Episodic context binding in task switching:
Evidence from amnesia

Beat Meier 1, 2, Alodie Rey-Mermet 1, 2, Todd S. Woodward 3, 4, René Müri 2, 5, & Klemens Gutbrod 2, 5

1 Department of Psychology, University of Bern, Switzerland
2 Center for Cognition, Learning & Memory, University of Bern, Switzerland
3 Department of Psychiatry, University of British Columbia, Vancouver, Canada
4 BC Mental Health and Addiction Research Institute, Vancouver, Canada
5 Department of Neurology, Division of Cognitive and Restorative Neurology, University Hospital of Bern, University of Bern, Switzerland.

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Corresponding Author:

Dr. Beat Meier, PhD
Department of Psychology
University of Bern,
Muesmattstr. 45
3000 Bern 9
Switzerland
Phone ++41 31 631 40 39
Fax ++41 31 631 82 12
Email: beat.meier@psy.unibe.ch
Abstract

The purpose of the present study was to investigate whether amnesic patients show a bivalency effect. The bivalency effect refers to the performance slowing that occurs when switching tasks and bivalent stimuli appear occasionally among univalent stimuli. According to the episodic context binding account, bivalent stimuli create a conflict-loaded context that is re-activated on subsequent trials and thus it is assumed that it depends on memory binding processes. Given the profound memory deficit in amnesia, we hypothesized that the bivalency effect would be largely reduced in amnesic patients. We tested sixteen severely amnesic patients and a control group with a paradigm requiring predictable alternations between three simple cognitive tasks, with bivalent stimuli occasionally occurring on one of these tasks. The results showed the typical bivalency effect for the control group, that is, a generalized slowing for each task. In contrast, for amnesic patients, only a short-lived slowing was present on the task that followed immediately after a bivalent stimulus, indicating that the binding between tasks and context was impaired in amnesic patients.

Keywords: cognitive control; conflict processing; anterior cingulate cortex; medial temporal lobe; bivalent stimuli; univalent stimuli
1. Introduction

Cognitive control is the ability to maintain current goal representations in face of conflict. It enables selection of goal-relevant features while suppressing distracting ones (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Botvinick, Cohen, & Carter, 2004). However, how cognitive control is exerted to adjust and fine-tune performance in face of conflict and more generally, whether cognitive control effects reflect the operation of a cognitive control system or instead should rather be conceptualized as memory binding processes (Altman & Gray, 2008; Egner, 2007; Hommel, 2004; Mayr, Awh, & Laurey, 2003; Verguts & Notebaert, 2009), or both, remains an open question. Here we investigate one particular effect of cognitive control -- the bivalency effect. We specifically test whether intact memory processes are required by comparing performance of amnesic patients to a healthy control group.

The bivalency effect refers to the phenomenon that, when people switch between a series of tasks and occasionally one of these tasks involves bivalent stimuli, that is, stimuli with relevant features to two tasks, subsequent performance is slowed. Critically, the slowing occurs even on trials that have no overlapping features with the bivalent stimuli. For example, in an initial study by Woodward, Meier, Tipper, and Graf (2003), participants switched between a parity decision (odd vs. even numerals), a colour decision (red vs. blue symbols), and a case decision (uppercase vs. lowercase letters), repeatedly and in a fixed order. On most of the trials the stimuli were univalent (i.e., black numerals for the parity decision, coloured shapes for the colour decision, and black letters for the case decision). However, occasionally, on some case decisions the letters were presented in colour, thus turning them into bivalent stimuli. The results showed that performance was not only slowed for these bivalent stimuli, but also for all the subsequent univalent trials, even those with stimuli that shared no relevant features with the bivalent stimuli (i.e., the parity decisions). Woodward et al. noted that this result challenges task-switching theories that focus primarily on bottom-up processes, that is, processes initiated and guided by the stimuli and their particular features (e.g., Allport & Wylie, 2000; Meiran, 2008; Monsell, Yeung, & Azuma, 2000; Rogers & Monsell,
1995). These theories can account for the slowing in response to univalent stimuli, which share a relevant feature with the bivalent stimuli (i.e., those used for case and colour decisions). However, they cannot account for the slowing in response to univalent stimuli, which share no features with the bivalent stimuli (i.e., those used for the parity decisions).

Follow-up studies have established that the bivalency effect is robust across a variety of different tasks, across different modalities and bivalent stimuli, and that it leads to an enduring slowing, affecting performance on univalent stimuli up to twenty seconds after the occurrence of a bivalent stimulus (Meier, Woodward, Rey-Mermet, & Graf, 2009). Moreover, it is independent from response set priming and it occurs for repetition trials that have overlapping stimulus features (Rey-Mermet & Meier, 2012a; 2012b). Theoretically, it is possible that when encountering bivalent stimuli, the cognitive system adjusts control, which results in a more cautious response style. The underlying processes may be related to binding processes that are consecutively associating stimuli, tasks and the context in which they occur. When a bivalent stimulus occurs, stimuli and tasks are associated with a context-loaded context. On subsequent trials, the reactivation of this episodic context representation interferes with performance on univalent trials. This account can explain why performance is slowed even on those univalent trials with non-overlapping stimulus features. Critically, memory processes are required for the expression of the bivalency effect (Meier et al., 2009; Meier & Rey-Mermet, 2012; Rey-Mermet & Meier, 2012a).

The available evidence from functional magnetic resonance imaging (fMRI) supports the notion that the bivalency effect reflects an adjustment of cognitive control. Woodward, Metzak, Meier, and Holroyd (2008) contrasted univalent stimuli from a condition with purely univalent stimuli and univalent stimuli from a condition in which bivalent stimuli were occasionally intermixed on one of the tasks. The results showed that the bivalency effect was associated with activation in the dorsal anterior cingulate cortex (dACC), a brain area recruited for the adjustment of cognitive control (see Botvinick et al., 2001). Similarly, using event-related potentials, Grundy et al. (2011), found amplitude differences at frontal electrodes within time
windows of 275-450ms and 500-550ms. They interpreted these modulations as “suppression of processing carried over from irrelevant cues”. Moreover, consistent with the fMRI results, source dipole analyses revealed dipole locations at or close to the dACC. Thus, the bivalency effect is associated with activations in brain areas that signal adjustment of cognitive control triggered by conflict processing. Here, we investigate what exactly triggers conflict in the absence of bivalent stimuli, that is, when processing purely univalent stimuli. We have suggested previously that the re-activation of a representation of conflict that has been build up by processing the conflict-loaded task-triplet is a likely explanation. According to this “episodic context binding” hypothesis, each sequential presentation of a task-triplet (e.g., parity – color – case) represents a separate context. When a bivalent stimulus is presented on one of these tasks, the conflict that is triggered spreads to the representation of the whole context. For the next task-triplet, this representation is reactivated and performance is slowed for all the stimuli, even for those that have no overlapping features with the bivalent stimulus (Meier & Rey-Mermet, 2012).

According to this explanation, we would expect that binding processes take place on each trial (i.e., stimuli, tasks, and task-triplets acquire a history, cf. Meier et al., 2009; Waszak, Hommel, & Allport, 2003) and thus, we would also predict memory-related brain activations. However, when contrasting univalent blocks with and without bivalent stimuli in an fMRI or ERP-study these activations cancel each other out, explaining the absence of memory-related brain activations in these studies. Here we specifically investigated the involvement of memory processes in the bivalency effect by testing a sample of amnesic patients who have a profound memory deficit.

We tested a group of 16 severely amnesic patients and a healthy control group. During three blocks, all participants performed a parity decision on numerals (odd vs. even), a colour decision on symbols (red vs. blue), and a case decision on letters (upper- vs. lowercase). In the first and third blocks (the purely univalent blocks), all stimuli were univalent. In the second block (the mixed block), some letters for the case decisions were presented in colour (red or blue), which turned them into bivalent stimuli. Our motivation for
involving amnesic patients was to test whether their profound deficit in memory binding, in particular binding an event to a particular context (e.g., Chun & Phelps, 1999; Hannula, Tranel, & Cohen, 2006; Pascalis, Hunkin, Bachevalier, & Mayes, 2009), would affect the bivalency effect. Several studies have demonstrated that the binding deficit in amnesia is not restricted to long-term memory, but also affects short-term bindings such as those thought to be involved in the bivalency effect (Olson, Moore, Stark, & Chaterjee, 2006; Ezzyat & Olson, 2008; Olson, Moses, Riggs, & Ryan, 2012 for a recent review). Thus, we hypothesized that if episodic context binding is involved in the bivalency effect, amnesic patients will show a considerable reduction in the magnitude of the bivalency effect. In contrast, if episodic context binding is not necessary for the formation of the bivalency effect, then amnesic patients will show a normal bivalency effect.

2. Method

2.1 Participants

Sixteen severely amnesic patients participated in this study (age between 53 and 75 years, \(M = 63.2\); education nine to fifteen years, \(M = 12.7\)). The criterion for inclusion was the presence of a lesion in memory-critical areas (cf. Aggleton, 2008), severe, circumscribed, and chronic memory impairment (time since onset > 3 months) and German as the first language. Table 1 provides an overview of the demographic characteristics, the aetiology, and time since onset. Seven patients had damage to the basal forebrain following a bleeding from a ruptured aneurysm of the anterior communicating artery (2, 6, 9, 11, and 13) or a herpes encephalitis (1, 4). Four patients showed amnesia following an episode of hypoxia due to cardiac arrest (3, 7 and 14) or a complication during birth (16, i.e. this patient suffered from a developmental amnesia). Although magnetic resonance imaging (MRI) did not reveal any visible brain damage in these hypoxic patients, they were included in the study since hypoxia is known to cause primarily damage to the hippocampus (cf. Zola-Morgan, Squire, & Amaral, 1986). In the patient with developmental amnesia a volumetric analyses revealed an isolated atrophy of 40% of the hippocampus.
bilateral compared to healthy controls. Two other patients had suffered thalamic infarction (8 and 12). One patient became amnesic following bleeding from an aneurysm of the right middle cerebral artery (5), one due to damage to the hippocampus following lupus erythematosus (10; cf. Schnider, Bassetti, Gutbrod, & Ozdoba, 1995) and one patient had suffered multiple insults in the vertebrobasilar system (15). Figure 1 shows overlap maps of brain lesions drawn on standard templates using MRIcro (Rorden & Brett, 2000) based on the latest available magnetic resonance imaging. The lesions of the four hypoxic patients were not drawn because no damage was visible on MRI. It is apparent from Figure 1 that the lesions of all the remaining patients comprised memory-critical structures with the highest overlap either in the basal forebrain, the hippocampus or the anterior thalamus.

Nine of the patients had already participated in a study by Gutbrod et al. (2006) for which a standard neuropsychological test battery was administered that revealed normal (or only slightly reduced) performance in rule deduction, control of interference, verbal fluency and design fluency for all the patients. For the present study, an additional examination confirmed that amnesic patients had normal or near normal intelligence. In contrast, all had severe impairments in tests of verbal learning and memory. These results are presented in Table 2. The control group consisted of sixteen healthy persons matched to the amnesic group with regard to age, years of education and handedness. The study was approved by the local ethics committee and all participants gave written informed consent.

2.2 Materials

To investigate the bivalency effect, we used the same method as Meier et al. (2009; Experiment 1) with predictable switches between parity-, colour-, and case-decisions. For parity decisions, the stimuli were the numerals 1 through 8, each displayed in black and in triplicate (e.g., 777). For colour decisions, the stimuli were the symbols $, %, &, #, displayed in triplicate (e.g., &&&), and either in blue or red. For case decisions, the stimuli were the consonants p, n, s, and d, each displayed in black and in triplicate (e.g., ddd), in either upper- or lowercase. We created a set of eight bivalent stimuli by presenting the same
four consonants (p, n, s, and d) in triplicates, either in blue or red colour. In order to control for performance on bivalent stimuli, uppercase and lowercase stimuli were coloured such that they required an incongruent response. The stimuli were presented at the centre of the computer screen in 60-point Times New Roman font.

2.3 Procedure

Participants were tested individually. They were informed that the experiment involved three different tasks: parity decisions about numerals, colour decisions about symbols, and case decisions about letters. They were instructed to press one of two keys on the keyboard with their left and right index fingers, respectively, for each of the three tasks. The mapping information, printed on paper, was displayed below the computer screen throughout the experiment. Participants were further informed that, on some of the case decisions, the letters would be presented in colour. They were specifically instructed to ignore the colour and to focus on making case decisions.

After these instructions, a block of 30 task-triplets was presented for practice. Each task-triplet required making a parity decision, a colour decision, and a case decision, always in the same order, as illustrated in Figure 2. The stimulus for each task was displayed until the participant responded. Then, the screen was cleared and after a 500 ms inter-stimulus interval, the next stimulus appeared. After each task-triplet, an additional blank interval of 500 ms was included, resulting in an interval of 1000 ms. After the practice block and a brief break, each participant completed three experimental blocks, each with 30 task-triplets, without break between blocks.

For the first and third blocks (the purely univalent blocks), only univalent stimuli were presented. For the second block (the mixed block), stimuli were univalent except on 20% of the case decisions in which bivalent stimuli (i.e., coloured letters) appeared. The specific letter selected for this purpose was determined randomly and without replacement. Task-triplets with bivalent stimuli were evenly interspersed
among the 30 task-triplets of the block; they occurred on every fifth task-triplet, specifically in the 3rd, 8th, 13th, 18th, 23th, and 28th triplets. The entire experiment lasted about 15 minutes.

2.4 Data analysis

For each participant, the error rates and the median decision times (DTs) for correct responses were computed for each task and each block. For the mixed block, error rates and median DTs for univalent and bivalent case decisions were computed separately. An alpha level of 0.05 was used for all statistical tests. Greenhouse-Geisser corrections are reported where appropriate and effect sizes are expressed as partial $\eta^2$ values.

3. Results

3.1 Performance on univalent stimuli

Consistent with the instruction to respond as quickly and accurately as possible, accuracy was high on all univalent stimuli in both groups. For amnesic patients, mean accuracy was 94% ($SE = 0.02$), 93% ($SE = 0.02$), and 94% ($SE = 0.02$) for blocks 1 to 3, respectively. For the control group, mean accuracy was invariable 98% ($SE = 0.02$) for each of the three blocks. An analysis of variance (ANOVA) with Group and Block showed no significant effects, all $F$s < 3.71, $p$s > .05, $\eta^2$ < .11.

Our main objective was to examine whether the amnesic group showed the bivalency effect, that is, the slowing on univalent trials in the mixed block, compared to the purely univalent block. Figure 3 shows the means of the median DTs on univalent trials with the associated standard errors. For analyses, we averaged the data from the purely univalent blocks 1 and 3 for each task to account for general training effects. A mixed three-factorial ANOVA with Block (purely univalent, mixed) and Task (parity, colour, case) as within-subject factors and Group (amnesic, control) as a between-subjects factor revealed a significant main effect of Group, $F (1, 30) = 13.22$, $p < .01$, $\eta^2 = .31$, caused by slower responses in the amnesic group than in the control group. Furthermore, it also showed a significant interaction between Block and Task, $F$
(2, 60) = 3.35, \( p < .05, \eta^2 = .10 \), reflecting a larger bivalency effect in parity decisions than in colour or case decisions. Critically, however, the interaction between Block and Group was also significant, \( F (1, 30) = 4.34, p < .05, \eta^2 = .13 \). This was due to the fact, that the control group showed a bivalency effect (67 ms), whereas the amnesic group did not (-6 ms). Separate follow-up ANOVAs with Block and Task as within-subject factors confirmed this observation. For the amnesic patients, this ANOVA gave no significant effects, \( F_s < 2.71, ps > .05, \eta^2 < .15 \). In contrast, for the control group, the ANOVA showed a significant main effect of Task, \( F (2, 30) = 7.08, p < .01, \eta^2 = .32 \), and, more importantly, of Block, \( F (1, 15) = 13.96, p < .01, \eta^2 = .48 \). The interaction was not significant, \( F (2, 30) = 0.86, p = .43, \eta^2 = .05 \).

As illustrated in Figure 3, for the control group a consistent bivalency effect (i.e., a performance slowing in the mixed block compared to the purely univalent block) emerged for all three tasks. In contrast, for the amnesic group, the difference was not reliable. However, on the parity decisions, although not statistically significant, the results suggest a performance slowing in the mixed block compared to the purely univalent blocks. To follow up on the possibility that the amnesic patients show at least a partial bivalency effect, we tested the temporal endurance of this slowing. Typically, the bivalency effect persists across several subsequent decisions following a bivalent stimulus (Meier et al., 2009). Thus, a sustained slowing would be indicative of a residual expression of the bivalency effect. In contrast, if the slowing dissipated quickly, this would rather indicate an orienting response to an infrequent event. A short-lived performance slowing after an infrequent event (such as a bivalent stimulus) is typically characterized as a time-consuming orientation to the infrequent event followed by a reorientation to the frequent events (e.g., Barcelo, Escera, Corral, & Periáñez, 2006; Notebaert et al., 2009).

For this purpose, we computed the median DTs for each task-triplet following the bivalent case decisions, separately for each task. As a bivalent stimulus was presented on each fifth task-triplet in the mixed block, we designated this task-triplet as triplet N and the first triplet following the bivalent stimuli with
the label N+1, etc. Figure 4 depicts the trajectory of DTs for each task on the task-triplets N+1 to N+4 from the mixed block and the corresponding DTs from the purely univalent block, separately for the patients and the control group. From Figure 4A, it seems that the amnesic patients showed slower DTs for the parity decision on task-triplet N+1 in Block 2. This was confirmed by a \( t \)-test, \( t(15) = 3.61, p < .01 \). In contrast, no slowing was evident for any other task in any other task-triplet. Figure 4B reveals that the results of the control group showed a reduction of the size of the bivalency effect across task-triplets as expected. Across tasks, the effect was 267 ms, \( t(15) = 4.26, p < .01 \) for N+1, 116 ms, \( t(15) = 3.46, p < .01 \) for N+2, 48 ms, \( t(15) = 1.35, p = .09 \) for N+3, and 43 ms, \( t(15) = 1.81, p < .05 \) for N+4. This pattern is consistent with our previous findings (Meier et al., 2009).

3.2 Performance on bivalent stimuli

The examination of case decisions in the mixed block showed that accuracy was higher for univalent stimuli (i.e., on black letters) compared to bivalent stimuli (i.e., on coloured letters). For the patients, mean accuracy was 92% (SE = 0.03) for univalent stimuli and 44% (SE = 0.09) for bivalent stimuli; for the control group, mean accuracy was 98% (SE = 0.01) for univalent stimuli and 86% (SE = 0.04) for bivalent stimuli. A two-factorial ANOVA with stimulus valence (univalent case, bivalent case) as a within-subject factor and group (amnesic, control) as a between-subjects factor showed significant main effects of stimulus valence, \( F(1, 30) = 34.97, p < .001, \eta^2 = .54 \), and group, \( F(1, 30) = 20.14, p < .001, \eta^2 = .4 \), and a significant interaction, \( F(1, 30) = 12.52, p < .001, \eta^2 = .29 \).

Both groups made faster responses to univalent than to bivalent case decisions. DTs for univalent stimuli were 1362 ms (SE = 147) for patients and 905 ms (SE = 51) for controls. DTs for bivalent stimuli were 1541 ms (SE = 174) for patients and 1402 ms (SE = 202) for controls. The two-factorial ANOVA with stimulus valence and group showed a significant main effect of stimulus valence, \( F(1, 26) = 10.12, p < .01, \eta^2 = .28 \). No other effect was significant, \( Fs < 2.33, ps > .05, \eta^2 < .08 \).
4. Discussion

The purpose of this study was to investigate whether amnesic patients show a bivalency effect. According to the episodic context binding account, memory processes are required to form a representation of the context in which a bivalent stimulus occurs. This representation is reactivated on subsequent trials and slows down performance on univalent trials. This account can explain why performance is slowed even on those univalent trials with non-overlapping stimulus features. We reasoned that due to their severe memory impairment amnesic patients would not be able to create a context representation and thus would not show the bivalency effect. The results confirmed our expectations. The control group showed a consistent bivalency effect, that is, a performance slowing on univalent trials after bivalent stimuli were presented occasionally. In contrast, no such effect was present in amnesic patients.

The amnesic patients showed a striking performance deficit on bivalent stimuli. However, as we have used bivalent stimuli with incompatible responses this indicates that the amnesic patients responded to stimulus colour which is the more dominant feature compared to case. The fact that the patients performed less accurate according to the initial instructions (following the sequence of the tasks) may thus be related to their severe memory problems. That is, it is possible that they simply forgot what task to perform when they encountered a bivalent stimulus or that it was harder for them to keep track with the sequence of the tasks. It is important to note, however, that even when they were not able to follow the instructions in terms of following the sequence of the tasks, they still responded to bivalent stimuli even when they responded to colour rather than case.

It is also noteworthy that performance of amnesic patients was generally slower than the control group. A possible explanation for this slowing is that the patients had difficulties remembering the response mapping for the three tasks. Hence, they had to consult the reminder that was positioned below the computer screen more often. This may also explain why the patients did not show faster decision times for the colour and parity decisions compared to case decision as the control group. One may wonder whether
the occasional demand to consult the response mapping may have also abolished the bivalency effect. The fact that patients were substantially slowed on the case decisions that involved bivalent stimuli (compared to the case decisions with univalent stimuli) speaks against this interpretation. This slowing demonstrates that the patients were sensitive to the conflict induced by bivalent stimuli. If the demand to consult the response mappings were the source for the lack of a bivalency effect then they would not be expected to show (bivalent) stimulus-specific slowing.

Similarly, a general deficit in decision-making speed cannot explain the whole pattern of results. As noted, performance was slowed on bivalent compared to univalent stimuli, thus demonstrating a Stroop-like effect. Second, performance was also slowed on the decision that immediately followed the bivalent stimulus. This short-lived after-effect can be considered as an orienting response towards an infrequent stimulus. It may also be related to post-error slowing as amnesic patients performed less accurate on bivalent stimuli than on univalent stimuli and also, compared to the control group (cf. Barcelo et al., 2006; Notebaert et al, 2009). We would like to emphasize that the bivalency effect inherently involves a slowing related to an orienting response, however this short-lived slowing cannot explain the enduring characteristic of the bivalency effect. Notably, both the slowing on bivalent stimuli and the resulting orienting response are data-driven effects that are triggered directly by the bivalent stimuli. On a neuroanatomical level, it is likely that they are related to activations in the dACC, a brain area intact in the amnesic patients.

Most importantly, the results showed that after these initial effects, subsequent performance of the patients was not further affected, that is, the amnesic patients showed no bivalency effect. The results suggest that this is due to a failure to build up a contextual conflict representation and, consequently, no such representation was available for re-activation. We suggest that generally, episodic context includes the whole sequence of the three different decision tasks (e.g., parity – color – case). Thus, a particular decision task always activates the representation of the whole task-triplet. When a bivalent stimulus is presented on one of these tasks, the conflict that is triggered spreads to the representation of the whole
context. For the next task-triplet, this representation is reactivated and performance is slowed for all the stimuli, even for those that have no overlapping features with the bivalent stimulus. These representations use coding mechanisms that allow the system to separate closely related representations which allows for clear boundaries between similar contexts. There is considerable evidence that hippocampal dysfunction manifests as a loss of the ability to encode contexts and to discriminate amongst them (cf., Nadel, 2008).

Thus, we would argue that the failure to build up such a representation in the amnesic patients was due to their severe memory deficit. It is known that the hippocampus is critically involved in memory binding, even over a relatively short time (e.g., Olson et al., 2006; 2012). However, because of the heterogeneous aetiology of the patient group, it is difficult to locate the specific brain areas required for this type of episodic context binding. The lesions of the patients were not restricted to the hippocampus, but variably included neighbouring structures in the medial temporal lobe and anterior thalamus or in the basal forebrain. There is a current discussion about the specific involvement of the hippocampus for short-term memory binding (e.g., Baddeley, Allen, & Rich, 2009, for a critical discussion). Our results suggest that a binding deficit may not be specific for hippocampal amnesia, but may be related to more general lesions, including the basal forebrain. The latter finding is particularly interesting because it would indicate that these brain areas are not only involved in the retrieval of time-contextual information but also in episodic context binding (cf. De Rosa, Desmond, Anderson, Pfefferbaum, & Sullivan, 2004; Fujii et al., 2002).

However, an alternative possibility is that forgetting may play a more general role. With accuracy emphasis, the subthalamic nucleus (STN) receives additional excitatory input from frontal areas, which produces slower and more accurate responses. Specifically, Frank, Scheres, and Sherman (2007) suggested that upon the detection of a conflict the anterior cingulate cortex provides input to STN which implements a more careful mode of responding. Thus, it is possible that even in patients STN is triggered upon encountering conflict; however, they forget to keep up a more careful mode of responding. Moreover, cortical-subcortical systems have in general an important role for regulating cognitive control (e.g.
Forstmann et al., 2008; Ivanoff, Branning, & Marois, 2008; van Veen, Krug & Carter, 2008). In particular, a disruption of these circuits is more likely in the patients with lesions to the basal forebrain. Not being able to keep up a more careful response mode would also result in an impossibility to reactivate a representation of conflict on subsequent univalent stimuli and thus would lead to the same response pattern as a binding deficit which is more likely for those patients with hippocampal lesions.

A further interesting result of the present study is the finding of a substantial bivalency effect in the control group. Although previous work has established the bivalency effect, the evidence was exclusively based on findings from undergraduate students. The present study indicates that the effect is stable and still present in older adults. This suggests that the bivalency effect represents a rather general phenomenon. The investigation of this effect across the lifespan is an interesting avenue for future research.

In summary, the present study demonstrates that memory processes are essential in the formation of the bivalency effect. They indicate that severe memory impairment results in a failure to build up a contextual representation of the conflict induced by bivalent stimuli. By consequence, there is no representation available for activation on subsequent trials that would interfere with performance. Thus, the present study suggests that memory binding processes are involved in cognitive control effects. The main novel finding is that memory binding does not only involve stimulus, response, or task features, but it can also involve the context in which stimuli, responses, and tasks are processed. Moreover, the lack of a bivalency effect in amnesic patients is not due to impairment in top-down “executive” functions, but rather due to severe problems in memory.
5. References


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Footnotes

1 These results are presented in the Supplementary Table 1.

2 Individual data are presented in the Supplementary Figure 1.

3 Three patients (Patients 4, 9 and 15) never responded to bivalent stimuli with a case decision. In order to exclude the possibility that the differences in the results were due to these patients we reran all the analyses without them and their corresponding matched control persons. These analyses showed the same results, supporting the lack of a bivalency effect in amnesic patients.
Acknowledgement

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Table 1

Demographic characteristic

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<td>M</td>
<td>Infarction</td>
<td>8</td>
</tr>
<tr>
<td>13</td>
<td>52</td>
<td>11</td>
<td>M</td>
<td>Haemorrhage</td>
<td>11</td>
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<tr>
<td>14</td>
<td>58</td>
<td>12</td>
<td>M</td>
<td>Hypoxia</td>
<td>5</td>
</tr>
<tr>
<td>15</td>
<td>22</td>
<td>11</td>
<td>M</td>
<td>Infarction</td>
<td>5</td>
</tr>
<tr>
<td>16</td>
<td>39</td>
<td>17</td>
<td>M</td>
<td>Hypoxia</td>
<td>birth</td>
</tr>
</tbody>
</table>

Mean 50.9 ± 11.8  14.4 ± 2.9  13 M  3 F

Control group

Mean 51.3 ± 11.7  15.0 ± 2.3  12 M  4 F

Note. F = female; M = male.
Table 2

Neuropsychological results of amnesic patients: Intelligence and verbal memory (raw scores with age-adjusted percentiles in parenthesis)

<table>
<thead>
<tr>
<th>Patient ID</th>
<th>IQ</th>
<th>Word list learning</th>
<th>Long delay free recall</th>
<th>Recognition (Hits - FA)c</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>94a</td>
<td>26 (&lt; 1)</td>
<td>0 (&lt; 1)</td>
<td>-2 (&lt; 1)</td>
</tr>
<tr>
<td>2</td>
<td>119a</td>
<td>27 (&lt; 1)</td>
<td>0 (&lt; 1)</td>
<td>8 (3.2)</td>
</tr>
<tr>
<td>3</td>
<td>89a</td>
<td>30 (&lt; 1)</td>
<td>0 (&lt; 1)</td>
<td>-5 (&lt; 1)</td>
</tr>
<tr>
<td>4</td>
<td>118a</td>
<td>30 (&lt; 1)</td>
<td>0 (&lt; 1)</td>
<td>-6 (&lt; 1)</td>
</tr>
<tr>
<td>5</td>
<td>128a</td>
<td>29 (&lt; 1)</td>
<td>1 (&lt; 1)</td>
<td>6 (1.1)</td>
</tr>
<tr>
<td>6</td>
<td>126a</td>
<td>30 (&lt; 1)</td>
<td>3 (&lt; 1)</td>
<td>-4 (&lt; 1)</td>
</tr>
<tr>
<td>7</td>
<td>126a</td>
<td>36 (3.7)</td>
<td>1 (&lt; 1)</td>
<td>-6 (&lt; 1)</td>
</tr>
<tr>
<td>8</td>
<td>102a</td>
<td>24 (&lt; 1)</td>
<td>0 (&lt; 1)</td>
<td>0 (&lt; 1)</td>
</tr>
<tr>
<td>9</td>
<td>115a</td>
<td>30 (&lt; 1)</td>
<td>0 (&lt; 1)</td>
<td>9 (&lt; 1)</td>
</tr>
<tr>
<td>10</td>
<td>91a</td>
<td>25 (&lt;1)</td>
<td>1 (&lt; 1)</td>
<td>-3 (3.2)</td>
</tr>
<tr>
<td>11</td>
<td>128b</td>
<td>33 (&lt; 1)</td>
<td>0 (&lt; 1)</td>
<td>2 (&lt; 1)</td>
</tr>
<tr>
<td>12</td>
<td>115b</td>
<td>37 (7.9)</td>
<td>5 (3)</td>
<td>-1 (&lt;1)</td>
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<tr>
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<td>85b</td>
<td>25 (&lt; 1)</td>
<td>1 (&lt; 1)</td>
<td>1 (&lt; 1)</td>
</tr>
<tr>
<td>14</td>
<td>95b</td>
<td>28 (&lt; 1)</td>
<td>3 (&lt; 1)</td>
<td>-1 (&lt; 1)</td>
</tr>
<tr>
<td>15</td>
<td>79b</td>
<td>23 (&lt; 1)</td>
<td>1 (&lt; 1)</td>
<td>0 (&lt; 1)</td>
</tr>
<tr>
<td>16</td>
<td>128a</td>
<td>37 (2.3)</td>
<td>1 (&lt; 1)</td>
<td>8 (2)</td>
</tr>
<tr>
<td>Mean</td>
<td>108.6 ± 17.5</td>
<td>29.4 ± 4.5</td>
<td>1.1 ± 1.4</td>
<td>.8 ± 4.7</td>
</tr>
</tbody>
</table>

Note. a Wechsler Adult Intelligence Scale-Revised (WAIS-R; Tewes, 1991), b Mehrfachwahl-Wortschatztest (MWT-A, a German equivalent of the North American Adult Reading Test; Lehrl, Merz, Burkhard, & Fischer, 1991). Both scales are standardized with a population mean of 100 and a standard deviation of 15; Verbal memory as assessed with the Rey Auditory Verbal Learning Test (RAVLT; Helmstädtter, Lendt, & Lux, 2001). c False Alarms
Figure 1. Overlap maps showing the region of highest overlap for the amnesic group. Lesions were drawn on templates with Z-values -24, -20, -16, and 12 in Talairach space (Talairach & Tournoux, 1988). The colour scale indicates the absolute number of shared lesions for every damaged area. Note that the lesions of four patients with hypoxic brain damage were not drawn because no damage was visible on MRI. The left hemisphere is shown on the right side and vice versa.
Figure 2. Example of two consecutive univalent task-triplets. On a bivalent task-triplet (not pictured here), the letters were presented in colour (either in blue or red).
Figure 3. DTs for univalent stimuli across blocks (blocks 1 and 3 purely univalent, block 2 mixed), for amnesic patients (empty symbols) and the control group (filled symbols), on parity decisions (diamonds), colour decisions (circles) and case decisions (squares). Error bars represent standard errors.
Figure 4. Trajectory of DTs in the task-triplets following bivalent case decisions in the mixed block (filled circles) and the corresponding decisions from the purely univalent block (empty circles) A: amnesic patients, B: control group. Error bars represent standard errors.