Confabulation: A Bridge Between Neurology and Psychiatry?

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Mr A is a 73-year-old resident of a nursing home, where the irate aides describe him as “a liar and a troublemaker.” Mr A’s “stories” were regarded by the staff as deliberate mischief on his part.

Confabulation was first described by Korsakoff in amnesic patients in 1889, yet its nature and etiology remain a source of some controversy. Indeed, in the literature since Korsakoff’s time, even the definition of “confabulation” has been inconsistent, reflecting the diverse theories concerning the origins of this syndrome. Thus, confabulation has been variously described as a falsification of memory in association with an organically derived amnesia, an extreme form of lying or deception, and “honest lying.” In more recent texts, confabulation is usually defined as statements and/or actions that are “unintentionally incongruous to the patient’s history, background, and present situation,” or as erroneous memories—either false in themselves or derived from true memories inappropriately retrieved or interpreted.

Here we broadly define confabulation as the production of false or erroneous memories without the intent to deceive. As our opening vignette illustrates, the confabulating patient’s intentions may sometimes be misread as malicious, when in reality his confabulations may be better understood as products of one or more neuropsychological deficits. The false memories of confabulation may range from inaccurate or distorted recollections of past events, to incongruous intrusions during memory tasks, to fictitious and bizarre narratives.

In this article, we place confabulation in a historical perspective, review current theories of confabulation, and discuss clinico-anatomical syndromes often seen by psychiatrists in which confabulation frequently occurs. Finally, we summarize the implications of recognizing and understanding confabulation in psychiatric practice.

TYPES OF CONFABULATIONS: HISTORICAL OVERVIEW

In 1901, Bonhoeffer distinguished the confabulation of embarrassment (later termed “momentary confabulation”) from spontaneous confabulation. Confabulation of embarrassment referred to fabricated memories that appeared to compensate for memory loss—in effect, the patient attempts to cover up an exposed “gap in memory.” In contrast, Bonhoeffer described spontaneous confabulation as exceeding the need to cover a memory deficit and often consisting of “fantastic” (grossly implausible) content. Berlyne, in 1972, also delineated 2 distinct forms of confabulation. Like Bonhoeffer, he referred to the first as “momentary” confabulation and the second as “fantastic” or “productive” confabulation. Momentary confabulations, for Berlyne, consisted of autobiographical content and were rooted in true memory. Furthermore, these confabulations occurred only in response to questioning. In contrast, fantastic (or productive) confabulations consisted of grandiose content and occurred without provocation.

Kopelman later revised this terminology, using the terms “provoked” (rather than “momentary”) and “spontaneous” (rather than “fantastic”) confabulation. Kopelman noted that provoked confabulations commonly occurred among amnesic patients given memory tests and resembled errors produced by healthy persons on tests of memory following prolonged retention intervals. Indeed, provoked confabulations have since been noted in various experimental studies to occur in healthy people. Accordingly, many authors consider provoked confabulations to be a normal strategy to compensate for memory deficits rather than a pathological process. Kopelman, for example, notes that provoked confabulations under experimental conditions—are defined as intrusion errors or distortions made in response to a challenge to memory—reflect the “reconstructive” nature of
normal memory retrieval. When a memory trace is particularly weak (for example, after long
retention intervals), reconstruction becomes distorted or frankly erroneous.\textsuperscript{11}

Provoked confabulations may reflect such normal compensatory mechanisms. However, several
recent studies have shown that provoked confabulations may also reflect neuropathological
conditions, such as Wernicke-Korsakoff encephalopathy\textsuperscript{14,15} and Alzheimer disease (AD).\textsuperscript{16,17}

Many authors continue to use Kopelman’s “spontaneous” versus “provoked” dichotomy. Some
argue, however, that these are not distinct types of confabulation. Rather, spontaneous
confabulation may simply represent a more severe form of memory falsification.\textsuperscript{12,18,19}

**CLASSICAL CONFABULATION HYPOTHESES AND THEIR CRITICS**

**Confabulation and memory impairment**

Confabulation is classically and historically associated with memory loss. As noted, one of the
earliest hypotheses proposed that confabulations occurred as a compensatory mechanism for
memory loss (ie, the patient produces confabulations to fill in memory gaps and avoid
embarrassment).\textsuperscript{4} Yet numerous authors have challenged this view, noting that patients with
memory deficits do not always demonstrate confabulations,\textsuperscript{4,20} that confabulation usually resolves
during the chronic stage of Korsakoff syndrome despite ongoing memory deficits,\textsuperscript{3} and that the
severity of amnesia does not correlate with the tendency to confabulate.\textsuperscript{21} Furthermore, confabulation has been observed in the complete absence of memory deficits.\textsuperscript{22-24}

**Frontal lobe lesions**

Because confabulation was frequently seen in patients with frontal lobe damage it led to the belief
that frontal lesions were the cause of confabulation.\textsuperscript{19,20,25,26} However, several experiments suggest
that frontal lobe pathology may be neither necessary nor sufficient for confabulation. On the one
hand, for example, there are reports of patients who confabulate but who show no signs of frontal
executive dysfunction or frontal lobe structural pathology.\textsuperscript{18,24} On the other hand, controlled studies
have found that common executive function deficits reflecting frontal lobe impairment do not
distinguish spontaneously confabulating amnesiacs from nonconfabulating amnesiacs—suggesting
that frontal dysfunction is not sufficient to produce confabulation.\textsuperscript{13,27,28} Moreover, in a study of
patients with AD who had provoked confabulations, the tendency to confabulate did not correlate
with performance on frontal/executive tasks.\textsuperscript{29}

Partly as a consequence of these uncertainties, the essential role of frontal lobe pathology in
confabulation has been challenged by several investigators.\textsuperscript{30-33} This skepticism, in turn, led to the
“dual-lesion” hypothesis, which states that confabulations arise from the concomitant presence of
frontal lobe pathology and an organic amnesia.\textsuperscript{19,25,34,35}

**MORE RECENT HYPOTHESES OF CONfabULATION**

More recent views of confabulation focus on 3 central problems:

- Deficits in reality monitoring
- Dysfunction of strategic retrieval processes
- Temporal confusion

**Reality/source monitoring**

Reality monitoring (or source monitoring) refers to the neural mechanisms by which memories are
“checked” to ensure that they correspond to actual (vs imagined) events.\textsuperscript{36} For example, one might
think, “Did I really see Jim at the office party last year, or did I just dream that?” According to the
reality- or source-monitoring deficit hypothesis, dysfunction or loss of these “fact-check”
mechanisms results in confabulations. However, source-monitoring deficits may be seen in
nonconfabulating patients, which suggests that such deficits may be necessary but not sufficient to
produce confabulation.\textsuperscript{28,33,37} Even more troubling for the source-monitoring hypothesis was the
demonstration by Dalla Barba and colleagues\textsuperscript{29} that the degree of source-monitoring deficits in a
group of patients with AD who had provoked confabulations did not correlate with the tendency to
confabulate.

**Strategic retrieval**

Confabulation affects remote memories—acquired before brain damage—as much as recent
memories acquired subsequent to injury. Thus, an elderly patient who suffered a stroke 2 years ago
may be as likely to confabulate about his army days during WW II as about his breakfast this
morning. This observation has led to the hypothesis that confabulation is more the result of a deficit
in retrieval than of a problem in encoding (registering) memories in the first place.\textsuperscript{38} (A problem with
encoding would predict confabulation only with respect to memories acquired since the brain
damage.) Strategic retrieval refers to memory processes in which the individual uses a purposeful,
“problem-solving” strategy to call up the desired memory. For example, someone trying to
remember the name of a person he met at a party might begin by thinking, “Let’s see, I was standing next to the punch bowl. Then Mary said she wanted to introduce me to a friend of hers. Then the friend said . . .,” etc. Thus, memory traces are deliberately organized by context, theme, and temporal order. According to the strategic retrieval hypothesis, a defect in these search processes ultimately leads to spontaneous and/or provoked confabulations. However, Nedjam and associates argue that if strategic retrieval deficits were responsible for confabulations, they should affect episodic and semantic memory equally. (Episodic memory involves autobiographical, contextual, and highly specific information, such as “I ate fish for dinner today”; semantic memory involves general or conceptual knowledge, such as “rectangles have 4 sides.”) In fact, however, confabulations have consistently been shown to affect episodic memory more than semantic memory—thus casting doubt on the strategic retrieval hypothesis.\(^{24}\)

**Temporal confusion**

Finally, there is the temporal confusion hypothesis of confabulation, which is derived from the observation that spontaneous confabulations can typically be traced back to actual (rather than fictitious) events.\(^{3,13,19}\) This hypothesis holds that confabulations arise from the misattribution of aspects of past events to ongoing reality.\(^{4,17,24}\) For example, Schnider and coworkers\(^{40}\) described a 62-year-old woman who exhibited confabulations following a stroke that appeared to consist of mistakenly arranged elements of actual events: “She seemed to recognize the personnel on the ward but often confused their names or confabulated on the circumstances of their meeting. For example, when asked whether she recognized one of the examiners, she explained: ‘You are Dr S (correct). We did physiotherapy this morning (she had had physiotherapy, but not with Dr S), then we worked on the computer (she had regular computer training, but not that morning and never with Dr S) where you showed me a camel and other animals composed of dots (referring to a perceptual priming test taken 2 weeks previously)’.”\(^{40}(p187)\)

Thus, consistent with the temporal confusion hypothesis, Schnider and coworkers\(^{40}\) demonstrated that a patient with spontaneous confabulations was capable of storing novel information normally but was unable to store the temporal order of the acquired information—what the authors aptly call, “memory without context.”

**CLINICO-ANATOMICAL CORRELATIONS**

Confabulation has been described in numerous neurological conditions, including dementia, traumatic brain injuries, anterior and posterior communicating artery aneurysm rupture or repair, subarachnoid hemorrhage, brain tumors, CNS infections, Wernicke-Korsakoff syndrome, and multiple sclerosis. Regardless of the inciting conditions from which they arise, spontaneous confabulations have been associated with lesions of the medial orbitofrontal cortex and its associated anterior limbic structures; namely, the basal forebrain, medial hypothalamus, right capsular genu, and dorsomedial thalamic nucleus.\(^{41}\) The medial orbitofrontal cortex and basal forebrain are supplied by the anterior communicating artery, which connects the left and right anterior cerebral arteries in the circle of Willis. As exemplified in our opening vignette, rupture or aneurysm of the anterior communicating artery is disproportionately associated with the development of spontaneous confabulations compared with other neurological conditions.\(^{19,31}\) Schnider and associates\(^{13}\) have previously observed that provoked confabulation has been associated with dorsolateral prefrontal as well as medial temporal (hippocampal) lesions. However, provoked confabulation has also been observed in neurologically healthy persons.\(^{12}\) Therefore concluded that provoked confabulations have no anatomic specificity.

In contrast, a recent neuroimaging study by Turner and coworkers\(^{42}\) provided striking evidence that the critical deficit for provoked confabulation has its anatomical location in the inferior medial frontal lobe. Curiously, whereas Turner’s group examined only provoked confabulations, the lesions observed are consistent with those that are associated with spontaneous confabulations as described above. This suggests there may be substantial overlap in the neuroanatomical loci mediating these 2 types of confabulation.

**CONFABULATION IN PSYCHIATRIC SETTINGS**

**Wernicke-Korsakoff syndrome**

As noted, Korsakoff was the first clinician to describe confabulation formally; he observed the syndrome predominantly in chronic alcoholics but also in patients with other conditions.\(^{1}\) Today, Korsakoff syndrome is generally defined as the chronic amnesic syndrome that frequently follows acute Wernicke encephalopathy. The latter comprises the classic triad of confusion, ataxia, and ophthalmoplegia.\(^{43,44}\) So-called Wernicke-Korsakoff syndrome is believed to result primarily from thiamine deficiency and can be seen in a myriad of medical conditions besides chronic alcoholism, including malnutrition, protracted vomiting, carbohydrate loading, chronic renal failure, and other
Patients with Korsakoff syndrome tend to confabulate most within the episodic/autobiographical memory domain. Although both spontaneous and provoked confabulations have been described in patients with Korsakoff syndrome, provoked confabulations are more common. Curiously, however, most neuroanatomical correlations have been established for the spontaneous confabulations less commonly seen in Korsakoff syndrome. Anatomical areas of the brain affected in patients with Korsakoff syndrome typically involve diencephalic regions such as the mammillary bodies and the region enclosed by the anterior and mediodorsal thalamic nuclei. Lesions in the medial thalamic nuclei are believed to give rise to spontaneous confabulations in patients with Korsakoff syndrome. These nuclei contain projections to and from the posterior orbitofrontal cortex.

**Alzheimer disease**

In addition to the well-known memory problems seen in patients with AD, so-called intrusions are also frequently observed in these individuals. Intrusions are defined as unintentional productions of inappropriate responses in a memory task, and they have similarities to provoked confabulations. A recent study suggests that intrusions in patients with AD may be the result of interference of strongly represented, over-learned material in episodic memory. For example, if patients with AD are told to remember a deliberately altered version of a well-known fairy tale—such as “Goldilocks and the Four Bears”—they will often recount the story in their habitual, over-learned mode as “Goldilocks and the Three Bears.” Some neuropsychologists consider such intrusions as essentially provoked confabulations.

More recently—and perhaps more relevantly for psychiatrists—confabulation in AD has been associated with psychotic features and aggressive behavior. Lee and associates studied 32 AD patients and 10 healthy controls and found that those with AD confabulated in response to all types of questions tested, including personal episodic memory, orientation to time, and future planning. However, those patients who also demonstrated delusional and/or aggressive behavior showed significantly more provoked confabulations than nondelusional and nonaggressive AD patients. Whether these results demonstrate that provoked confabulations in AD patients differ from those seen in other conditions, or whether delusions and aggression simply aggravate mechanisms that underlie all provoked confabulations requires further investigation.

**Schizophrenia**

It has been argued that the formal thought disorder observed in schizophrenia strongly resembles spontaneous confabulation, in that it is often unprovoked and the content is frequently bizarre or fantastic. In fact, some authors have asserted that delusions and formal thought disorder may be indistinguishable from spontaneous confabulations except for the clinical context in which they arise. These observations have led to the investigation of confabulations in patients with schizophrenia. Although formal thought disorder is typically associated with spontaneous confabulations, most studies in patients with schizophrenia have focused on provoked confabulations, because these can be elicited in a structured setting.

In a study of provoked confabulations by Nathaniel-James and Frith, 12 persons with schizophrenia and 12 controls were asked to recall 6 separate stories read aloud to them. A confabulation was defined as recall of information not present in the original narrative. Each person was also given a neuropsychological battery that included tests of memory and executive function. All of the schizophrenic participants confabulated to varying degrees, whereas only 1 control did so. Moreover, the authors found an association between the tendency to confabulate and the presence of formal thought disorder (eg, tangential thinking or loose associations). However, those with schizophrenia but without any formal thought process disorder also confabulated. The authors concluded that the presence of thought disorder may contribute more to the severity of confabulations than to their presence in these patients. They also found an association between the number of confabulations in schizophrenic patients and an impaired ability to suppress inappropriate responses, as demonstrated by tests of executive functioning. Not surprisingly—Simpson and Done found that delusions in those with schizophrenia also increased the frequency of confabulations, compared with nondeluded and nonpsychiatric controls.

**CONCLUSION AND FUTURE DIRECTIONS**

Confabulation is a significant clinical problem in many patients with neuropsychiatric disorders. As our opening clinical vignette suggests, a misunderstanding of confabulation can lead to
inappropriate counter-transference on the part of clinical staff. We have also tried to show how confabulation may represent a “bridge” between psychiatry and neurology. Indeed, we believe that our understanding of several neuropsychiatric syndromes may be enhanced by our knowledge of confabulation.

To cite one example: Eack and coworkers\(^{51}\) have pointed to deficits in “foresight”—the ability to think of the long-term consequences of one’s behavior—in many patients with schizophrenia. These researchers have amassed MRI data that suggest foresight in patients with schizophrenia is related to the amount of gray matter in the right orbitofrontal and ventromedial prefrontal cortex. In particular, they hypothesize that reductions in gray matter volume in these regions may be associated with impaired foresight in schizophrenia. We have noted considerable (though not unequivocal) evidence that links damage to the orbitofrontal cortex with spontaneous confabulation. We now ask: is there a link between impaired foresight and spontaneous confabulation in patients with schizophrenia? Furthermore, could any patient with damage to the orbitofrontal cortex—whether from stroke, demyelinating disease, or head trauma—be at increased risk for both impaired insight and confabulation? Now, both foresight and the accurate recollection of events require brain structures that can correctly assess and sequence past, present, and future. Could problems with such “temporal manipulation” be one thread that ties together schizophrenia and other conditions that involve orbitofrontal damage?

Answers to these questions must await further research but could have important implications for the cognitive rehabilitation of patients with various types of brain injury or disease.\(^{52}\) In short, we believe that confabulation is an excellent heuristic concept for building bridges between neurology and psychiatry.

**References**


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