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Capture by misleading information and its false acceptance in patients with traumatic brain injury

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Exposure to misleading information, presented after a critical episode, can alter or impair memory reports about that episode. Here, we examine vulnerability to misleading information in patients with traumatic brain injury (TBI). The ability to initiate an effective retrieval strategy and inhibit irrelevant or interfering information requires participation from the prefrontal cortices, which are susceptible to damage following brain injury. We report that TBI patients are more prone to interference effects produced by misleading information during a cued-recall task and are more likely to accept this information as the product of 'remembering' compared with healthy controls. The results are consistent with a model proposing that patients are captured by highly accessible responses eliminating their opportunity to engage in recollection. Correlations between the cued-recall interference task and other executive measures helped elucidate the processes underlying 'capture'. In TBI patients, reduced recollection produced by a misleading prime was associated with impaired prospective remembering when engaged in a background task. A common functional deficit that may underlie poor performance on both tasks is the failure to inhibit previously relevant but currently irrelevant information. Subjective reports pertaining to the subject's cued-recall response were indexed by electrodermal activity. In control subjects, larger skin conductance responses (SCRs) were associated with a greater frequency of guess reports, suggesting that SCRs provide a marker for uncertainty regarding the candidacy of a selected response. TBI patients did not show this relationship, suggesting that impairments of post-retrieval evaluation might also underlie greater false acceptance of misinformation. Discussion focuses on the role of the prefrontal cortex and cognitive processes that mediate the selection and evaluation of memories.

Keywords: misinformation; capture; false memory; traumatic brain injury; prefrontal cortices

Abbreviations: CFQ = cognitive failures questionnaire; EDA = electrodermal activity; GCS = Glasgow coma scale; HAD = hospital anxiety depression; PTA = post-traumatic amnesia; R-SAT = revised strategy application test; SCR = skin conductance response; TBI = traumatic brain injury

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Introduction

When recollection fails us we are often fortunate in that we can rely on more automatic influences of memory to provide us with the most accessible, and often correct, response. However, if misleading information is presented when our recollection fails we may inadvertently and falsely provide this information as the most accessible alternative and exhibit false remembering. For the most part, it is likely that proneness to deception from misinformation will be most effective when an individual fails to recollect a prior episode and reverts to accessible alternatives. Nevertheless, for people with frontal lobe impairments, who lack the capacity to adopt an efficient retrieval strategy or the interference

control processes to inhibit irrelevant information, a heightened vulnerability to deception, persuasion or undue influence is a considerable risk.

Following traumatic brain injury (TBI), diffuse white matter damage typically co-occurs with more focal lesions or haemorrhages predominating in the frontal cortex (Mattson and Levin, 1990; Richardson, 2000). Fronto-striatal pathways and the connections between prefrontal regions and the association areas of the posterior cortex are vulnerable to disruption (Goldman-Rakic, 1987; Fuster, 1989). Brain trauma of this kind has negative implications for maintaining a focused retrieval mode and inhibiting irrelevant or interfering information. The role of the prefrontal cortex is important for both the initiation and maintenance of retrieval strategies (Moscovitch, 1992; Fletcher *et al.*, 1997). Neuroimaging has demonstrated that regions of the right prefrontal cortex, including dorsolateral regions, fronto-polar areas and the frontal operculum, are key neuroanatomical correlates of an episodic 'retrieval mode' (REMO) (Lepage *et al.*, 2000). Moreover, a subset of prefrontal regions, including the right middle frontal gyrus and left inferior frontal gyrus, has been associated with resolving interference from task-irrelevant information (Bunge *et al.*, 2001). Interference control processes may enable greater efficiency for REMO sites within the prefrontal cortex facilitating frontally mediated bias of competing streams of information processing.

Neuropsychological evidence demonstrates that patients who have sustained frontal lobe damage exhibit disproportionate impairments on tasks that require an effective retrieval strategy and inhibition of previous memory associations, such as word fluency tasks and proactive interference tasks (Shimanura, 1995). Furthermore, case study evidence (Delbecq-Derouesne *et al.*, 1990; Parkin *et al.*, 1996) shows that frontal patients who have suffered an anterior communicating artery aneurysm (causing damage to ventromedial regions and adjoining areas) experience false recall of material that had not been studied previously. These 'intrusions' may occur because patients fail to engage a sufficient retrieval focus to activate specific details of the study episode and are more susceptible to general accessibility of non-target or related episodes that may interfere with the sought-after target. These frontal patients may lack the interference control processes necessary to avoid capture by inappropriate memory associations. A similar reasoning applies in the attention domain under conditions that require goal maintenance or the suppression of irrelevant stimulus information exemplified in the Stroop task. In this task, reading a word (the irrelevant information) eliminates an opportunity to name the colour of the printed word (the intended goal) (MacLeod, 1992). Both TBI patients (Leahy and Lam, 1998; Rios *et al.*, 2004) and patients with focal prefrontal lesions (Stuss *et al.*, 2001) show an increased number of word reading errors on the Stroop task, suggesting that goal neglect or poor inhibitory control underlie these errors. Together, these findings suggest that frontal patients and TBI patients are particularly vulnerable to intrusions in the memory and attention domains because their ability to control and select memories or stimulus features is impaired.

It is clear that prefrontal areas are implicated in retrieval and interference control processes and patients with frontal lobe damage are impaired on tasks that recruit these processes leading to difficulties in initiating and maintaining preliminary retrieval processes to recall specific characteristics of the study episode. When a complete set of features has been matched and a candidate response has been generated, a second evaluation stage is necessary to test the validity of the retrieved representation to ensure it pertains to the actual memory trace as opposed to an inaccurate construction of some other internally generated or misleading information. These processes of verification have been described as criterion setting, in which an individual must evaluate the perceptual and semantic details that serve as a criterion match for the retrieved response (Schacter *et al.*, 1998), and source monitoring, which determines the origin of a memory trace in terms of how and when it was acquired (Johnson *et al.*, 1993; Schacter *et al.*, 1998). Event-related functional MRI procedures have been informative with respect to brain regions involved in the evaluation of retrieval output. Schacter *et al.* (1997) found anterior prefrontal activations during post-retrieval monitoring. Furthermore, Parkin *et al.*'s (1996) frontal patient often reported confident remember judgements when falsely recalling information, suggesting that the anterior communicating artery aneurysm and related frontal atrophy may have impaired post-retrieval evaluation processes leading the patient to falsely conclude, based on more general characteristics of the study episode, that the product of their retrieval was correct.

Recently, Jacoby *et al.* (2005) have examined the efficacy of a dual-process model for the interpretation of misinformation effects in younger and older adults (Jacoby, 1999). The dual-process model draws a distinction between recollection (consciously controlled search processes) and accessibility bias (reflecting automatic forms of memory), which are assumed to be independent of each other. Accessibility bias is conceived as an alternative basis for responding when recollection fails. Although accessibility bias is similar to the familiarity process of some models (Mandler, 1989; Gardiner and Java, 1993), we avoid the term 'familiar' because it is typically used with regard to recognition memory rather than cued recall. Additionally, 'familiar' sometimes refers to a subjective report, whereas accessibility bias specifically refers to an underlying process.

It was predicted that increased interference from misinformation can result from an age-related deficit in the ability to recollect without any change in accessibility bias. Conversely, increased interference can also result from an increase in the accessibility of interfering information without any change in recollection. A cued-recall task was employed to test this model. Subjects were presented with pairs of related words (knee-bone) during an initial study phase. After this initial study phase, subjects alternated between studying briefly presented words (e.g. bone), for a later memory test,

and recalling word pairs from the initial word-pair learning. The critical manipulation occurred when subjects studied briefly-presented words prior to cued recall of the initial word pairs. In fact, there was no later memory test for these briefly presented words. These words were intended to facilitate cued recall (e.g. bone; knee b_n_) or interfere with cued recall (e.g. bend; knee b_n_), with the latter being an example of the provision of misleading information before recall. To compare effects of the primes, a third baseline condition was included in which cued recall was preceded by a ampersand symbols (e.g. &&&&; knee b_n_).

For young adults, the findings were straightforward: when recollection failed, young adults generated the most accessible response in a cued-recall task and accessibility was influenced by a prime. The recollection/accessibility model predicted that a valid prime would increase correct recall to the same extent that an invalid prime would decrease it, and that is what happened with young adults. In contrast, for older adults, the recollection/accessibility bias model was insufficient to account for the data. Older adults were disproportionately affected by the invalid prime such that misinformation reduced performance to a greater extent than correct information facilitated performance. An additional parameter, described as capture, expanded the model to account for these findings. Capture, as described in Jacoby's Capture Model (Jacoby *et al.*, 2005), is a consequence of an interference control (West, 1996) or inhibitory control deficit (Hasher and Zacks, 1988; West, 1996) or, alternatively, the result of goal neglect (Duncan, 1995).

The capture framework encompasses an early-selection stage and a later-evaluation stage (Jacoby, 1999; Jacoby *et al.*, 2005). The early-selection stage is similar to other models that describe preliminary retrieval processes in which a refined search of the characteristics of a retrieval episode is initiated with a high degree of specificity, described as focusing (Norman and Schacter, 1996; Schacter *et al.*, 1998) or as cue-specification, which serves to constrain the responses that come to mind (Burgess and Shallice, 1996). We argue that capture by an invalid prime occurs when early selection becomes unconstrained due to poor focusing of retrieval or poor cue-specificity. Reliance upon an unconstrained retrieval focus or more general cue information will activate both the sought-after target word as well as the associated invalid prime, increasing the likelihood of false recall.

Older adults were more prone to false recall because highly accessible misleading information in the form of an invalid prime captured their response due to impaired inhibitory control or goal neglect. Moreover, older adults showed a greater propensity to accept their incorrect responses as a valid product of remembering in contrast to younger adults, who rarely claimed to remember an incorrect response. Jacoby and co-workers argued that there are two means by which false remembering arises. One route involves poor evaluation of a candidate response. The capture framework's later-evaluation stage is in accord with signal detection theory accounts of remember/know judgements (Tulving, 1985; Donaldson, 1996), which propose that once a potential response is generated it is only accepted as a 'remember' response if it passes a diagnostic threshold of accessibility. Poorer post-retrieval evaluation and more lenient source monitoring give rise to a lower threshold of acceptance for accessible information and thus greater false remembering. Factors such as source confusion (Lindsay and Johnson, 1989) and impaired post-retrieval evaluation of contextual details (Schacter *et al.*, 1997) have been identified in older adults, implying that age-differences in evaluation processes could underscore greater false remembering in the elderly.

The second route to false remembering, proposed by Jacoby and co-workers, was only apparent in older adults—when older adults were captured by an invalid prime this was often accompanied by a remember response. Disproportional remembering under conditions of invalid priming suggests that capture may be a more important route to false remembering than deficient evaluation of potential responses after they have been brought to mind. Jacoby and co-workers argue that false remembering via the capture route occurs early, before an opportunity to engage in recollection and response evaluation. Being captured and falsely accepting an invalid prime is likened to the Stroop effect, whereby falsely reporting the colour of the word forgoes the opportunity to read the word. The association between age and capture may leave older adults more susceptible to scams because they are less able to constrain responses that come to mind, thus allowing a deceitful person to provide false information about past agreements that an older adult may inadvertently accept.

The goal of the present experiment is to determine whether Jacoby *et al.*'s framework combining recollection, accessibility bias and capture can inform us as to whether TBI patients are also vulnerable to misinformation effects in a way similar to older adults. In addition to recording subject's false recall and subjective experience of their reports we measured electrodermal activity (EDA) following the presentation of an invalid cue. Skin conductance responses (SCRs) were extracted over an epoch of 5 s after a misleading cue to index the physiological reactivity during this period. Several studies have shown that cortical damage, particularly to right frontal areas, attenuates SCRs selectively to psychologically significant stimuli but not to simple tones (Morrow *et al.*, 1981; Zoccolotti *et al.*, 1982; Zahn *et al.*, 1999). These findings suggest a specific impairment of cognitive or emotional processing leaving the orienting response to physical stimuli (e.g. a startling noise) intact. Lehrer *et al.* (1989) reported that brain-injured patients who showed reduced SCRs on a range of cognitive tasks showed less situation-appropriate modulation of physiological activity than controls. Relatively larger SCRs in the control group were interpreted as adaptive responses to facilitate task processing.

In the current experiment enhanced SCRs may reflect the subjective significance of the false memory. That is, the SCR may index the subjective significance of a retrieval error that in turn informs subjective judgements regarding the certainty of a response. Previous work in the context of

sustained attention has demonstrated that TBI patients (O'Keeffe *et al.*, 2004) and children with ADHD (O'Connell *et al.*, 2004) show diminished SCRs during error processing indicating the impaired appraisal of an error, even to errors of which they were aware. One possibility in the current study is that healthy controls will utilize their SCRs to false memories to judge the accuracy of their performance. Reduced confidence in their false memory will be associated with increased SCRs. In contrast, TBI patients may be less able to utilize SCRs as an emotional or cognitive marker to mediate decisions regarding the accuracy of their response and SCRs will not vary as a function of subjective report. A second possibility is that SCRs will vary as a function of the level of resources assigned to post-retrieval evaluation. That is, SCRs may reflect the product of an efficient retrieval evaluation. Under these circumstances we would expect group differences in SCR amplitude with diminished responses in the TBI group irrespective of subjective judgement.

In the present experiment we anticipate that TBI patients will be vulnerable to capture by misleading information if they are unable to deploy the necessary executive skills to reject this information. Moreover, we predict that post-retrieval verification processes will also be compromised increasing the likelihood that TBI patients will falsely claim to remember incorrect responses. Impaired error processing abilities as indexed by EDA may also negatively contribute to poor cognitive evaluation of response accuracy. The present experiment examines the utility of the capture framework for the purpose of identifying brain-injured patients who may be vulnerable to deception from misinformation, undue persuasion or other bias influences. In parallel with the ongoing work with the elderly (Jacoby *et al.*, 1996; Jacoby, 1999) this work aims to better specify interference effects in frontally impaired groups that will ultimately lead to the development of training procedures to ameliorate executive dysfunction in these groups.

In addition to the misinformation task, subjects completed a battery of commonly used cognitive tests to measure free recall, recognition and executive function. An exploratory analysis was conducted to examine, with greater specificity, the candidate executive functions that may be associated with memory capture. Performance on tasks that measured attention, goal monitoring, strategy application and verbal fluency was examined in relation to recollection in the presence of misleading information. Questions were focused on specific executive components and their relationship with patient's susceptibility to memory capture. For instance, is susceptibility to misleading information associated with neglect of our intended goals? Are those who are vulnerable to capture during cued recall poorer at adhering to a strategy, as measured by the strategy application test, or less able to generate alternatives under conditions of free recall during verbal fluency? A correlation matrix of the aforementioned variables provides a useful way to fractionate intact processes from dysfunctional executive components that may underlie capture.

Method

Participants

A total of 18 TBI patients and 18 neurologically healthy controls participated in this experiment. The TBI patients were recruited from the National Rehabilitation Hospital, Dún Laoghaire. Clinical data for the TBI patients are presented in Table 1 and include the following: cause of brain injury, post traumatic amnesia (PTA) severity,

Table 1 Clinical data for the TBI patients

TBI patient	TBI cause	TBI severity (PTA)	TBI severity (GCS)	Months since TBI	Location of damage
AMB	Fall	Extremely severe	Moderate	31	Right subdural haemotoma, right frontal (CT)
BE	Assault	Very severe	Severe	11	Left temporal region, cerebellar haemorrhage (CT)
CMCG	RTA—motorbike	Extremely severe	Severe	38	Left frontoparietal (CT)
DD	RTA—car	Very severe	Moderate	34	Normal CT scan
DF	RTA—car	Extremely severe	N/A	N/A	N/A
ER	Fall	Very severe	Severe	21	Right frontal, SAH (CT)
FL	RTA—car	Extremely severe	Severe	38	SAH, left frontal, left superior temporal, left occipital (CT)
GB	RTA—car	Mild	N/A	84	DAI (MRI)
GC	Fall	Very severe	Severe	44	Right frontal ICH (CT)
GL	Fall	Severe	N/A	39	Right frontal (MRI)
JC	RTA—motorbike	Very severe	Severe	25	Left fronto-temporal haemotoma (CT)
JG	N/A	Severe	N/A	N/A	N/A
KM	Assault	Very severe	Moderate	13	Right fronto-parietal (CT)
LM	Assault	Severe	Severe	76	Right fronto-parietal, temporal (CT)
MOM	Assault	Very severe	Mild	19	N/A
OW	RTA—motorbike	Extremely severe	Severe	23	Frontal bilaterally, basal ganglia, right temporal (CT)
RB	RTA—car	Extremely severe	Severe	9	Right frontal, left occipital, DAI (CT)
RC	RTA—pedestrian	Extremely severe	Severe	29	SAH, left occipital (CT)

PTA, post-traumatic amnesia (mild, <1 h; moderate, 1–24 h; severe, 1–7 days, very severe, 7–28 days; extremely severe, >28 days); GCS, glasgow coma scale (mild, >13; moderate, 9–12; severe, <8); RTA, road traffic accident; DAI, diffuse axonal injury; SAH, subarachnoid haemorrhage; ICH, intracerebral haemorrhage; CT; computerized tomography.

Table 2 Participant characteristics

Participant characteristics	TBIs (n = 18)	Controls (n = 18)
Gender		
M	15	15
F	3	3
Age (years)		
Mean	27.44	24.89
Standard deviation	(9.51)	(6.75)
Level of education		
Primary	2	2
Secondary	8	8
Tertiary	8	8

Glasgow coma scale (GCS) severity, and the number of months since injury and location of brain damage. To summarize, the cause of injury fell into one of the three categories—road traffic accident, assault or fall. The PTA severity for one patient was 'mild', for three patients 'severe', for seven patients 'very severe' and a further seven were classed as 'extremely severe'. Records from the GCS were available from 14 of the 18 TBI patients. Out of them one patient was registered as 'mild', three patients were 'moderate' and 10 patients were classed as 'severe'. The average number of months since injury was 33.38 (SD = 21.04). Of the 18 patients, 11 patients sustained frontal damage as part of their injury (7 right frontal, 3 left frontal and 1 bilateral frontal). Patients also exhibited damage in parietal, temporal and occipital areas, and sub-cortical damage to the basal ganglia and the cerebellum. Location of damage was determined by radiologist assessment of MRI/CT records. The full details are presented in Table 1.

The TBI patients were screened for major psychiatric disorders, drug or alcohol dependency, a pretrauma history of epilepsy and any other neurological disorder. The control participants fulfilled the latter requirements and additionally had never suffered from loss of consciousness from a head-injury. The Department of Psychology, Trinity College, Dublin and the National Rehabilitation Hospital, Dún Laoghaire, Dublin granted ethical approval for the study. All participants signed an informed consent form according to the Declaration of Helsinki before taking part in the study. The groups were matched according to gender, age and level of education, and these participant characteristics are presented in Table 2.

Design and materials

Misinformation paradigm

The experiment was designed as a two-way mixed factorial, with Group (TBIs, Controls) as the between-subjects factor and Prime Type (valid, invalid and baseline) as the within-subjects factor. Stimuli consisted of word-sets that comprised one cue word (e.g. knee) and two word-associates that are described as response words (e.g. bone and bend). The study list contained 60 critical word pairs (the cue word paired with one of the two possible response words) and 9 buffer word pairs, 6 of which were presented at the beginning of the list and 3 at the end to prevent primacy and recency effects.

The word-sets were divided into three groups of 20 critical words. All stimuli were presented in a previous experiment (Jacoby *et al.*, 2005) and were selected from a pool of norms (Jacoby *et al.*, 1996). The two response words in each word-set were equal in the number of letters and each could be used to complete the same word fragment (e.g. b_n_). Cue words and response words were matched for word frequency (Thorndike and Lorge, 1944) and word length. Response words were also matched in terms of their probability of completing the fragment, and the number of fragments with a missing first letter was matched across the three word-set groups. Word-set groups were rotated across subjects for the valid-prime, invalid-prime and baseline conditions. Each response word was presented as the target response or the alternate response equally often. This produced six formats (3 test conditions × 2 response groups). The test list included the 60 critical cue words (20 valid-primes, 20 invalid-primes and 20 baselines) followed by a word fragment of the response words.

Neuropsychological tests

All participants completed a neuropsychological battery that was designed to measure attention, memory and planning/strategy performance. Two subtests from the Test of Everyday Attention (TEA) (Robertson *et al.*, 1996) were administered to participants: the 'Telephone Search' and the 'Telephone Search while Counting' subtests. Memory performance was assessed with 'Logical Memory I and II' from the Wechsler Memory III (Wechsler and Wycherly, 1998). Verbal fluency was measured using the FAS procedure (Spreen and Strauss, 1998), in which subjects are given 1 min to generate as many words as they can that begin with the letter F. The same task is repeated for the letters A and S. The total number of words generated provides a measure of verbal fluency that reflects the participants' ability to organize and search for information in semantic memory. Prospective memory function was measured using an event-based task (Walker, 2003). Participants carried out a sentence verification task in which they indicated whether a sentence was true ('bishops drink tea') or false ('dogs have wheels'). Embedded within six sentences was a target word ('hand') that served as a prospective cue for subjects to press the enter key. Number of 'hits' was taken as a measure of prospective memory performance. To measure planning/strategy performance the revised strategy application test (R-SAT) (Levine *et al.*, 2000) was administered. The task presents an unstructured environment in a laboratory setting whereby the most efficient strategy can be broken by salient external cues and internal habits. The optimal strategy involves completing the briefest items in three separate activities: figure tracing, sentence copying and object numbering. The primary score reflecting strategy application is the proportion of brief items from all the three tasks that are completed.

Subjective-report measures

In order to measure cognitive and emotional functioning that pertains to everyday events, the Hospital Anxiety Depression (HAD) scale (Zigmond and Snaith, 1983) and the Cognitive Failures Questionnaires (CFQ-self, CFQ-other) (Wallace *et al.*, 2002) were administered to all participants. The HAD scale comprises 14 items, 7 that reflect anxiety levels and 7 that reflect depression levels. The CFQ records reported slips of action and memory in everyday life from the perspective of the participant (CFQ-self) and a significant other (CFQ-other).

EDA

EDA was measured using the Biopac Student Lab system (Version 3.6.6.1; Biopac Systems). EDA was continuously recorded from the distal phalanges of the index and middle fingers of the non-dominant hand. Two Ag/AgCl electrodes were mounted in individual housings and shielded to minimize noise interference. They were attached

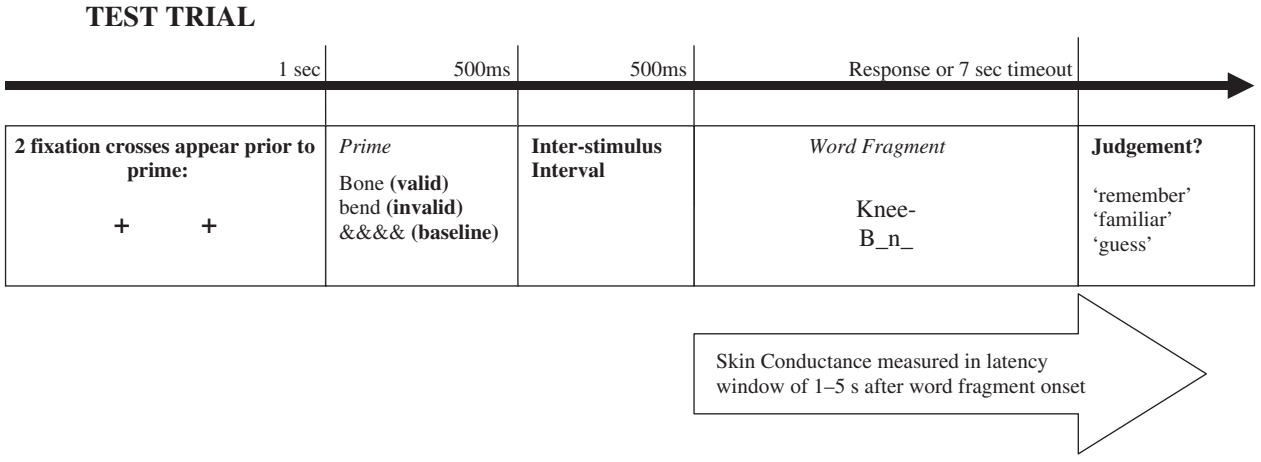

Fig. 1 Schematic diagram representing a single test trial during the cued-recall procedure.

to the distal phalanges by a Velcro strap. Each electrode was 16 × 17 × 8 mm. The electrodes used had a 6-mm contact area with a 1.6 mm cavity for accommodating the electrode gel. Multipurpose gel was used as the electrolyte. The incoming skin response signals were converted to digital signals via an MP30 data acquisition unit and processed with BIOPAC Student Lab PRO for an offline analysis.

For each of the three cued-recall conditions (valid, invalid and baseline) a latency window of 1–5 s after stimulus onset was specified for elicited SCR. Measurement of the amplitude of SCR was taken as the conductance at the peak minus the conductance prior to the response, as defined by Stern *et al.* (2001). The amplitude of the largest SCR that had an onset within this latency window was measured. The criterion for the smallest scorable SCR was set at 0.02 μS.

Procedure

All participants were assessed in two testing sessions. In the first session the neuropsychological tests and subjective-report questionnaires were completed, and in the second session the misinformation paradigm and EDA acquisition was undertaken. The misinformation task was administered as follows:

Phase 1: initial study phase

Subjects were instructed to learn the word pairs presented serially, one pair at a time. They were informed that the words in each pair formed an association related in meaning (e.g. stanza-poem), and their task was to read the word pairs silently and to remember them for a later memory test. Subjects were recommended to use the time during learning to think about the association between the words in each pair. Each study pair was presented for 3 s with a 500 ms inter-stimulus-interval.

Phase 2: cued-recall fragment completion

Subjects were told that they were required to do two tasks, one after the other: alternate between studying briefly presented words for a later memory test and recalling word pairs from the initial word-pair learning in Phase 1. Instructions specified that each trial would begin with the brief presentation of a single word that was to-be-remembered for later. After the offset of the single word, a word pair from Phase 1 was presented with letters missing from the second word of the pair creating a 'word fragment' (e.g. stanza-p_e_). Subjects were asked to use the first word of the pair and the second fragment to help recall the prior presentation of the pair studied in Phase 1. They were told that if they were unable to recall the prior word pairing they should complete the fragment with their best guess.

It was made clear to the subjects that sometimes the briefly presented single word (that is to-be-remembered) would be the same as the word that completes the subsequent fragment (e.g. 'poem' followed by 'stanza-p_e_'), and other times the word would be different (e.g. 'poet' followed by 'stanza-p_e_'). Additionally, subjects were told that sometimes a string of symbols (&&&&&) would be presented instead of a single word. They were informed that these trials would serve as a baseline measure for memory performance and did not have to be remembered for later.

Each test trial began with a 1 s presentation of two fixation crosses to mark the location in which the prime was to appear. The subsequent prime was presented for 500 ms followed by a blank screen interval of 500 ms.

Phase 3: subjective reports

Finally, after fragment completion subjects were asked to judge the accuracy of their recall by stating 'remember', 'familiar' or 'guess'. If subjects were certain they could recall specific details from the word-pair learning in Phase 1 they were instructed to respond 'remember'. If the response was based on familiarity without the supporting details the subjects were to report 'familiar'. If the subjects were purely guessing and had no idea what the early word pair was they were to respond by saying 'guess'. Subjects were given 15 s after the offset of the word fragment for cued recall and their subjective report. Figure 1 presents a schematic diagram summarizing the experimental procedure.

Subjects were given a short practice before beginning the actual test session using six word pairs that were later used as the buffers at test. Subjects were also asked to explain the instructions to the experimenter in their own words to ensure their full comprehension.

Results

Subjective everyday cognitive and emotional functioning

TBI patients reported a greater frequency of everyday cognitive failures as measured on the CFQ-for-self, compared with controls, $t(34) = 2.32$, $P = 0.027$. Reports from the patients'

relatives on the CFQ-for-other also confirmed that TBI patients were more prone to cognitive failures than the control subjects, $t(32) = 2.63$, $P = 0.013$ (CFQ-for-other scores were unavailable for two control subjects). Emotional functioning as measured by the HAD revealed significant differences, with patients reporting higher levels of anxiety [$t(34) = 3.04$, $P = 0.005$] and depression [$t(34) = 4.66$, $P = 0.0001$] compared with control subjects. Mean scores and standard deviations are presented in Table 3. In subsequent factorial analyses of variance examining false recall, false remembering and skin conductance, the HAD-anxiety and HAD-depression scores were included as covariates to control for any influence of impaired emotional functioning in the TBI group.

Objective cognitive measures

Subtests from the WMS-III showed differences in logical memory performance, with TBI patients remembering significantly less story information upon immediate recall, $t(34) = 3.32$, $P = 0.002$, and after a 30 min delay, $t(34) = 2.66$, $P = 0.012$. TBI patients showed a trend towards poorer story recognition performance compared with controls but this did not reach a significance level, $t(34) = 1.96$, $P = 0.065$. Subtests from the TEA, the telephone search and telephone search while counting (as measured by the dual task decrement score) did not differentiate between TBI patients and controls, $t(34) = 1.70$, $P = 0.10$. On the revised strategy application task (R-SAT) TBI patients completed significantly fewer brief items than controls, $t(34) = 3.75$, $P = 0.001$, indicating poor adherence to an effective strategy in the TBI group. Event-based prospective remembering was also poorer in the TBI patients, who were less likely to execute a delayed intention in response to a prospective cue compared with controls, $t(34) = 4.19$, $P = 0.0001$. Verbal fluency was reduced in the TBI patient sample compared with controls, $t(34) = 4.25$, $P = 0.0001$, indicating poor retrieval strategies for semantic information. Table 3 also presents the means and standard deviations for these test scores. Finally, for 7 of the 18 patients, there was no evidence of frontal damage (*see* Table 1) so we compared neuropsychological performance on each of the above measures between the non-frontal patients ($n = 7$) and those patients who had sustained direct frontal damage as part of their injury ($n = 11$). The only measure to discriminate performance between these subgroups was the R-SAT. The frontal subgroup exhibited a significantly poorer R-SAT score, as determined by fewer brief items, than the non-frontal subgroup, $t(16) = 3.14$, $P = 0.006$.

Table 3 Tests of difference for questionnaires and neuropsychological tests

	TBIs	Controls	*P*-value
Questionnaires			
Hospital anxiety depression scale (HAD)			
HAD anxiety			
Mean	8.94	5.39	0.005**
SD	(3.77)	(3.24)	
HAD depression			
Mean	7.72	2.06	0.0001**
SD	(4.80)	(1.89)	
CFQ			
CFQ-self			
Mean	43.22	29.78	0.027*
SD	(21.72)	(11.56)	
CFQ-other			
Mean	14.72	8.44	0.013*
SD	(8.55)	(4.52)	
Neuropsychological tests			
Test of everyday attention (dual task decrement score)			
Mean	2.35	1.09	0.101
SD	(2.75)	(1.46)	
Logical memory I (immediate recall)			
Mean	43.94	54.39	0.002**
SD	10.11	8.73	
Logical memory II (delayed recall)			
Mean	25.83	33.50	0.012*
SD	10.22	6.72	
Logical memory II (recognition)			
Mean	24.44	27.72	0.065
SD	6.91	1.57	
Verbal fluency (FAS)			
Mean	14.33	20.06	0.0001**
SD	3.36	4.56	
Prospective memory score			
Mean	3.00	5.67	0.0001**
SD	2.59	0.76	
Revised strategy application task (R-SAT)			
Mean	0.69	0.88	0.001**
SD	0.14	0.14	

*significant at the 0.05 level.
**significant at the 0.01 level.

Cue-recall performance

The overall probability of correct recall was examined as a function of prime (valid, baseline and invalid) and group (TBIs, controls). (All reported ANOVAs are adjusted for the covariates, HAD-anxiety and HAD-depression.) A 2 × 3 mixed-factorial ANOVA showed no reliable effect of group, $F(1,34) = 1.64$, $P = 0.21$. A main effect of prime was significant, $F(2,68) = 17.09$, $P = 0.0001$, such that valid primes facilitated recall and invalid primes reduced recall relative to the baseline primes. Moreover, a group × prime interaction was observed, $F(2,68) = 4.54$, $P = 0.014$, and simple effects showed that there was a greater reduction in the probability of correct recall produced by an invalid prime in TBI patients than controls ($P = 0.008$) (This and all subsequent paired comparisons have been Bonferonni corrected), suggesting that TBI patients are more prone to false recall because they have been captured by the invalid primes (*see* Fig. 2). There was no difference in cued recall between TBI patients and controls under valid-prime conditions ($P = 0.19$), suggesting that valid primes did not benefit either group more than the other. The invalid primes reduced recall relative to the valid primes (all $P < 0.01$) and baseline primes (all $P < 0.05$) for both TBI patients and controls.

In a subsidiary analysis, cue-recall performance was examined with respect to the TBI subgroups based on region

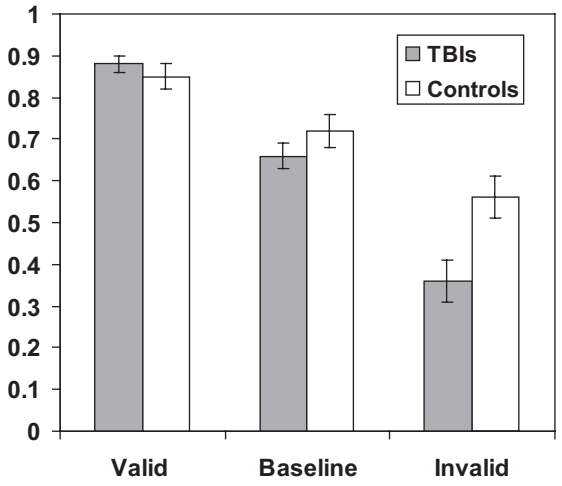

Fig. 2 Overall performance for correct recall in TBI patients and controls as a function of prime-type. The error bars represent the standard error of the mean.

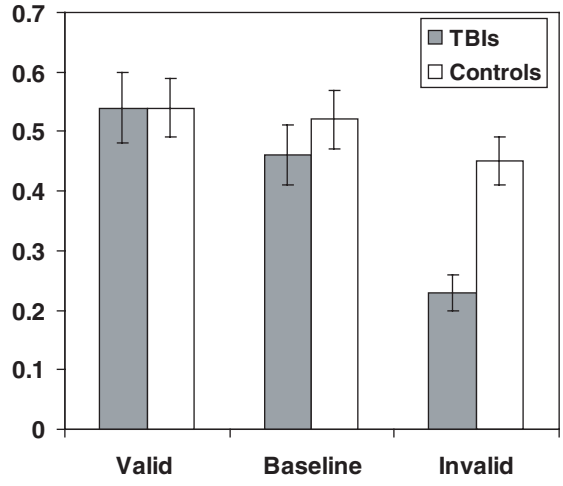

Fig. 3 Probability of a 'remember' response for correct recall in TBI patients and controls as a function of prime-type. The error bars represent the standard error of the mean.

of injury. A 3 × 3 mixed-factorial ANOVA was conducted with prime as the within-subjects factor and group (frontal, non-frontal and control) as the between-subjects factor. A reliable main effect of prime, $F(2,68) = 17.09$, $P = 0.0001$, revealed that valid primes improved recall and invalid primes reduced recall relative to the baseline primes. There was no main effect of group, $F(2,31) = 2.04$, $P = 0.14$, and no group × prime interaction, $F(4,62) = 2.34$, $P = 0.07$.

Subjective reports

In a separate analysis, the probability of a remember response was conditionalized on correct recall. A 2 × 3 mixed-factorial ANOVA showed no effect of group, $F < 1$. A main effect of prime, $F(2,68) = 9.30$, $P = 0.0001$, revealed that remember responses were significantly reduced by the invalid prime condition compared with valid and baseline prime conditions. Furthermore, a group × prime interaction, $F(2,68) = 5.86$, $P = 0.005$, was driven by reduced remember responses for TBI patients when exposed to invalid primes compared with controls ($P = 0.001$). There were no group differences in the valid condition ($P = 0.10$). Within-subject comparisons showed that TBI patients also showed a reduced probability of remember responses when invalidly primed than when exposed to valid or baseline primes (all $P < 0.0001$). For control subjects, the probability of a remember response was not significantly affected by the presentation of a prime (all $P > 0.1$). These results suggest that invalid information can disproportionately reduce the probability of a remember response for TBI patients compared with controls (*see* Fig. 3).

Remember responses were also analysed in accordance with TBI subgroups (frontal and non-frontal). Again a 3 × 3 mixed-factorial ANOVA was conducted with prime and group (frontal, non-frontal and control) as factors. A main effect of prime, $F(2,62) = 9.78$, $P = 0.0001$, showed that remember responses were reduced by the invalid prime condition compared with valid and baseline prime conditions. No main effect of group was apparent, $F < 1$. However, a significant group × prime interaction, $F(4,62) = 2.94$, $P = 0.027$, was driven by reduced remember responses for the frontal subgroup compared with the controls ($P = 0.017$) but there were no differences between non-frontal subgroup compared with controls ($P = 0.229$) or between each of the TBI subgroups ($P = 1.000$). There were no remember-response differences between groups in the valid prime condition (all $P > 0.1$). Both subgroups showed a reduced probability of remember responses when invalidly primed than when exposed to valid or baseline primes (all $P < 0.05$).

Remember responses were also conditionalized on false recall; thus, we examined the probability of false remembering. A 2 × 3 mixed-factorial ANOVA showed a significant effect of group, $F(1,34) = 4.17$, $P = 0.049$, with TBI patients showing a greater probability of false remembering compared with controls (0.094 versus 0.056). A main effect of prime, $F(2,68) = 10.83$, $P = 0.0001$, was indicative of increased false remembering when subjects were invalidly primed compared with conditions of valid or baseline priming. A significant group × prime interaction, $F(2,68) = 5.59$, $P = 0.006$, was driven by increased false remembering in TBI patients compared with controls when invalidly primed ($P = 0.020$). Under valid prime conditions there was no difference between the groups ($P = 0.503$). TBI patients showed significantly more false remembering when invalidly primed than when validly primed ($P = 0.001$) or when exposed to a baseline prime ($P = 0.033$), suggesting that capture by an invalid prime leads to falsely subjective experience of remembering. No simple effects reached the significance level for the control group (all $P > 0.1$) (*see* Fig. 4).

To examine potential differences across TBI subgroup a further 3 × 3 mixed-factorial ANOVA was undertaken. A main effect of prime, $F(2,62) = 11.34$, $P = 0.0001$, showed

the expected pattern—remember responses were less in the invalid prime condition compared with valid and baseline prime conditions. There was no main effect of group, $F(2,31) = 2.66$, $P = 0.086$. A significant group × prime interaction, $F(4,62) = 2.82$, $P = 0.033$, was also apparent. Paired comparisons showed that only the frontal subgroup showed significantly more false remembering when invalidly primed compared with controls ($P = 0.048$). False remembering in the invalid condition between the non-frontal subgroup and controls did not reliably differ ($P = 0.470$). No differences were apparent between the two TBI subgroups in the invalid condition ($P = 1.000$). Furthermore, the valid prime conditions did not discriminate remember response across the three groups (all $P > 0.1$). Within-subject comparison show that both the TBI subgroups showed significantly more false remembering when invalidly primed than when validly primed (all $P < 0.05$). However, although the frontal subgroup exhibited increased false remembering when invalidly primed than when exposed to baseline primes, ($P = 0.002$) this difference was short of significance in the non-frontal subgroup ($P = 0.055$).

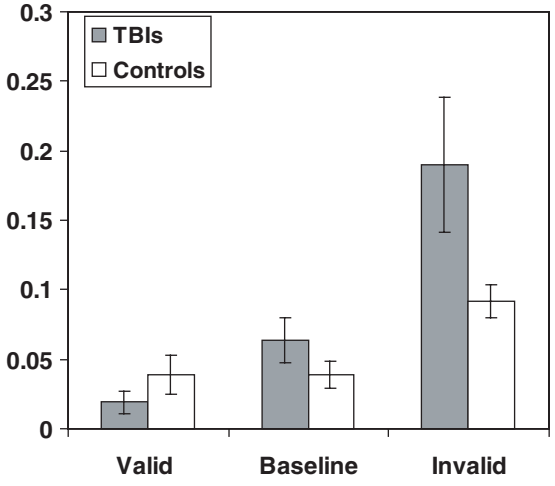

Fig. 4 Probability of a 'remember' response for false recall in TBI patients and controls as a function of prime-type. The error bars represent the standard error of the mean.

Electrodermal data

SCRs were first examined as a function of group and prime for false recall responses. Two TBI patients were EDA stabiles (non-responders) and were excluded for all SCR analyses. In the current analysis, a proportion of subjects under valid prime conditions did not falsely recall (4 controls and 2 TBI patients). Consequently, the group × prime analysis was restricted to a reduced set of subjects (14 controls and 14 TBI patients). A 2 × 3 mixed-factorial ANOVA showed a main effect of prime, $F(2,52) = 8.56$, $P = 0.001$. Repeated contrasts revealed that SCRs to false responses in the invalid condition were significantly greater in amplitude than SCRs in the valid ($P = 0.002$) or baseline ($P = 0.001$) conditions, irrespective of the group. There was no main effect of group ($F < 1$) or group × prime interaction ($F < 1$) (*see* Fig. 5A).

TBI patients' and controls' subjective judgements of their false recall were examined with respect to their EDA. Analysis of EDA was restricted to invalid prime trials only [There was an insufficient number of valid and baseline trials as a function of judgement (remember, familiar, guess) to warrant the analysis]. A group × judgement (remember, familiar, guess) factorial analysis was conducted for SCRs during false recall. There was no main effect of group ($F < 1$). A significant main effect of judgement [$F(2,64) = 3.58$, $P = 0.034$] and a reliable group × judgement interaction [$F(2,64) = 3.45$, $P = 0.038$] was observed. Within-subject contrasts for the control group revealed higher mean SCRs for familiar judgments

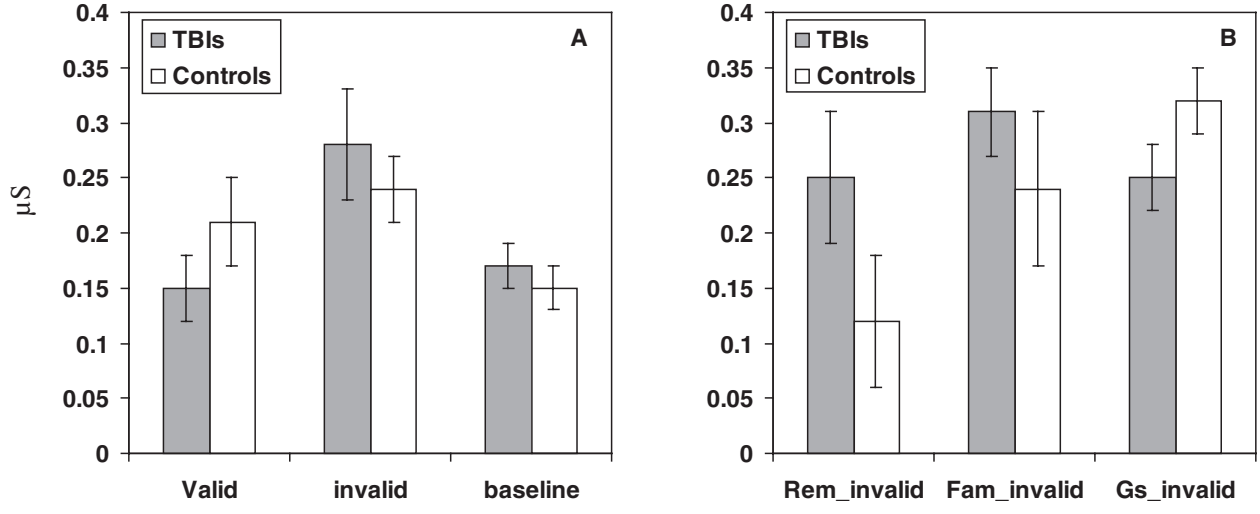

Fig. 5 (**A**) Skin conductance (μS) as a function of prime for false recall. TBI ($n = 14$) and control ($n = 14$). (**B**) Skin conductance (μS) as a function of judgement when invalidly primed for false recall. TBI ($n = 16$) and control ($n = 18$). The error bars represent the standard error of the mean.

Table 4 Control group: correlation matrices examining associations between recollective performance and executive task performance

	Correct remember responses—baseline	Correct remember responses—invalid prime	Logical memory I—immediate recall	Logical memory 2—delayed recall	Prospective memory score	Verbal fluency (FAS)	Strategy application task (R-SAT)
Correct remember responses—baseline							
Correct remember responses—invalid prime	0.396						
Logical memory I—immediate recall	0.151	0.203					
Logical memory 2—delayed recall	−0.001	0.209	0.879**				
Prospective memory score	−0.194	0.017	−0.304	−0.160			
Verbal fluency (FAS)	−0.247	0.137	0.008	−0.005	−0.013		
Strategy application task (R-SAT)	−0.091	0.068	0.319	0.201	0.003	0.233	

NB: Uncorrected for multiple comparisons. **Correlation is significant at the 0.01 level (two-tailed).

Table 5 TBI patients: correlation matrices examining associations between recollective performance and executive task performance

	Correct remember responses—baseline	Correct remember responses—invalid prime	Logical memory I—immediate recall	Logical memory 2—delayed recall	Prospective memory score	Verbal fluency (FAS)	Strategy application task (R-SAT)
Correct remember responses—baseline							
Correct remember responses—invalid prime	0.335						
Logical memory I—immediate recall	0.492*	0.361					
Logical memory 2—delayed recall	0.458	0.508*	0.637**				
Prospective memory score	0.106	0.487*	0.112	0.107			
Verbal fluency (FAS)	0.289	0.120	0.149	−0.017	0.568*		
Strategy application task (R-SAT)	−0.007	0.075	0.228	0.198	0.586*	0.311	

NB: Uncorrected for multiple comparisons. **Correlation is significant at the 0.01 level (two-tailed). *Correlation is significant at the 0.05 level (two-tailed).

than remember judgements ($P = 0.014$) and higher SCRs for guess judgements compared with remember judgements ($P = 0.001$). No within-subjects comparisons reached significance in the TBI group (all $P > 0.1$) (*see* Fig. 5B). Between-subject comparisons revealed lower mean amplitude SCRs for controls compared with TBI patients ($P = 0.019$) when they falsely remembered a word during invalid prime trials. No reliable group differences in mean SCR were observed during familiar and guess judgements (all $P > 0.1$).

Correlation coefficients were calculated in order to assess whether the level of uncertainty of false recall, measured by the frequency of guess responses, was associated with enhanced SCR amplitudes in controls subjects and TBI patients. A higher frequency of guess reports was associated with larger SCRs in the control group ($r = 0.500$, $P = 0.035$). This relationship was absent in the TBI group ($r = -0.056$, $P = 0.837$).

Relationships between correct remembering during invalid cueing and other cognitive measures

The specific cognitive measures that dissociated performance in the control and TBI groups (*see* Table 3) were included in two correlation matrices, one for each group, to assess relationships between correct remembering under conditions of invalid cueing and under baseline conditions, logical memory I and II, prospective memory performance, verbal fluency and strategy application. In the control group, one significant positive correlation was observed between logical memory I and II. No other measures were significantly associated (*see* Table 4). In the TBI group, correct remembering when invalidly cued showed a significant positive association with logical memory II and with the prospective memory score. Furthermore, prospective memory performance also correlated positively with verbal fluency and the strategy application measure (*see* Table 5).

Discussion

Traumatically brain injured patients were more likely to incorrectly recall misleading information in the form of an invalid prime than the healthy control subjects. Recall performance in the baseline condition was equated for TBI patients and controls discounting the possibility that greater susceptibility to misleading information in the TBI group was the result of differences in original learning. TBI patients who recalled the invalid prime were more likely to report the subjective experience of 'remembering' the invalid prime than

the control subjects. These findings, in a TBI sample, are consistent with the pattern of results reported by Jacoby and co-workers in older adult samples (Jacoby, 1999; Jacoby *et al.*, 2005). Furthermore, in TBI patients, reduced recollection in the context of background misinformation was associated with poor prospective remembering when engaged in a background task. In both instances we argue that failure to inhibit background information may prevent the further engagement of retrieval processes. Finally, subjective judgements of control subjects' false memories varied as a function of their SCR. Larger SCRs were associated with a higher frequency of guess reports in the control group suggesting that physiological reactivity may serve as a marker for response choice uncertainty. This relationship was absent in TBI patients.

The pattern of results in neurologically healthy controls is consistent with the recollection/accessibility bias model. During the early selection of response, upon the presentation of the cue, subjects must constrain the response that comes to mind by specifying cues that link retrieval processes directly to earlier studied words to enable recollection. If these processes fail then accessibility bias will influence response selection and accessibility is influenced by the prime. Valid primes facilitated recall to the same extent as invalid primes reduced recall, producing a symmetrical pattern around the baseline condition in the control group. In contrast, the recollection/accessibility bias model could not account for the pattern of results in the TBI patient group. For the patients the invalid prime reduced performance to a greater extent than the valid prime facilitated performance. This pattern of results around the baseline is supportive of Jacoby's capture framework. Capture by misinformation reflects a deficit in the early-selection of a response and occurs before any recollection attempts have been made.

Additionally, capture by the invalid prime was disproportionately associated with the subjective experience of 'remembering' in the TBI patients. This finding supports the conjecture that capture provides a route to false remembering that is separate from proposed differences in post-response evaluation abilities across patients and controls. We maintain that during early selection TBI patients are dependent on more unconstrained or general cues relating to earlier presented words which activate both the specific target responses and the more general invalid primes. Poor cue-specificity during early selection of a response will place greater demand on patients' abilities to disregard misinformation using executive control processes.

We hypothesized that prefrontal damage in brain-injured patients or diffuse axonal injury disrupting pathways to the prefrontal cortex would compromise executive control processes. It is important to note that not all TBI patients in this study directly sustained prefrontal damage but disruption to this region together with others is probable in the context of diffuse damage that characterizes brain injury. However, there was evidence that a subgroup of 11 patients with documented prefrontal damage (in contrast to a non-frontal subgroup of 7 patients) more strongly differentiated from controls in terms of reduced correct remembering and increased false remembering under invalid prime conditions. These findings strengthen our claim that that the performance differences exhibited are attributable to disrupted frontally-mediated processes.

The functional deficits that underlie capture are not well understood; impaired monitoring, goal neglect or inhibitory failure may increase vulnerability to capture by misinformation. To examine these possibilities, candidate executive measures were examined in relation to subjects' ability to recollect during interference. Specifically, we found that TBI patients with impaired prospective memory performance showed poorer recollection during interference. The prospective memory task in question required subjects to disengage from a background task (verifying whether sentences are true or false) in order to respond to prospective cues that were relevant to an earlier established intention. The cues were embedded within target sentences throughout the task. Successful performance depended not only on the prospective cue (the word 'hand') triggering the associated action representation (press 'enter') but also on subjects' ability to disengage from the background task. Previous work has demonstrated that the specificity and distinctiveness of the prospective cue influence the performance (McDaniel and Einstein, 1993; Einstein *et al.*, 1995) as does the level of engagement conferred by the background task (Einstein *et al.*, 1997).

Prospective memory performance did not correlate with other measures of episodic memory neither did it correlate with recollection ability in the baseline condition of the cue-recall task, but only with recollection under conditions of interference. What might prospective remembering and cued recall during interference have in common? Patients' inability to disengage from sentence verification may sacrifice their opportunity to monitor for a prospective cue. In a similar way, failure to gate processing of misleading information prevents engagement of an effective retrieval mode during cued recall. A common functional deficit that may underlie poor performance on both tasks is the inability to inhibit previously relevant but currently irrelevant information. Separately, prospective memory did correlate with two other tasks that also require interference control processes: verbal fluency, which involves maintenance of an effective retrieval strategy and suppression of earlier memory associations, and strategy application, which requires adherence to a plan and inhibition of salient external cues or internal habits. Recent evidence from changes in regional cerebral blood flow (Burgess *et al.*, 2003) suggest that prospective memory tasks recruit lateral prefrontal areas for maintaining internally generated goals but also involve the participation of medial prefrontal structures in order to withdraw attention or suppress processes of external (i.e. ongoing) stimuli. We propose that the latter function is also critical for avoiding interference from misleading information.

Evaluation of false recall was indexed by EDA. For both TBI patients and controls the invalid prime condition generated larger SCRs compared with the valid prime and baseline conditions. Control subjects exhibited larger SCRs when they experienced a false memory as 'familiar' or as the result of a 'guess' compared with when they claimed to 'remember' the false memory. Furthermore, control subjects with a higher frequency of guess responses to false memories also exhibited larger SCRs. In contrast, in the TBI sample, SCRs did not differ as a function of their subjective report and a relationship between frequency of guess response and SCR amplitude was not apparent in the TBI patient group. One possibility is that high SCRs in the control group may reflect strong activation of the invalid prime or conflict between the invalid response choice and the correct response that, in turn, engenders a stronger judgement of response uncertainly. Alternatively, high SCRs may reflect the product of an efficient retrieval evaluation. That is, the subjective experience of 'guessing' may reflect greater deployment of resources to test the validity of the false response. These two possibilities suggest that TBI patients are either unable to utilize SCRs as a physiological marker for retrieval evaluation to guide their subjective judgements or they do not allocate sufficient resources during post-retrieval evaluation. The insufficient resource argument is less plausible because TBI patients produced the same SCR amplitude to invalid trials as controls and further, they show larger SCRs when they falsely remember and do so more often than controls. The more likely explanation is that TBI patients fail to use SCRs as a marker to guide their post-retrieval evaluation or perhaps even misuse SCRs to conclude that misinformation in the form of an invalid prime is acceptable.

Models of EDA have proposed that SCR amplitude reflects activity of an appraisal system during goal-directed behaviour (Zimmer, 2000) or heightened processing of stimuli with cognitive or affective significance to healthy individuals (Zahn *et al.*, 1999). TBI patients and patients with focal frontal damage normally show attenuated SCRs across a range of tasks involving meaningful, significant or demanding stimulus processing (Zahn and Mirsky, 1999; Zahn *et al.*, 1999; O'Keeffe *et al.*, 2004). In contrast, the SCRs of TBI patients in the present study did not differ from controls during cued recall. Nevertheless, SCRs failed to discriminate between patients' subjective evaluation of their false memories in the same way as controls. Damasio's somatic marker hypothesis (Damasio, 1996) proposes that connectivity between ventromedial areas of the frontal lobes and the limbic system may be functionally important to allow the emotional evaluation of information in order to guide decision-making. In the context of TBI we cannot make strong inferences between lesion location and functional outcome but it is widely acknowledged that diffuse axonal injury can disrupt interconnections between the prefrontal cortex and subcortical areas. It is therefore possible that disruption to fronto-limbic pathways may impair subjective evaluation of an invalid response insofar as EDA no longer provides an index of uncertainty regarding its candidacy. The correction of an invalid response in frontal patients may therefore be less likely and the acceptance of misleading information more likely.

Cautious evaluation of potential responses after they have been brought to mind is important in view of the fact that emotional responsiveness as measured by EDA failed to index the subjective uncertainty of a response in TBI patients. A useful strategy to avoid deception is to refuse to respond unless one is certain of recollection. Knowing when not to respond will help curtail the number of situations where one is susceptible to being misled. Furthermore, understanding the capture route to false remembering has important implications for frontally impaired groups being able to adjust their responding through cognitive training. If poor cue-specification increases susceptibility to capture and false remembering then learning to generate alternatives to a misleading prime will help counter the influence of more general accessibility bias brought about by a poorly constrained retrieval focus during early selection of a response.

In conclusion, the current study demonstrates that Jacoby's capture framework can account for false remembering in patients with TBI. This extends the original application of the model with healthy older adults. Prefrontal damage or disruption to innervations of the prefrontal cortex may be responsible for TBI patients' increased susceptibility to capture compared with healthy controls. With regard to the executive processes underlying capture, the current study suggests that interference control processes prior to the engagement of cued recall may be a critically important frontally-mediated function that prevents capture and enables the initiation and maintenance of a retrieval mode during the cued recall. After cued recall, the evaluation of a false response in control subjects may profit from an enhanced electrodermal signal that indexes the uncertainty of the false response. The absence of this relationship between EDA responsiveness and subjective uncertainty in TBI patients may be partly responsible for their increased acceptance of false misinformation. Increased capture and poor evaluation of false misinformation in TBI patients raises concerns that this group, like older adults, is vulnerable to deception in everyday circumstances. The capture framework might be useful to identify individuals who are at risk from scams and may benefit from training strategies to counter deception and to exercise caution.

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