial phenomena for studying binding are illusory conjunctions where subject perceives the features of different objects correctly but does not bind them correctly into integrated objects: the features of two objects are combined into one. For example, simultaneously presenting a green square and a red circle might create a perception of a green circle - the features were indeed presented on the screen but not in this combination. This phenomenon is particularly interesting because it differentiates conditions where subject fails to perceive some of the features from conditions where subject only fails in binding but succeeds in identifying features. There have been many demonstrations of insufficient binding in conditions where correct binding is perturbed by short duration of stimuli or challenging attentional conditions. In these experimental setups illusory conjunctions often occur (Treisman and Gelade, 1980). Importantly, these illusory conjunctions appear and have been mostly studied when two objects are presented simultaneously in different locations. However, another research tradition is to present objects from the very same visual location in different times with rapid succession. If such objects also have several features, illusory conjunctions could also arise in such experimental paradigms.

Hommuk and Bachmann (2009) used masking paradigm to study temporal limitations of feature binding. To avoid confounding perceptual object processing with the effects of spatial attention, they presented the stimuli from a single location. In the masking task, two stimuli were presented with a short stimulus onset asynchrony (SOA: 47 ms) and subjects had to either identify the stimuli or search for the stimulus based on cued feature before the stimuli. Although the subjects were quite successful in identifying the stimuli, this perceptual availability and focusing attention on the target’s searched property did not improve identification of the other feature of the target stimulus. Instead, strong masking effect was found. They showed that temporal limitations of presenting the stimuli can result in limitations of binding in errors and misbinding the features of presented stimuli. They also found that misbinding is asymmetrical. When participants searched for shape, there was higher rate of misbindings where the subject reported orientation of the second stimulus instead of orientation of the first stimulus when
compared to misbinding where the subject reported orientation of the first stimulus instead of the orientation of the second stimulus.

There has also been debate whether the illusory conjunctions are a real perceptual phenomenon at all. Namely it could be argued that in swiftly presented stimulus displays subjects’ performance is generally low and they have very low confidence in their responses, thus “misbinding” responses are only artifacts that happen by insufficient processing of features and guessing (Donk, 1999). This perspective would predict that the misbinding responses are not very different from the error responses. On the other hand, most proponents of the illusory conjunction phenomenon claim that subjects indeed perceive objects with misbound features, i.e. there is evidence that illusory conjunctions are something different from mere guessing and reported features are not randomly chosen (Ashby, Prinzmetal, Ivry, & Maddox, 1996; Hazeltine, Prinzmetal, & Elliot, 1997; Prinzmetal, Diedrichsen, & Ivry, 2001).

1.1 Research question and hypothesis

As only a limited set of studies have investigated feature misbinding of objects presented from the same location in rapid succession, we wanted to add weight to that line of research. Presenting objects swiftly in time can reveal the fundamental limitations and mechanisms of perception (e.g. Bachmann, 2000) and thus also provide new questions and answers for the study of feature binding. In the current work, we present three experiments concerning temporal dynamics and neural correlates of misbinding. We used similar stimuli as in the experiments of Hommuk and Bachmann (2009). However, we did not instruct our subjects to attend to a particular feature, but rather wanted them to report perceived objects. More importantly, we aimed at examining temporal dynamics of misbinding by varying the SOA between the two stimuli and measuring how misbinding depends on SOA (experiments 1 and 2). In the third experiment we studied the electrophysiological correlates of misbinding. We hoped that looking at the temporal dynamics and the electrophysiological correlates of misbinding would reveal whether misbinding is a real perceptual phenomenon or whether misbinding responses are indistinguishable from errors.

As this research paradigm has not been used frequently for studying feature misbinding, our goal is to explore and describe the misbinding phenomena observed in this experimental setting.
In accordance with that our hypotheses are rather general. We expect that 1) the phenomenon of misbinding can be robustly observed in the present experimental setup, 2) misbinding is a different phenomenon from simple error trials. Based on Hommuk & Bachmann (2009) we also expect that 3) there are asymmetries in misbinding that rise from the different temporal processing characteristics of the features.

2. EXPERIMENT 1

2.1 The aim and background of experiment 1

The aim of experiment 1 was threefold: 1) to extend the results of Hommuk and Bachmann (2009), 2) to examine temporal dynamics of misbinding in masking condition by varying the SOA and 3) to find the most promising temporal window for the subsequent EEG experiment.

2.2 Methods

The experimental setup was a modified version of the Hommuk and Bachmann (2009) study with varying SOA.

2.2.1. Subjects. Eight subjects (3 male, aged 19-28 (M = 23.0, SD = 2.8)) participated in the experiment. All reported normal or corrected to normal vision. Subjects gave written informed consent prior to participation. The study was approved by the ethics committee of University of Tartu and the experiments were undertaken in compliance with national legislation and the Declaration of Helsinki.

2.2.2. Stimuli and apparatus. The stimuli were presented on an Eizo Flex Scan T550 monitor (Eizo Nanao Corp., Hakusan, Japan), with a refresh rate of 85 Hz. All stimuli were monochromatic and of maximum contrast, the viewing distance was approximately 60 cm. The targets were geometrical figures (square, disc and triangle), presented at the fixation point, and each shape delineated a surface of one of the three possible gratings with an orientation of 0°, 45°, or 90°, giving altogether 9 different stimuli (figure 1). Targets had no border contours. The sizes of all stimuli were about 0.6° x 0.6°. The stimuli were presented on gray background.
2.2.3. Design and the procedure. Each trial started with a fixation period with a fixed length of 720 ms. After that two stimuli were presented in the fixation point: the first for 24 ms and the second for 12ms. Five SOAs (48, 72, 96, 120, 144 ms) were used in randomized order. The two stimuli on each trial were always different. The stimuli for presentation were selected quasi-randomly. As identifying both features of both stimuli was accomplished with very low correct response rate (10%) in Hommuk and Bachmann (2009) study and as our pilot study also confirmed that subjects cannot successfully identify both features of both stimuli, subjects were asked to report only the stimulus that they perceived more clearly. Each subject performed 500 trials. The responses were categorized into three groups: correct response if subject reported a stimulus with either both features from the first or both features from the second stimulus; misbinding response if subject reported a stimulus with the shape of one and the orientation from the other stimulus and errors if the subject reported a stimulus that did not have both features from either of the presented stimuli.

2.3. Results

To investigate the temporal dynamics of misbinding, a one-way repeated-measures ANOVA was conducted on the relative amount of misbinding with the factor SOA. The analysis yielded a main effect of SOA, $F(4,52) = 20.39, p < 0.0001$. As can be observed from figure 2 there is more misbinding for shorter SOAs. To analyse the asymmetry of misbinding, two types of misbinding
Temporal dynamics of misbinding

(type 1: shape from first object and orientation from second object; type 2: orientation from first and shape from second object) were contrasted over SOAs. Two-way repeated measures ANOVA yielded the main effect of misbinding type (F(1,13) = 108.6, p < 0.0001). As can be seen from figure 3 there was significantly more type 1 misbinding over all SOAs. The interaction between misbinding type and SOA (F(4,52) = 5.883, p = 0.0006) was also significant showing that the two misbinding types have different temporal dynamics. While type 2 misbinding was relatively constant over all probed SOAs, there was proportionally more type 1 misbinding for short SOAs (figure 2). This result points at the possibility that the two types of misbinding might represent different phenomena.

To analyse correct responses, a one-way repeated-measures ANOVA was conducted and it yielded main effect of SOA (F(4,52) = 23.44, p < 0.0001). As the subject was instructed to report the stimulus he or she perceived more clearly, in the correct trials the subject could report either the first or the second stimulus. Thus, next we analyzed the correct response type (first stimulus, second stimulus) over SOAs. In addition to the reported SOA effect we observed a main effect of correct response type (F(1,13) = 75.98, p < 0.0001) and a significant interaction between SOA and correct response type (F(4,52) = 5.376, p < 0.001). As can be seen from figure 4 the second stimulus is reported more often, but its advantage of the second stimulus is smaller on the shortest and longest SOAs.

The stimulus onset asynchrony did not have effect on the percentage of error responses, F(4,52) = 1.905, p = 0.124. Curiously enough the temporal dynamics of the error trials (figure 3) were qualitatively similar to the temporal dynamics of the type 2 misbinding (figure 2) and differed from those of type 1 misbinding (figure 2). Could it be that what we consider as type 2 misbinding is actually an error trial? We tried to quantify that intuition by conducting a two-way repeated measures ANOVA with one factor being SOA and the other the percentage of the error trials or type 2 misbinding. If type 2 misbinding is not similar to the error trials we should observe an interaction between the factors. The ANOVA showed no significant interaction between two factors (F(4,52) = 0.903, p = 0.469) which corroborates the idea that type 2 misbinding trials might be a type of error trials. In contrast, when we ran a very similar ANOVA where instead of type 2 misbinding trials we used type 1 misbinding trials, we observed a clear interaction (F(4,52) = 6.698, p < 0.0001) between SOA and the type of trial (error vs. type 1
misbinding trial). This interaction shows that type 1 misbinding has different temporal characteristics and thus most likely different underlying mechanisms as compared to error trials.

Figure 2. Percentages of misbinding types for each SOA. Type 1: subject reported the shape of the first stimulus and orientation of the second stimulus, type 2 subject reported the shape of the second stimulus and orientation of the first stimulus.

Figure 3. Percentages of misbinding, correct and error responses for each SOA.
2.4. Discussion

We used an experimental setup where two stimuli are presented from the same location in rapid succession. We observed that with SOAs of 48-144 ms, the second stimulus is reported more often than the first stimulus and the advantage of the second stimulus is smaller on the short SOAs. These results are consistent with classic findings from the visual masking research (e.g. Bachmann & Allik, 1976).

As we expected, the phenomenon of misbinding can be robustly observed in the simple visual masking task. However, similarly to Hommuk and Bachmann (2009), we found asymmetries of misbinding. We use the term misbinding type 1 for the case where the subject reports an object consisting of the shape from the first object and orientation from the second whereas in type 2 misbinding orientation from the first and shape from the second object are combined. Our first experiment showed that these two misbinding types might represent different phenomena. Although we classified them both as “misbinding” their relative proportion behaved differently dependent on the SOA between stimuli as evidenced by the interaction between SOA and misbinding type. Furthermore, although type 1 misbinding had different temporal dynamics from the error trials, type 2 misbinding did behave over the SOAs very similarly to error trials. Could it be that the type 2 misbinding is actually a simple error trial and we just artificially classify it to be “misbinding”?

Figure 4. Percentages of correct responses for each SOA.
However, notice that our analysis cannot show conclusively that error trials and misbinding type 2 are indeed covered by the same underlying mechanism. We observed that the temporal dynamics over SOAs of error trials and misbinding type 2 were not different as indicated by the non-significant interaction between the SOA and the proportion of error trials/misbinding type 2. We acknowledge that the absence of evidence for the difference between error trials and type 2 misbinding is not "evidence of absence" of the difference - maybe misbinding type 2 and error trials are indeed different but our data was not sufficient to show it (too few trials). Therefore, one of the key reasons for experiment 2 was the question, whether we can replicate these results. Furthermore, in principle it could be that the difference between the two misbinding types might be a result of unequal presentation time of the stimuli. Thus, in experiment 2 we used equal presentation times for both stimuli.

One of our goals was to find the most promising temporal window for the subsequent EEG experiment. Our results suggest that shorter SOAs are more promising to produce enough misbinding trials for the EEG experiment. However, with our set of stimuli, there is high probability of random occurrence of misbinding. If the subject correctly identifies one of the features, there is 33% of probability of randomly guessing the second feature. To reduce this risk, we conducted the 2nd experiment with an extended set of possible features.

3. EXPERIMENT 2

3.1 The aim of experiment 2

The aim of experiment 2 was to repeat experiment 1 with conditions that reduce the probability of random occurrence of misbinding by adding one shape and one orientation to the set of possible features. We were also keen to see whether we can replicate our results from experiment 1 and observe that type 2 misbinding has similar temporal dynamics to error trials.

3.2 Methods

3.2.1. Subjects. Eight subjects (2 male, aged 19-36 (M = 26.0, SD = 5.2)) participated in the experiment. All reported normal or corrected to normal vision. Subjects gave written informed consent prior to participation. The study was approved by the ethics committee of
University of Tartu and the experiments were undertaken in compliance with national legislation and the Declaration of Helsinki.

3.2.2. Stimuli. The stimuli were geometrical figures (square, diamond, triangle, inverted triangle), presented at the fixation point, and each shape delineated a surface of one of four possible gratings with an orientation of 0°, 45°, 90°, 135°, thus combining for a total of 16 different stimuli (figure 5). The sizes of the stimuli were 0.6° x 0.6°. The background was grey (in RGB measure, red = 158, green=158, blue=158). Space-average luminance was for background 9.9 cd/m² and for stimuli 7.1 cd/m².

![Figure 5. The response window with stimuli of experiment 2 and 3](image)

3.2.3. Design and procedure. Subjects were seated in a dimly lit room, 85 cm from the screen. Stimuli were presented on a SUN CM751U monitor (1024x768 pixels) at 100 Hz refresh rate. The experiment comprised 576 trials in total. After every 100 trials, participants were instructed to have a pause to rest their eyes. Each trial started with a fixation period with a variable length of 1230-1500ms. In each trial, 2 stimuli were presented. The duration of both stimuli was 10 ms and 3 SOAs (40, 60 and 80ms) were used in random order. The features of stimuli were randomized. The two stimuli of each trial were always different. The subject had to report the stimulus that was perceived more clearly.
3.3 Results

To investigate the temporal dynamics of the different types of misbinding, a two-way repeated-measures ANOVA was conducted on the relative amount of misbinding with the factors SOA and the type of misbinding (type 1: shape from first object and orientation from second object; type 2: orientation from first and shape from second object). We observed the main effect of SOA ($F(2,14) = 13.51$, $p < 0.00054$), but no significant effect of misbinding type ($F(1,7) = 1.539$, $p = 0.255$). Importantly, there was again a significant interaction between SOA and misbinding type ($F(2,14) = 7.071$, $p = 0.0076$). From figure 6 it can be observed that the proportion of the two misbinding types indeed again depend differently on the SOA. Post-hoc analysis revealed that there were significantly more type 1 misbinding responses with the shortest SOA (40ms ($t(7) = 3.3733$, $p = 0.012$)), but there were no significant differences for either 60 ms SOA ($t(7) = 0.24$, $p = 0.817$) or 80 ms SOA ($t(7) = 0.262$, $p = 0.801$).

To analyse correct responses, a two-way repeated measures ANOVA with the factors SOA and type of correct trial was conducted. The results can be seen on figure 7. We observed a main effect of SOA ($F(2,14) = 11.74$, $p < 0.001$). There was also main effect of correct response type ($F(1,7)=22.85$, $p=0.002$) and there was significant interaction between SOA and correct response type ($F(2,14) = 28.91$, $p < 0.0001$).

The stimulus onset asynchrony did not have an effect on the percentage of error responses ($F(2,14) = 3.024$, $p = 0.081$). As in experiment 1 we ran a two-way repeated measures ANOVA with one factor being SOA and the other the percentage of the error trials or type 2 misbinding. The ANOVA showed no significant interactions between two factors ($F(2,14) = 0.42$, $p = 0.665$) which again supports the idea that type 2 misbinding trials might be a type of error trials. In contrast, as in experiment 1, we observed a significant interaction between SOA and the type of trials when instead of type 2 misbinding we compared type 1 misbinding to error trials ($F(2,14) = 4.164$, $p = 0.038$). Thus, we replicated our findings from experiment 1 that type 1 and type 2 misbinding trials have different temporal properties and type 2 misbinding trials are not different from error trials.
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Figure 6. Percentages of misbinding types for each SOA. Type 1: subject reported the shape of the first stimulus and orientation of the second stimulus, type 2 subject reported the shape of the second stimulus and orientation of the first stimulus.

Figure 7. Percentages of trials with misbinding, correct and error responses for each SOA.
3.4. Discussion

In the second experiment we could replicate all the main results from first experiment. The aim of the second experiment was to reduce the probability of random occurrence of misbinding. Comparing the results of the second and the first experiment, one could notice that the proportion of misbinding was smaller in the second experiment. Although this could be a result of adding extra features, it should be noted that the proportion of the correct responses increased while the proportion of errors was similar to the proportion in the first experiment. These results suggest that the subjects were simply more effective in identifying the stimuli.

We also wondered whether the unbalanced duration of stimuli in experiment 1 was the cause of asymmetry of misbinding. This assumption was not confirmed in our second experiment where both stimuli had equal duration. On the contrary, asymmetry between the misbinding types found in the first experiment was also present in the second experiment. Again, the relative proportion of the two misbinding types did behave differently dependent on the SOA between stimuli as indicated by the interaction between SOA and misbinding type. Also, as in experiment 1, misbinding type 2, where the shape of the second stimulus is bound with the orientation of the first stimulus, had similar dynamics to error trials. Misbinding type 1 trials, where the shape of
the first stimulus is bound with the orientation of the second stimulus, did behave differently from error trials over time as evidenced by the significant interaction between the trial type and SOA.

Although the second experiment confirms all the main results of the first experiment and shows that type 1 misbinding is different from error trials, we have no information about the processing stage where the differences between these two types of trials originate. Also, our comparisons up to now have not successfully quantified how type 1 misbinding differs from the correct trials. As electrophysiological evidence can sometimes help to understand in which stage the processes differ, we measured and compared the electrophysiological signatures of both misbinding types and contrasted them with correct and error responses in experiment 3.

4.EXPERIMENT 3

4.1 The aim of experiment 3

The aim of experiment 3 was to examine ERPs of misbinding trials contrasted to errors and correct answers with the hope that ERPs would shed further light on the mechanisms of feature misbinding. In particular based on our behavioral experiments we expected that misbinding 1 trials would be different from error trials whereas misbinding 2 trials are not.

4.2 Methods

4.2.1. Subjects. Eleven subjects (2 male, aged 20-29 (M = 25.0 , SD = 2.9)) participated in the EEG experiment. All reported normal or corrected to normal vision. Subjects gave written informed consent prior to participation. The study was approved by the ethics committee of University of Tartu and the experiments were undertaken in compliance with national legislation and the Declaration of Helsinki.

4.2.2. Stimuli, design and procedure. The stimuli, apparatus and the design of the behavioral experiment were the same as in the experiment 2. In experiment 3 only one SOA (40ms) was used and in addition to the behavioral task EEG was recorded.

4.2.3. EEG recordings. We used Nexstim eXimia EEG-system with 60 carbon electrodes cap (Nexstim Ltd, Helsinki, Finland). 60 electrodes were prepared for recording. The impedance
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at all electrodes was kept below 10KΩ. The EEG signals were referenced to an additional reference electrode placed on the forehead and sampled at 1450 Hz sampling rate. All signals were amplified with a gain of 2000 and with a hardware based lowpass filter of 350 Hz. Vertical electrooculogram (VEOG) was recorded in addition to the EEG.

4.2.4. EEG preprocessing. All EEG data were analyzed with Fieldtrip (http://fieldtrip.fcdonders.nl; version 04-10-2011; Oostenveld, R. et al., 2011). Data were filtered (30 Hz low-pass zero phase shift Butterworth filter, to prevent edge artifacts, 1.4 s padding was applied to filtering) and epoched around the first stimulus onset (-200 to +1000 ms). Epochs were baseline corrected with a 200 ms time period before first stimulus onset. Trials were inspected manually for artifacts. All trials containing eye movements and other artifacts were removed from the data before analysis. Channels with noisy signal were detected manually and repaired by nearest-neighbor interpolation. No more than 6.6% of data was interpolated for each subject.

4.2.5. EEG analysis. After the preprocessing, ERP's were computed for each subject in each condition and for each electrode. For further analysis, electrodes were pooled together. For frontal area electrodes Fp1, Fpz, Fp2, AF7, AF5, AF3, AF1, AFz, AF2, AF4, AF6, AF8, F7, F5, F3, F1, Fz, F2, F4, F6, F8, FC5, FC3, FC1, FCz, FC2, FC4, FC6 were pooled together. For central area electrodes: C5, C3, C1, Cz, C2, C4, C6, CP5, CP3, CP1, CPz, CP2, CP4, CP6 were pooled together and as there were no significant differences between occipital and parietal electrodes parietal and occipital electrodes P9, P7, P5, P3, P1, Pz, P2, P4, P6, P8, P10, PO9, PO7, PO5, PO3, PO1, POz, PO2, PO4, PO6, PO8, PO10, O1, Oz, O2 were pooled together.

The experimental conditions were: correct answers (the subject reported either the first or the second stimulus), errors, misbinding type 1 and 2 trials (type 1: shape from first object and orientation from second object; type 2: orientation from first and shape from second object). Mean amplitude was used for analysis of ERP amplitudes. Peaks were identified from a grand-average over all conditions and all subjects. The following criteria were applied: for P1 109 ± 30 ms; for N1, 185 ± 40 ms; for P3 300 - 600 ms mean amplitude.

4.3 Results

To test our hypothesis that misbinding type 1 trials are different from error trials, response types (correct, error, misbinding type 1 and misbinding type 2) were pairwise contrasted over
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time epochs of interest. We observed no effects involving misbinding type 2 trials, which most likely reflects the fact that there were only a handful of trials per subject in the misbinding type 2 condition (i.e. the signal to noise ratio was not good enough to obtain any reliable results for the comparisons with misbinding type 2).

Contrasting misbinding type 1 trials with error trials revealed the expected difference: in the P3 time window misbinding type 1 trials elicited more augmented P3 over all electrode groups (see table 1 and figure 8-9). In accordance with that, correct trials were also associated with higher P3 mean amplitude than observed during error response condition (see table 1 and figure 9). This effect could be measured from the frontal and central electrodes. Correct trials and misbinding type 1 trials did not differ from each other in the P3 time window. Interestingly, correct trials and type 1 misbinding trials were different in the parietal-occipital electrodes in the P1 time window so that correct trials led to more positive P1 responses (table 1 and figure 8).

Table 1. Pairwise analysis of response type. Significant results are marked with asterisk.

<table>
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<th>Pair</th>
<th>Area</th>
<th>Component</th>
<th>Statistics</th>
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<tbody>
<tr>
<td>Correct-Error</td>
<td>Frontal</td>
<td>P1</td>
<td>t(10) = 0.908, p = 0.3852</td>
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<td></td>
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<td>N1</td>
<td>t(10) = 0.6807, p = 0.5115</td>
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<td>P3</td>
<td>t(10) = 4.1469, p = 0.00199*</td>
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<tr>
<td></td>
<td>Central</td>
<td>P1</td>
<td>t(10) = 0.6166, p = 0.5513</td>
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<td></td>
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<td>N1</td>
<td>t(10) = 0.6226, p = 0.5475</td>
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<td>P3</td>
<td>t(10) = 3.5664, p = 0.005126*</td>
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<td>Parietal-Occipital</td>
<td>P1</td>
<td>t(10) = 0.1648, p = 0.872</td>
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<td></td>
<td></td>
<td>N1</td>
<td>t(10) = 0.1922, p = 0.852</td>
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<td>P3</td>
<td>t(10) = 1.0871, p = 0.3025</td>
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<td>Correct- Misbinding type 1</td>
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<td>P1</td>
<td>t(10) = -0.1324, p = 0.8973</td>
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<td>t(10) = 0.4485, p = 0.6633</td>
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<td>P3</td>
<td>t(10) = -0.2533, p = 0.8052</td>
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<td>Central</td>
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<td>t(10) = 1.2627, p = 0.2354</td>
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<td>N1</td>
<td>t(10) = 0.5013, p = 0.627</td>
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### Temporal dynamics of misbinding

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<tr>
<td><strong>Parietal-Occipital</strong></td>
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<td>t(10)=2.5855, p=0.002*</td>
<td>t(10)=0.0484, p=0.962</td>
<td>t(10)=-1.3064, p=0.2207</td>
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<tr>
<td><strong>Error-Misbinding type 1</strong></td>
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<td>t(10)=1.4056, p=0.1901</td>
<td>t(10)=0.1471, p=0.886</td>
<td>t(10)=2.5855, p=0.002*</td>
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<td>t(10)=0.1017, p=0.336</td>
<td>t(10)=-0.2825, p=0.7833</td>
<td>t(10)=-2.8583, p=0.01701*</td>
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<tr>
<td><strong>Central</strong></td>
<td></td>
<td>t(10)=0.1471, p=0.886</td>
<td>t(10)=-0.0936, p=0.9273</td>
<td>t(10)=-2.7226, p=0.02147*</td>
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<td><strong>Parietal-Occipital</strong></td>
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<td>t(10)=-1.0107, p=0.336</td>
<td>t(10)=-0.2825, p=0.7833</td>
<td>t(10)=-2.8583, p=0.01701*</td>
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**Figure 8.** Event related potentials for correct, error and misbinding type 1 responses. Parietal and occipital electrodes.
Temporal dynamics of misbinding

Figure 9. Event related potentials for correct, error and misbinding type 1 responses. Central electrodes.

Figure 10. EEG topography for correct responses 0-600ms by 100ms steps.
Figure 11. EEG topography for error responses 0-600ms by 100ms step.

Figure 12. EEG topography for misbinding type 1 responses 0-600ms by 100ms step.
4.4 Discussion

Conducting the experiment 3 we hoped that ERPs would shed further light on the mechanisms of feature misbinding. We expected that misbinding 1 trials would be different from error trials whereas misbinding 2 trials are not. This expectation was met, differences between error and misbinding 1 trials were found in all observed areas whereas there were no differences between error and misbinding 2 trials. These results suggest that misbinding type 1 is a different phenomenon than error, whereas misbinding type 2 is not. However, the lack of differences between misbinding type 2 and error trials might also be a result of very little amount of trials of misbinding type 2 in our analysis. Differences between error and misbinding type 1 in P3 component suggest that misbinding can be a result of decision processes. However, if we consider that we also did not find any differences between error and correct trials in earlier components, it might be that the lack of significant differences between error and misbinding is a result of insufficient amount of trials, as it is known that more trials are required to find differences in early components (Luck, 2005).

We also investigated how misbinding type 1 differs from correct trials and found that there are respective differences in the amplitude of the P1 component. We speculate that the lower P1 amplitude in misbinding type 1 condition compared to correct trials could be a result of prestimulus alpha activity that is not corrected in 200 ms baseline. Namely, it is known that 1) prestimulus alpha phase is directly related to the amplitude of P1 (e.g. Gruber et al., 2005) and that 2) stimuli arriving at certain alpha phases can be processed more efficiently (Matthewson et al., 2009; Busch et al., 2009). Thus, when stimuli are presented at optimal alpha excitability phase, their features are processed and bound together quicker, which leads to correct responses, while the underlying alpha activity also leads to a stronger P1 response. If the presented stimuli arrive at a suboptimal phase, the processing of the second stimulus is facilitated and it can result in binding of the orientation of the second stimulus and the shape of the first stimulus. This speculation needs further investigation in subsequent studies or in the re-analysis of the present data.
4. GENERAL DISCUSSION

In the present work we presented three experiments concerning temporal dynamics and neural correlates of misbinding. We found that the phenomenon of misbinding can be robustly observed in simple visual masking experiments. Comparing the temporal dynamics of misbinding and error trials we found that misbinding is a different phenomenon from simple error trials. However, asymmetries observed between misbinding type 1 and misbinding type 2 suggest that only misbinding type 1 is a real perceptual phenomenon and suggest that misbinding type 2 is indistinguishable from error trials. These asymmetries might arise from the different temporal processing characteristics of the features. The behavioral results were supported by electrophysiological evidence that also revealed differences between misbinding type 1 and error trials and uncovered the asymmetries between misbinding types.

Illusory conjunctions have been classically studied in displays where several objects are presented simultaneously (e.g. Treisman & Gelade, 1980). However, many interesting phenomena arise when stimuli are presented in close succession from the same location. Visual masking is one of such key phenomena that have been used to study visual processes and visual consciousness (Bachmann, 1994, 2000). In the present experiments we followed the work of Hommuk and Bachmann (2009) and demonstrated that the visual masking paradigm can be successfully applied to study feature misbinding. In particular, we showed that when using stimuli that have several features, those features can get misbound between the stimuli. In our experiments, the subject could for example perceive an object which consists of the shape of the first stimulus and the orientation of the surface grating of the second stimulus.

In both cases, when many stimuli are presented simultaneously and when several stimuli are presented in succession from one location, the performance of the subjects is degraded. Therefore, it is only natural to doubt whether the “misbinding” phenomenon is a perceptual phenomenon at all: when the subjects see quickly flashing stimuli, they often are relatively unsure about their answers and they base a fair amount of their responses on their “intuitions” or “best guesses”. If “misbinding” responses arise only under such guessing conditions, they would not be a real phenomenon of perception, because the subjects would not really perceive objects
Temporal dynamics of misbinding

with misbound features - they just respond as if they would have perceived them (Donk, 1999). Therefore, one necessary next step for experiments such as ours is to measure the subjective clarity and confidence of the misbinding responses (Seth et al., 2008). Our present data suggest that as misbinding type 2 responses were not different from error trials in neither the temporal dynamics nor the ERP-correlates, they would be given with low confidence and low subjective clarity, i.e. they could indeed arise from guessing-responses driven by unconscious perception. On the other hand, as misbinding type 1 trials were consistently different from error trials in their temporal dynamics and in their electrophysiological correlates, we believe that misbinding type 1 responses would be given with similar confidence and subjective clarity as correct trials. However, we must acknowledge that although both behavioral and electrophysiological evidence suggests that only misbinding type 1 is a real perceptual phenomenon and misbinding type 2 is not, this could have been a result of insufficient amount of type 2 misbinding trials. Thus, it could be that subjects actually rate type 2 misbinding as clear percepts too and are confident in their perception. In sum, subsequent studies where the perceptual clarity and confidence is measured could be helpful for deciding whether type 2 misbinding is a perceptual phenomenon or just an artifact arising from simply guessing the feature combination that we label as “misbinding type 2” (Donk, 1999).

It is in general interesting to ask why such misbinding asymmetries (type 1 vs type 2 misbinding) arise and whether these two types of misbinding could indeed have different mechanisms. Indeed, it is known that shape is analysed higher up in the cortical hierarchy than orientation (e.g. Koch, 2004). Shape is analysed at the level of V4 and inferior temporal cortex, whereas orientation is one of the simplest visual features, analysed in V1. Thus, incoming stimuli will first activate the orientation selective areas and later the shape specific regions of the brain, which means that the shape of the first stimulus will be processed relatively close in time with the orientation of the second stimulus (the orientation of the first stimulus will be processed fastest and the shape information of the second stimulus is the latest to arrive). As objects have to be parsed and features have to be grouped in time somehow, it is to be expected that some errors will happen so that the orientation of the second stimulus and the shape of the first one are bound together and a misbound object is perceived instead of the real one. In this sense, studying feature misbinding with two stimuli presented in quick succession from the same location creates new interesting phenomena where time is the essential variable. Another explanation for the
asymmetry of misbinding could be based on the theory that for a stimulus to reach consciousness a modulatory process needs to be involved (Bachmann, 1984). As this modulatory system is slower, the second stimulus might in general gain advantage in perception, because when the timing conditions are optimal it “steals” some of the modulation evoked by the first stimulus (Bachmann, 2000). This theory explains why the second stimulus often prevails in consciousness when two successive stimuli are presented (Bachmann, 1984, 1994, 2000). It also explains why misbinding type 1 can happen: features of the first object which are processed slower (the shape of the first stimulus) and the earliest features of the second object (the orientation) could benefit more from this modulatory process and thus get brought to consciousness together as the misbound object. Another, and actually the opposite interpretation is that although neurons coding for shapes start firing later than the neurons coding for orientation, the representation of the shape higher up in the hierarchy is activated earlier and then this “Gestalt” information is sent back to the earlier cortical areas where this contour is filled in with surface information (Roelfsema, 2006; Gilad et al., 2013; see also Hommuk and Bachmann, 2009). As the surface information is processed later in time, it can be “overwritten” by the freshly arriving surface information of the second stimulus and thus the shape of the first stimulus is filled in with the surface of the second (Hommuk and Bachmann, 2009).

In this respect it is also interesting that the misbinding type 1 trials, where the shape of the first stimulus and the orientation of the second stimuli are misbound, are associated with significantly smaller P1 responses than the correct trials. As discussed in part 4.4, we believe that one possibility to explain this pattern of results is by relying on the fact that the P1 response is strongly dependent on the prestimulus alpha activity (Gruber et al., 2005). The ongoing alpha activity is a stochastic process and the stimulus will arrive sometimes at some alpha phase, sometimes at others. The phase of the ongoing alpha oscillation reflects windows of excitability in visual cortex (Matthewson et al., 2009). When the first stimulus arrives during the phase of the alpha oscillation where the cortex is more excitable the stimulus is processed efficiently and its features are bound quickly. At the same time, the same alpha phase is also related to the stronger P1 responses (Gruber et al., 2005). However, when the first stimulus arrives during a suboptimal alpha phase, it is not processed so quickly and its shape could be bound with the more efficiently processed orientation of the second stimulus. Thus, a type 2 misbinding response would arise and the P1 response would be smaller. However, for now this is only a conjecture, which could be
tested in the future. Hopefully subsequent studies and data analysis will show whether the
dynamics of prestimulus alpha activity could explain a part of the misbinding process.

In conclusion, in three simple masking experiments we studied misbinding in time domain.
We found that although the temporal dynamics and EEG correlates of misbinding differ from
simple errors, this only applies to misbinding type where the shape of the first stimulus is bound
with the orientation of the second stimulus. These results show that visual masking can be a
fruitful experimental paradigm for unraveling fundamental mechanisms of feature binding and
misbinding.
6. REFERENCES


7. ACKNOWLEDGEMENTS

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