Spontaneous confabulators fail to suppress currently irrelevant memory traces

Armin Schnider and Radek Ptak

Division for Neuropsychological Rehabilitation, Department of Neurology, University Hospital, Bern and Clinique de Rééducation,Hôpital Cantonal Universitaire, 26, av. de Beau-Séjour, CH-1211 Geneva 14, Switzerland

Correspondence should be addresssed to A.S. (armin.schnider@hcuge.ch)

Human actions require integration of past experiences, ongoing percepts and future concepts. To adapt behavior to reality, the brain must identify mental representations of current relevance. Occasional amnesic subjects act according to invented stories ('spontaneous confabulations'), disregarding present reality. We used repeated runs of a continuous recognition task to measure the ability to distinguish currently relevant from previously encountered but currently irrelevant information. Spontaneous confabulators detected target items as accurately as nonconfabulating amnesics, but increasingly failed to suppress false-positive responses, confusing presentation in previous runs with presentation in the current run. Lesions involved the anterior limbic system: medial orbitofrontal cortex, basal forebrain, amygdala and perirhinal cortex or medial hypothalamus. We suggest that the anterior limbic system represents 'now' in human thinking by suppressing currently irrelevant mental associations.

Planning actions requires complex integration of stored knowledge (memory) and incoming information (perception) into future goals. Although memory and perception constitute partially dissociated processes, they rely on the activity of similar cortical neuronal networks[1,2]. At any moment, ongoing thinking is reflected in the activity of distributed networks that temporarily store information for processing[1–3]. It is obvious that, to plan meaningful actions that relate to current reality, the brain needs some mechanism to distinguish mental activity representing ongoing reality from memories and ideas.

Occasional amnesic patients fail to adapt behavior to current reality: they invent untrue stories about their recent doings and describe impossible plans for the day. These patients insist on the veracity of their stories and often act according to them[4,5]. These false ideas have been called spontaneous confabulations[4,6,7]. The patients typically consider themselves in different places, assume earlier dates and indicate other reasons for hospitalization than their memory impairment[8]. Whether the stories seem simple or fantastic, they can virtually always be traced back to fragments of actual experiences[4,5,9].

We have previously demonstrated that the only reliable distinction of spontaneous confabulators from nonconfabulating amnesics is an increased tendency to confuse the time at which information was last encountered[4]. Spontaneously confabulating amnesics failed to distinguish between pictures seen in the present run of a continuous recognition task and pictures that they had seen in a previous run one hour before. The failure paralleled the clinical course of spontaneous confabulators precisely[10]. These findings, in agreement with clinical observation, indicate that spontaneous confabulators confuse ongoing reality (the present run) with the past (the run one hour before).

In the present study, we explored the mechanism of this confusion. One possible explanation was that the patients fail to represent new, incoming information with normal saliency in memory, so that associations of old, firmly established information intrude into ongoing thinking[4,5]. A difficulty with this interpretation is that it would not explain why patients with extremely severe amnesia with no measurable storage capacity do not normally confabulate[11,12] nor why occasional patients also confabulate about old events[13–15]. An alternative explanation was that spontaneous confabulators cannot distinguish between representations of ongoing reality and previously acquired information because they fail to suppress activated memory traces and mental associations in the face of current reality.

Results

Two experiments were conducted. Experiment 1 involved a continuous recognition task for meaningful designs, some of which were repeated. Subjects were asked to indicate item recurrences (Fig. 1a). After the first run, three additional runs were made using the same set of pictures, but in randomized order with different items selected as the target items (recurring items). Before each run, subjects were instructed to forget that they had already seen the pictures and to indicate picture recurrences only within that run. The idea behind this procedure was that failure to strongly represent incoming information would be mirrored in defective target detection. If this was the reason for the confusion between previously presented and currently relevant information, spontaneous confabulators should have more difficulty in detecting target items than the nonconfabulating amnesics, that is, they should produce fewer hits. However, if the confusion resulted from an inability to suppress

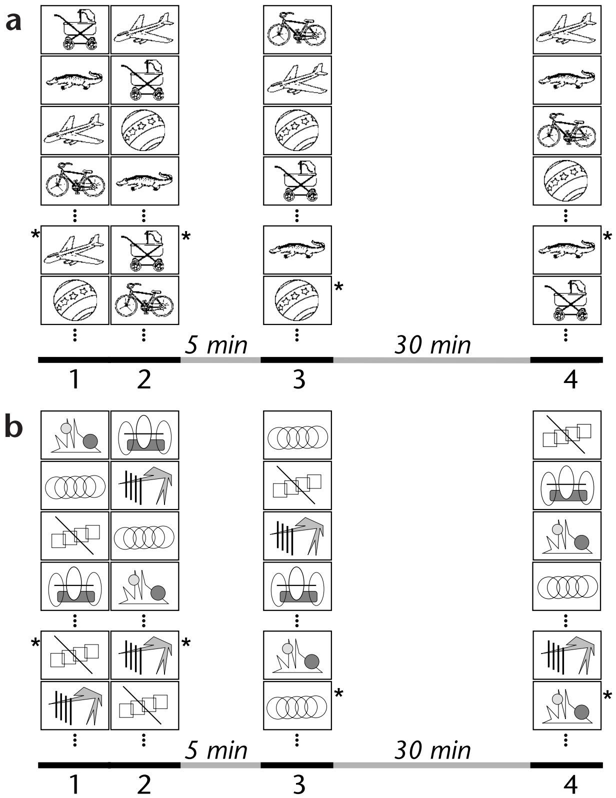

Fig. 1. Design of the experiments. **(a)** Experiment 1 with meaningful pictures. **(b)** Experiment 2 with nonsense geometric designs (see text and Methods for explanation). Stars (*) indicate target items, that is, recurring items in the respective run. The black bars at the bottom indicate test runs. Run 2 was made immediately after the first run. Run 3 was made 5 minutes after the end of the second run, run 4, 30 minutes after the end of the third run.

previously acquired information, spontaneous confabulators should produce increasingly more false-positive responses from run to run than the nonconfabulating amnesics. Because such an increase of false positives might depend on the interval between the runs (that is, the time allowed to forget previous targets), different intervals between the runs ranging from zero to thirty minutes were used. An implicit assumption in this experiment was that the confusion of memory traces is based on a defective representational process, independent of stimulus type; the same result should thus be obtained with meaningful (familiar) and meaningless (novel) information. Therefore, a second experiment was devised with exactly the same design as experiment 1 but with meaningless geometric designs (Fig. 1b).

Six spontaneous confabulators, 12 nonconfabulating patients with similarly severe amnesia and 10 healthy controls were examined (see Methods). As in our previous study[4], spontaneous confabulators performed similarly to nonconfabulating amnesics on the frontal executive tasks (Table 1). In both experiments, spontaneous confabulators and nonconfabulating amnesics had significantly fewer hits than the controls (two-way repeated measure ANOVA; experiment 1; Fig. 2a; effect of group, $p = 0.02$; experiment 2, Fig. 2c; effect of group, $p = 0.002$) but did not differ from each other. The number of hits did not significantly change between the test runs in either experiment (Fig. 2a and c). In contrast, in both experiments, spontaneous confabulators differed from both the nonconfabulating amnesics and the normal controls by their steep increase of false positives from run to run (experiment 1, Fig. 2b; effect of group, $p = 0.01$; interaction of group × run, $p = 0.0002$; experiment 2, Fig. 2d; effect of group, $p = 0.01$; interaction of group × run, $p = 0.0008$). Even after an interval of 30 minutes (between runs 3 and 4), their false positives increased (meaningful designs, Fig. 2b) or failed to decrease (meaningless designs, Fig. 2d). Target items from previous runs were more likely to be falsely recognized as a target item in a subsequent run: although only 12% of all distracter items in all runs had been a target item in a previous run, they accounted for 21% of all false-positive responses by spontaneous confabulators in experiment 1 ($\chi^2 = 30$; $p < 0.0001$) and for 26% in experiment 2 ($\chi^2 = 48$; $p < 0.0001$).

Interestingly, both nonconfabulating amnesics and controls experienced some interference in the second run of experiment 1 (Fig. 2b), but they suppressed this interference in the third and fourth run. In experiment 2, normal controls had surprising initial difficulty in differentiating among geometric designs and had more false positives than both patient groups, but they decreased their false positives in subsequent runs (Fig. 2d).

Lesion analysis

There was extensive overlap of the spontaneous confabulators' lesions in the basal forebrain and the medial orbitofrontal cortex (Fig. 3a). Only one patient with herpes simplex encephalitis had damage markedly extending beyond this area. The smallest lesion was a medial hypothalamic granuloma due to neurosarcoidosis. These areas were not damaged in nonconfabulating patients; their lesions overlapped extensively in the posterior medial temporal lobe and the dorsolateral prefrontal cortex (Fig. 3b).

Table 1. Neuropsychological test results.

	Spontaneous confabulators	Other amnesics	Normal value
CVLT			
Learning	28.6 ± 10.5	25.2 ± 6.5	55.5 ± 8.5
Long-delay recall	3.0 ± 2.8	2.9 ± 1.6	12.9 ± 2.5
Recognition (hits)	14.2 ± 2.9	9 ± 4	15.4 ± 1.0
Verbal fluency			
Correct words	14.3 ± 3.1	15.1 ± 7	33.3 ± 8.2
Perseverative errors	4.0 ± 3.0	2.4 ± 3.6	0.7 ± 1.3
Nonverbal fluency			
Correct designs	13.2 ± 2.7	16.8 ± 7.9	37.7 ± 9.7
Perseverative errors	4.3 ± 6	1.8 ± 1.8	1.9 ± 2.8
Color-word interference			
Time	33.0 ± 12.5	34.8 ± 19.1	23.8 ± 3.2
Errors	2.0 ± 2.0	1.9 ± 2.6	0.8 ± 0.9

Performance of spontaneous confabulators and other amnesics and normal range on tests of memory (CVLT, California Verbal Learning Test) and the following frontal executive tasks: verbal fluency[45], nonverbal fluency[46] and color-word-interference task[47].

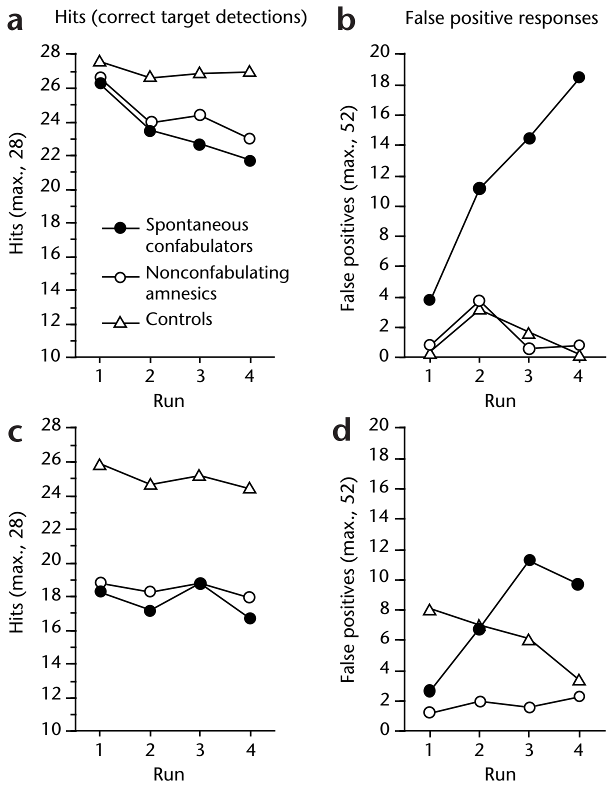

Fig. 2. Results of experiment 1 with meaningful designs **(a, b)** and experiment 2 with meaningless, geometric designs **(c, d)**. In both experiments, spontaneous confabulators and nonconfabulating amnesics had significantly fewer hits than the controls but did not differ from each other **(a, c)**. In contrast, in both experiments, spontaneous confabulators differed from both nonconfabulating amnesics and normal controls by their steep increase of false positives from run to run **(b, d)**.

Discussion

In this study, two experiments were conducted to explore why spontaneous confabulators confuse memory traces with ongoing reality[4,5]. Both experiments yielded essentially the same result: failure to detect new target items was common to both confabulating and nonconfabulating amnesics. Therefore, failure to saliently represent incoming information does not explain spontaneous confabulations. In contrast, spontaneous confabulators, but not nonconfabulating amnesics or healthy subjects, steeply increased false positive responses from run to run and failed to suppress this interference even after 30 minutes. This finding indicates that spontaneous confabulators experience information encountered 30 minutes ago as if it had just been presented in the current test run—the 'now'.

The present findings are compatible with the idea that spontaneous confabulations reflect a defective 'monitoring' process on memory retrieval, that is, a failure to monitor the veridicality of thoughts[16–24]. The nature of this monitoring process has never been specified. Our findings indicate that spontaneous confabulators specifically fail to suppress mental associations that do not pertain to the present; memories thus seem to be as real and pertinent for present behavior as representations of current reality.

This mechanism may also explain the clinical observation that even information acquired weeks or months ago may intrude on the thought of spontaneous confabulators[5]. It is conceivable that the weaker a patient's ability is to suppress even distant mental associations, the more bizarre and fantastic the confabulations appear. Incidentally, in this study group, the patient producing the most bizarre confabulations, often composed of events from several decades, clearly performed worst in experiment 1 (highest increase of false positives, constant number of hits).

The increase of false positives observed in our experiments cannot be attributed to a change of response behavior (a bias to say "yes") or an insufficiently focused, broad search in memory[23,24]. In both cases, the number of hits would be expected to increase in parallel with the false positives. However, the number of hits did not increase (Fig. 2c) or even tended to decrease (Fig. 2a).

The observed behavior also cannot be explained in terms of a nonspecific failure of action planning, that is, frontal executive failures: in agreement with our previous study[4], spontaneous confabulators did not differ from nonconfabulating amnesics in common measures of frontal lobe function. This finding opposes the suggestion that spontaneous confabulations are based on the combination of an amnesia with frontal executive failures[7,9,16,25–28].

Our experiment demands the ability to detect in each run the currently relevant (repeated) items. The spontaneous confabulators' failure might thus be interpreted as a loss of a 'temporal label' of stored information. Previous studies have indeed demonstrated spontaneous confabulators who failed to indicate the temporal order of previously presented information but recognized the information itself[5,29]. However, this failure has also been demonstrated in nonconfabulating amnesic patients[30–36] and nonamnesic patients with dorsolateral prefrontal lesions who did not confabulate[37–40]. The present study suggests that the sense for what information pertains to 'now' depends on the saliency of the mental representation of this information rather than on a separate 'temporal tag'. This saliency seems to depend critically on ability to suppress currently irrelevant mental associations of previously encountered information.

The part of the brain critical for suppression of currently irrelevant information appears to be the anterior limbic system. Spontaneous confabulators' lesions differed from nonconfabulating amnesics' lesions by the involvement of the medial

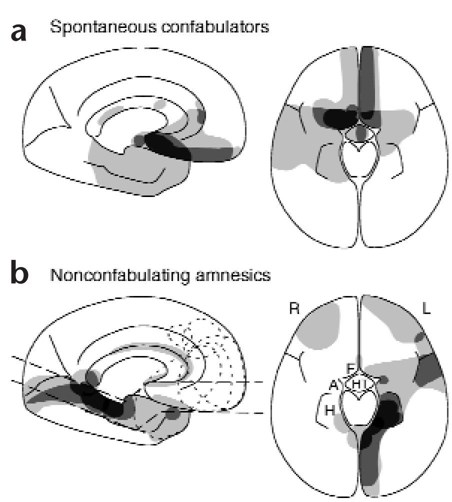

Fig. 3. Lesion analysis of **(a)** the spontaneous confabulators and **(b)** the nonconfabulating amnesics. The left column shows the sagittal view. Shaded areas indicate paramedian lesions; dashed lines indicate lateral lesions. The straight, parallel dashed lines in the lower part of the sagittal view in **(b)** indicate the composite axial slice used in the right column to indicate lesions of the amygdala (A), basal forebrain (F), hippocampus (H), hypothalamus (HT) and orbitofrontal cortex.

orbitofrontal cortex, the basal forebrain or the medial hypothalamus (Fig. 3). These findings complement our previous data demonstrating a similar lesion overlap[4,8]. In addition, one spontaneous confabulator had amygdala damage on one side and anterior insular damage on the other side[4]. Another patient had a lesion of the right capsular genu with interruption of the fibers connecting the thalamus (dorsomedial nucleus) with the orbitofrontal cortex[5]. Lesions were not consistently lateralized either in our previous[8] or present study.

Our findings have several implications. First, they indicate that spontaneous confabulations result from a previously unrecognized deficiency: an inability to suppress activated memory traces and associations at the right time. In addition, this study shows that inability to freely recall information from memory—the most typical aspect of amnesia[41]—may be associated with activation of too many memory traces, rather than a lack of traces.

The second implication goes beyond the clinical syndrome: The anterior limbic system seems to constitute a mental control system that is essential for adjusting thinking and behavior to ongoing reality. This system represents ongoing reality by suppressing mental associations that lack current behavioral relevance. The system is probably not specific to humans. Animals with orbitofrontal lesions show similarities to our patients' behavior: they fail to suppress previously established habits and continue to react to stimuli that are no longer rewarded[42,43]. Although it is unknown how these animals would spontaneously behave outside the laboratory, their behavior, too, indicates a failure to distinguish between currently relevant and previously encountered information; that is, between 'now' and the past.

Methods

Patients. All subjects gave informed consent before being tested. The study was approved by the Ethical Committee of the Medical School of the University of Bern. Selection criteria were similar to those used in previous studies[4,8]. Patients were asked to participate if they had a severe memory impairment, but lacked evidence of a confusional state (digit span ≥ 5, normal sleep–wake cycle, good attention) or any indication of aphasia or visual agnosia. As in our previous study[4], patients were classified as spontaneous confabulators if they produced confabulations with no external trigger and occasionally acted according to the confabulations. Because all spontaneous confabulators had a delayed free recall of ≤ 6 in the California Verbal Learning Test[44], the nonconfabulating amnesics were also selected on the basis of this criterion. At the time of the experiments, all patients were also tested on the following measures of frontal executive functioning: number of correct items and perseverative errors in a verbal fluency[45] and a nonverbal fluency task[46] and time and number of errors in the interference run of a color-word-interference task[47]. All were inpatients at the time of testing except for one spontaneous confabulator.

Etiologies of brain damage of the spontaneous confabulators ($n = 6$) were as follows: hemorrhage and operation of an aneurysm of an anterior communicating artery aneurysm ($n = 3$), hypothalamic granuloma due to neurosarcoidosis ($n = 1$), bleeding and operation of a macroadenoma of the pituitary gland with destruction of the hypothalamus ($n = 1$), herpes simplex encephalitis ($n = 1$); mean age ± standard deviation was 50 ± 12.4 years, mean years of education was 13.8 ± 2.6. The nonconfabulating patients ($n = 12$) had the following etiologies of brain damage: brain trauma ($n = 5$), brain infarction ($n = 3$), hemorrhage from an aneurysm of the anterior communicating artery ($n = 1$), herpes simplex encephalitis ($n = 1$), age-associated memory impairment ($n = 1$), meningioma of the falx ($n = 1$); mean age was 46.4 ± 13.7 years, mean years of education was 11.6 ± 2.5. The controls ($n = 10$) had similar age (42 ± 12 years) and education (13 ± 3 years) and were mostly patients' spouses.

Experiments. The general design of the experiments is explained in Fig. 1. Experiment 1 (Fig. 1a) was composed of meaningful pictures from Snodgrass and Vanderwart[48], experiment 2 was composed of nonsense geometric designs (Fig. 1b). Both experiments had the same design: in all runs, 80 items drawn from a constant set of 52 items were presented. Unknown to the subjects, a series of 80 items was composed of 8 groups of 10 items each. Four of these 10 items were present in all 8 groups and thus recurred 7 times after initial presentation (28 targets); the other 6 items would not recur (52 distractors). Subjects were requested to indicate item recurrences. All items were presented on a computer screen for 2000 ms. The test subjects' responses were typed in by the examiner. A warning beep sounded 700 ms after the response, and the next item was presented

Target items were different in each run: the four target items from one run were among the distractors in all subsequent runs, whereas four previous distractors were randomly selected as the target items of the next run. The distribution of the target items within each group of ten items and the order and distribution of all distractors were randomized in each run. Immediately after the first run, a second run was made. Subjects were asked to forget that they had just seen all items and that they should indicate item recurrences only within this second run. During the run, the question "Have you seen this picture in this very run yet?" was repeated several times. Five minutes after the second run, the third run was made. Thirty minutes after the third run, the fourth run was made (Fig. 1).

Lesion analysis. The lesion analysis was made in a similar fashion as in our previous study[8]. The CT scans or MRI performed for clinical reasons were used. Lesions were reconstructed with the templates of Damasio and Damasio[49] and referred to a composite axial slice containing the hippocampus, amygdala and basal forebrain and to the midsagittal plane (Fig. 3b). The lesion areas were superimposed in a commercial drawing program.

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