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## A Cognitive Neuroscience Perspective on Confabulation

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# NEURO-PSYCHOANALYTIC DIALOGUE

## A Cognitive Neuroscience Perspective on Confabulation

John DeLuca

**Abstract:** *The purpose of this paper is to provide an overview of the behavioral and neuroscience research on confabulation. While most forms of organically induced confabulatory syndromes are reviewed, special emphasis on confabulation following anterior communicating artery (ACoA) aneurysm is provided. "Psychiatric" forms of confabulation will not be discussed at length in this paper. For instance, while confabulation has been observed in patients with schizophrenia, such a presentation appears to be related to a thought disorder (Nathaniel-James and Frith, 1996). This paper will focus on confabulation following acquired brain damage.*

### Introduction

In the late nineteenth century, Korsakoff (1889) described a behavioral disturbance in which patients would verbally present seemingly erroneous recollections that they believed were accurate or correct. In fact, when Korsakoff traced a considerable number of these recollections he found many of them to be actually true. This behavioral phenomenon was later labeled *confabulation* by Kraepelin (Koehler and Jacoby, 1978). Today it is well known that confabulation occurs across a variety of neurologic patients including those with Korsakoff disease, anterior communicating artery (ACoA) aneurysm (e.g., DeLuca and Cicerone, 1991), stroke and traumatic brain injury (TBI; e.g., Shapiro, Alexander, Gardner, and Mercer, 1981), and dementia (e.g., Kern, van Gorp, Cummings, Brown, and Osato, 1992). In fact, in his original articles, Korsakoff described confabulation in patients with lead poisoning, carbon monoxide poisoning, and bacterial infection (Korsakoff, 1889). Nevertheless, despite the numerous papers for over a century that have documented the existence of confabulation, the debate over what it is and what causes it remains to this day.

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### What Is Confabulation? Problems in Definition

The major conceptual problem encountered with any discussion of confabulation is the lack of a generally accepted definition for what it is. The problem that this creates should not be underestimated. Is a patient with anosognosia for hemiplegia with left visual neglect confabulating when they deny having problems in utilizing their paralyzed limb (Feinberg, Roane, Kwan, Schindler, and Haber, 1994)? Are patients with Anton's syndrome (i.e., cortical blindness), who deny their blindness, confabulating? Are patients who indicate that they have two wives who look exactly the same or state that there are two identical hospitals (Capgras syndrome/Reduplicative paramnesia) confabulating? While these are all manifestions of unawareness, it is unclear that they are all confabulation. Can all of these diverse behavioral expressions truly result from a single (or distributed) central mechanism in the brain critical to the expression of a singular confabulatory state? Or are there different forms of confabulation, each with its own underlying mechanisms? Or is confabulation a subset of a larger problem of unawareness of deficit or anosognosia? These questions remain unresolved.

Given the difficulties in defining confabulation, the following definition from Mosocovitch and Mello (1997) is perhaps the most general yet descriptive. These authors describe confabulations as "*statements or actions that involve unintentional but obvious distortions. . . .*" While this definition will likely not be universally accepted, it is a working definition that will be used in the present paper.

It should be recognized that some form of "confabulation" is a normal human phenomenon. This is because remembering is a reconstructive process, often resulting in memory distortion rather than memory loss (Mosovitch, 1995). However, when neural circuits involved in the reconstructive process are damaged, memory distortion can become prominent and is then recognized as confabulation. As a result, in clinical practice, there is the tendency to recognize the more "bizarre" responses as confabulatory because the distinction between normal memory distortion and "abnormal" responses is more obvious. Yet confabulation can be subtle, nonbizarre, and elicited, for instance, only upon provocation (i.e., provoked confabulation, see below).

At a minimum, confabulation involves both distortions of content and temporal context. That is, confabulatory recollections frequently include additions, distortions, or elaborations of events that either actually or plausibly occurred. As such, when a confabulatory patient is asked what they did today, they may incorporate events that may have actually occurred (e.g., seeing a therapist or doctor), but with the content distorted or intertwined with other seemingly unrelated events (which also may have occurred). However, confabulations are also frequently the intrusion of events which have actually taken place, but become displaced temporally. These two forms of distortion are illustrated by the following ACoA patient:

| | |
|---|---|
| Doctor | What did you do today? |
| Patient VR | Today I got up this morning and visited the rehabilitation institute . . . then I went home and I was expecting some material and we received it. Then I came to the rehabilitation institute, no I actually went to the Jimsburg store and we had a small meeting there. Then I came to the hospital and we had lunch and then met with you. . . . |
| Doctor | What did you do this past weekend? |
| Patient VR | There was a friend that used to work at the Jimsburg store, that moved away, that was a friend of mine for 10 years or so, who came by my house for a visit with his family. We went to New York City, and as a matter of fact, we stopped by the Jimsburg store to say hello to one manager that he also has become friendly with, but this store is not operating anymore. . . . |

Patient VR illustrates several key aspects of confabulation. First, content distortion is illustrated: Despite having been in the hospital for several weeks postsurgery, he states that he has been home, had a meeting at work, yet also correctly incorporates his knowledge about the rehabilitation hospital and the doctor's role within it. These events had not actually occurred on that day. Second, impaired temporal context is clearly evident in that VR had indeed owned the "Jimsburg store," but he had sold this store years earlier. After viewing the videotape, patient VR's wife indicated that the story about the friend visiting, going to New York City, and then seeing a mutual friend from the store had actually occurred, but years earlier. The third point illustrated by this excerpt is impaired self-monitoring. While first acknowledging having visited the Jimsburg store with his friend, in the same sentence he acknowledges that the store is no longer operational. Patient VR could not realize the temporal, nor the logical implausibility of being at the store and the hospital.

As such, confabulation often is a combination of temporal displacement of an actual event from the past into the present as well as the distortion of events that may be salient in the immediate environment (i.e., distorted content). However, it is the lack of the patient's own self-monitoring about the implausibility of the temporal and content displacement that has fascinated researchers and clinicians and resulted in theories to understand what drives such behavior.

### *Two Prominent Viewpoints on Confabulation*

In general, two broad schools of thought have developed in regard to the understanding of confabulation. The first conceptualizes confabulation as an "unaware" process while the second recognizes a conscious "awareness" aspect to confabulation. In general, the former conceptualization appears to come from the neurological literature while the latter tends to reflect work from the psychiatric literature.

Since Korsakoff's discoveries, many researchers have recognized that confabulation is frequently a temporal displacement of an actual event that had occurred in the patient's life, although this event can be embellished with the intrusion of nonactual events (Talland, 1965; Schnider, Gutbrod, Hess, and Schroth, 1996; Ptak and Schnider, 1999). In other words, while these confabulatory recollections frequently include additions, distortions, or elaborations, they are thought to be based on actual events. Importantly, confabulators are unaware that they are confabulating, nor are they aware of the temporal displacement of their confabulations. For instance, Talland (1965) reported that in patients with Korsakoff's syndrome, "The patient himself is almost certainly not aware of the gaps in his knowledge when he confabulates; he transposes information from an earlier period in his life, condenses or distorts material without consciousness of his deficit or of the confabulatory process, and hence without intent" (p. 57).

| | |
|---|---|
| Doctor | You indicated that last night you were working on a number of projects at home . . . . What would you say if I told you you were actually here in the hospital last night? |
| Patient OJ | I'd be surprised, because my experience, what I learn from my eyes and ears tells me differently . . . . I'd want some evidence. I'd want some indication that you knew about my private world before I gave any cognizance. |
| Doctor | Would you believe me? |
| Patient OJ | Not out of the blue, especially since we haven't even met (an illustration of the patient's amnesia). |
| Doctor | What if your wife was here and she agreed with me, what would you think at that point? |
| Patient OJ | I'd continue to resist, but it would become more difficult. |

Patient OJ was firm about the truth of his recollections, and resisted attempts to convince himself otherwise. It was clear from his words, his affect, and his behavior that there were no "gap-filling" responses.

A handful of studies were designed to experimentally examine the temporal context hypothesis (Schnider, von Daniken, and Gutbrod, 1996; Moscovich and Melo, 1997). For instance, Schnider, von Daniken, and Gutbrod (1996) found that all spontaneous confabulators, but not nonconfabulating amnesics, confused present with previously acquired information on a continuous recognition task. However, Moscovitch and Melo (1997) argued that defective temporal order is not the cause of confabulation, but is a symptom of a more fundamental deficit in strategic retrieval (see below).

Ptak and Schnider (1999) reported an amnestic ACoA patient whose confabulations could always be traced back to actual experiences, supporting the notion that the key difficulty in confabulation is the temporal monitoring of actual memories. The patient was unaware of his memory problems, and was convinced that his beliefs were correct, arguing with hospital staff. His confabulation gradually disappeared over a 4-month period.

In summary, the temporal displacement concept has been recognized for over 100 years, and is widely accepted today by many researchers and clinicians. A major focus of this position is that patients are unaware of the temporal displacements and do not have the self-monitoring ability to critically examine the inconsistencies in their discourse.

However, by the turn of the century, a second line of thinking began to emerge, emphasizing the fabricated or fictitious elements of confabulation more prominently. First Bonhoffer (1901; cited in Talland, 1965) and later van der Horst (1932; cited in Schnider, von Daniken, and Gutbrod, 1996) felt that confabulation reflected a desire to fill gaps in memory, which was termed "confabulation out of embarrassment." That is, confabulators somehow contrive a story to protect themselves from embarrassment. This notion of "gap filling" would then require some awareness of a memory disorder on the part of the patient. The problem is that there is very little empirical support for this position. In fact, the few studies that empirically examined gap-filling behavior did not support this hypothesis (Moscovitch and Melo, 1997; Schnider, von Daniken, and Gutbrod, 1996). For instance, Schnider, von Daniken, and Gutbrod (1996) classified amnesic patients (including ACoA) into "spontaneous" versus "provoked" confabulators. They reported that confabulators did not show an increased tendency to fill in gaps in memory. Despite the lack of evidence, this notion of gap filling is maintained even today. For instance, the DSM-IV (American Psychiatric Association, 1994) defines confabulation as "the recitation of imaginary events to fill in gaps in memory" (p. 157). Numerous psychiatry textbooks also define confabulations as conscious gap filling, with a conscious wish to deceive (Whitlock, 1981). Weinstein and Lyerly

(1968) defined confabulation "as the *fictitious* narrative of some past event or events" (p. 348). Benson et al. (1996) define confabulation as "compensation for loss of memory by the fabrication of details" (p. 1239).

Joseph (1986) reported that confabulation from gap filling can occur following cerebral disconnection syndromes. For instance, Gazzaniga and colleagues (Phelps and Gazzaniga, 1992; Gazzaniga, 1998) described a laboratory-induced confabulation in split-brain patients resulting in a form of confabulation that requires awareness of one's own behavior, but unawareness of why one is behaving in a particular way (gap filling without awareness). Gazzaniga (1998) reported that when the speechless right hemisphere of a split-brain patient is given a command to "take a walk," the patient begins to execute the demand. However, when the experimenter now asks the patient (i.e., the left hemisphere) "where are you going," the left (language) hemisphere responds "to get a drink of water." Such a confabulatory response is made because the language hemisphere does not have access to the information in the right hemisphere that initiated the original behavior. As such, according to Gazzaniga, the left hemisphere contains a mechanism (the "Interpreter") that seeks to interpret information both internally and externally to logically explain the events in the environment. In this example, the split-brain patient was able to examine the immediate situation and construct a plausible, but untrue response. Is this confabulation from unawareness? While they are indeed filling in gaps, are such patients aware of a cognitive disorder that they need to cover up? The answer is clearly no. In this example, the left hemisphere did not have access to the knowledge that prompted the original behavior (to take a walk), and thus was not aware of the reason for the behavior (see Schacter, 1991, for a distinction between unawareness of deficit and unawareness of knowledge). While the Interpreter is conscious of its own behavior, it is not aware of the motivation that is driving the original behavior, and hence is not consciously hiding a memory or any other cognitive disorder.

There are several other problems with the simple gap-filling explanation of confabulation. For instance, confabulatory amnesics typically only carry out gap filling during the early stages of their amnesia, despite the fact that their amnesia is long lasting. Why would such patients only fill in gaps early on in their illness? Confabulation does not occur among many amnesics, including many Korsakoff patients, and is very rare among mesial temporal amnesics. In addition, many amnesics readily admit to gaps in their memory without confabulating (McGlynn and Schacter, 1989).

Other early conceptualizations of confabulation included: "a disturbance in symbolism, comparable to that which occurs in dreaming, . . . wish-fulfilling fantasies, . . . the means whereby sexually traumatic material could be expressed and gratified" (Weinstein and Lyerly, 1968, p. 348). However, there is very little evidence to support these very early conceptualizations of confabulation, typically relying on subjective observations of behavior. Some believe that premorbid personality traits are important determinants in who will eventually confabulate (Weinstein and Kahn, 1955; Talland, 1965). However, this notion is based exclusively on anecdotal data, usually involves individual case reports, and has not been systematically evaluated experimentally. Weinstein (1996) argues that confabulations are "in some degree, symbolic representations, dramatizations, or explanations of some current personal experience, disability, or problem" (p. 336). Weinstein indicates that in such cases the patient has some knowledge of their disability or problem. He does, however, acknowledge that this type of symbolic confabulation is less likely to occur in patients with ACoA aneurysm.

In summary, confabulation involves both the temporal displacement of actual events, and distortions of content. Most contemporary researchers believe that confabulators believe and defend their confabulations and are not engaged in an active need to fill in gaps to avoid embarrassment or hide a memory disorder. Nonetheless, these two disparate conceptualizations of confabulation remain today: unaware temporal displacement versus an active, aware gap filling.

## Factors Associated with Confabulation

As discussed above, confabulation typically includes some element of both content distortion and temporal displacement of actual events. Severe confabulation (e.g., following ACoA aneurysm) usually appears acutely, and typically lasts for a period of weeks to months, although it can, rarely, last for years (Talland, 1965; Weinstein, 1996). Several variables important in understanding confabulation are presented in Table 1.

It is very important to differentiate confabulation from what is commonly observed in patients during an acute confusional state. While confabulation can be observed during such confusional states, continued confabulation following resolution of an acute confu-

TABLE 1
**Important Variables in the Understanding of Confabulation**

| |
|---|
| Awareness vs. Unawareness |
| Temporal Displacement vs. Content Distortion |
| Premorbid Personality Factors |
| Impaired Reality Monitoring |
| Requires Coexistence of Amnesia vs. No Amnesic Requirement |
| Frontal (or Executive) Dysfunction Alone Sufficient for Confabulation |
| Differentiated from Acute Confusional State |
| Impaired Strategic Retrieval |
| Disconnection Syndrome |
| Subtypes of Confabulation |
| Indifference or Apathy vs. Deceit or Lying |

sional state is rare, and usually limited to circumscribed lesions. For instance, DeLuca and Cicerone (1991) studied confabulation in ACoA patients in two naturally occurring conditions: while disoriented and again with the return of orientation to person, place, and time. Confabulatory responses to directed questions were studied in ACoA patients and a heterogeneous group of patients with bleeds elsewhere in the brain. These authors reported that confabulation was observed in both groups during the disoriented stage (100% of ACoA subjects and 41% of the other group). However, with the return of orientation, all ACoA patients continued to confabulate, while confabulation was virtually nonexistent in the other intracranial hemorrhage group. DeLuca and Cicerone (1991) suggested that the prolonged confabulation observed in the ACoA subjects was a result of specific cerebral disturbance that included the frontal lobes, which is different from mechanisms involved with confabulation from an acute confusional state.

A distinction between confabulation and delusion is also important. While confabulation may at times seem difficult to distinguish from a delusion, confabulations typically involve specific episodes or events, while most delusions concern false beliefs (Weinstein, 1996). The American Psychiatric Association defines delusion as "A false belief based on incorrect inference about external reality that is firmly sustained. . . . The belief is not one ordinarily accepted by other members of the person's culture . . ." (p. 765).

Confabulators, particularly following ACoA aneurysm, will defend their confabulations as veridical, and defend their statements readily, as illustrated by patient OJ above.

## Subtypes of Confabulation

Korsakoff (1889) described a continuum of confabulation based on severity. However, Kraepelin (1904, 1907, 1919) proposed two subtypes: (1) *Simple* confabulation was defined as minor distortions or recall of fact, time, or detail; (2) *fantastic* confabulation consisted of bizarre, exaggerated, florid, or impossible verbalizations. Berlyne (1972) referred to "momentary" (i.e., provoked by questions probing the subject's memory, consisting of temporal displacement of actual memories) and "fantastic" (i.e., spontaneous, grandiose) confabulation respectively. Most recently, Kopelman (1987) argued for a distinction between "spontaneous" and "provoked" confabulation, which mirrors the fantastic and momentary distinctions respectively. Although the terms *provoked* versus *spontaneous* (Kopelman, 1987) have gained acceptance recently, the general distinction remains similar to those first conceptualized by Kraepelin. The argument as to whether confabulation reflects a continuum of severity versus distinct subtypes remains a central issue even today.

While the dichotomous distinctions between a minor and major form have survived, the various terms and definitions in the literature have contributed to the confusion regarding confabulation. A major issue to address is whether provoked and spontaneous confabulations represent two distinct forms with different neuropathological mechanisms or if each is an extreme on a continuum from a single underlying mechanism.

Support for the contention that spontaneous and provoked confabulation represent extremes of a continuum was presented by DeLuca and Cicerone (1991). As described above, these authors examined confabulation first when ACoA patients were disoriented and again when they regained orientation to person, place, and time. They showed that as patients progressed from the disoriented to oriented state, confabulation in the same subject changed from spontaneous to provoked in nature. Since these were the same subjects over the two orientation conditions, differences between types of confabulation were unlikely to be due to differences in lesion location. Also, both spontaneous and provoked forms of confabulation were often observed in the same patient during either orientation condition, suggesting that different lesions were not required to be present with either of the two forms of confabulation. Based on these data, DeLuca and Cicerone (1991) concluded that confabulation fol-

lowing ACoA aneurysm may represent differences in degree and not kind.

Others have supported the hypothesis that forms of confabulation represent different degrees of a common disorder (Kapur and Coughlan, 1980; Shapiro et al., 1981; Dalla Barba, 1993; Fischer, Alexander, D'Esposito, and Otto, 1995). Several authors have noted that a more substantial degree of frontal lobe pathology is required to manifest spontaneous versus provoked confabulation. For instance, Kapur and Coughlan (1980) reported on an ACoA case whose confabulation changed from fantastic (i.e., spontaneous) to momentary (i.e., provoked) over several months, with this change paralleled by improvements on tests of "frontal lobe functioning."

In contrast, several authors have suggested that the two forms of confabulation reflect different underlying pathology. Berlyne (1972) concluded that "fantastic confabulation seems to be a distinct entity having nothing in common with momentary confabulation . . ." (p. 33). Schnider, von Daniken, and Gutbrod (1996) reported a double dissociation between spontaneous and provoked confabulation. These authors classified patients as either spontaneous (defined as acting-out self-generated confabulations) or provoked (defined as emitting intrusions on a verbal list learning task). They reported that provoked but not spontaneous confabulation was correlated with performance on measures of verbal learning and verbal fluency. In contrast, spontaneous but not provoked confabulation was associated with difficulties in temporal order processing on a continuous recognition task. Based on this double dissociation, the authors concluded that the two forms represent different disorders rather than different degrees of the same disorder. One significant limitation with this study is how spontaneous and provoked confabulation was operationally defined. While few would argue that acting-out confabulations more likely reflect spontaneous confabulations, equating intrusions during list learning performance with confabulation is problematic. This is primarily because intrusion errors on such instruments are not uncommon among a broad spectrum of neurologic populations, most of whom do not confabulate.

Kopelman (1987) examined immediate and 45-minute delayed story recall in Korsakoff and Alzheimer patients as well as healthy controls, examining provoked confabulation (defined as intrusions on story recall). Additionally, healthy subjects were also asked to recall the stories one week after learning. The results demonstrated evidence of provoked confabulation in both the Korsakoff and Alzheimer groups at the immediate and 45-minute delay intervals. In contrast, healthy control subjects produced provoked confabulations only at the one-week interval. However, the intrusions and distortions observed by the healthy group at one week resembled that observed for the two clinical groups during immediate and 45-minute delays. Kopelman (1987) concluded that there are indeed two types of confabulation. Spontaneous confabulation results from superimposing a "frontal" dysexecutive dysfunction on an organic amnesia. In contrast, although common in some amnesic patients, provoked confabulation "resembles the errors produced by healthy subjects at prolonged intervals, and may represent a normal response to a faulty memory" (p. 1436).

### *Attempts at Operational Definitions of Confabulation*

One common theme apparent among most of the attempts to differentiate among types of confabulation is the notion of less versus more severe confabulation. These have taken the form, for instance, of contrasting nonconfabulators with low versus high confabulators (Cunningham, Pliskin, Cassisi, Tsang, and Rao, 1997). Some have defined confabulation operationally as intrusions on verbal list learning or prose recall tests (Mercer, Wapner, Gardner, and Benson, 1977), with some referring to this form of confabulation as "provoked," and contrasting them (spontaneous) with those who commit overt behavior signs of confabulatory behavior such as acting out one's confabulation (Schnider, von Daniken, and Gutbrod, 1996). Such distinctions are based on the hypothesis that severity of confabulation represents different forms of confabulation, each with a different mechanism. The need for such operational definitions stems, in large part, from the lack of a universally accepted definition of confabulation and its various components. Until some consensus on such issues is accepted, attempts to fully understand the behavioral, cognitive, and neurologic mechanisms of confabulation will remain hindered.

In summary, the dichotomy between differences in confabulation that reflect differences in severity versus distinct forms, first established at the turn of the nineteenth century by Korsakoff and Kraepelin respectively, remain with us today. There is no definitive support for either hypothesis at the turn of the twentieth century. This work remains burdened by the lack of a clear definition and conceptualization of what confabulation truly is.

## Neurobehavioral Models of Confabulation

The underlying neurobehavioral mechanisms responsible for confabulation have been of interest since confabulation was first identified by Korsakoff. In general, three neurobehavioral models of confabulation have been proposed. These are: the memory impairment model, which stresses the importance of impaired memory in confabulation; the executive impairment model, which identifies frontal/executive dysfunction as the key element responsible for confabulation; and the combined memory and executive dysfunction model, which states that confabulation is observed only in the presence of both a significant memory disorder and frontal/executive dysfunction. Each model is discussed in turn.

The memory impairment model stresses the need for amnesia in order for confabulation to be present (Talland, 1965; Talland, Sweet, and Ballantine, 1967). The association of confabulation with impaired memory or amnesia has long been recognized. However, both Korsakoff and Kraepelin recognized that, although associated with memory impairment, defective memory alone could not account for confabulation. Perhaps the most compelling evidence that impaired memory or amnesia alone is not sufficient to cause confabulation is the fact that not all amnesic patients confabulate. This lack of confabulation among amnesics is particularly true among mesial–temporal amnesics (Parkin, 1984; Moscovitch and Melo, 1997). Additional evidence against the impaired memory model of confabulation is that confabulation clears in most patients (e.g., ACoA) over a period of weeks or months, yet no change is observed in the memory disorder (e.g., Kapur and Coughlan, 1980; Benson et al., 1996). Further, there are several papers that show that lesions restricted to the basal forebrain, resulting in impaired memory, do not produce spontaneous confabulation (Berti, Arienta, and Papagno, 1990; Morris, Bowers, Chatterjee, and Heilman, 1993; Abe, Inokawa, Kashiwagi, and Yanagihara, 1998). For instance, discrete lesions of the septal nuclei alone may result in amnesia, but without confabulation (von Cramon, Markowitsch, and Schuri, 1993; Berti, Arienta, and Papagno, 1999). Fischer et al. (1995) showed that ACoA patients with basal forebrain lesions can show provoked confabulation. However, it is unclear whether such responses were more a function of a confusional state (DeLuca and Cicerone, 1991).

The second model hypothesizes that confabulation is a consequence solely of executive dysfunction, resulting from disinhibition, lack of self-monitoring, and decreased awareness (Kapur and Coughlan, 1980; Joseph, 1986; Johnson, Hashtroudi, and Lindsay, 1993; Benson et al., 1996). The notion that the frontal lobes are necessary for confabulation is now well established. Several studies have shown convincingly that the ventromedial region of the frontal lobes is critical for spontaneous confabulation (Vikki, 1985; Fischer et al., 1995; Schnider, von Daniken, and Gutbrod, 1996; Moscovitch and Mello, 1997; Ptak and Schnider, 1999). Evidence for the executive model has come from studies showing that confabulation diminishes as performance improves on measures of executive functioning (e.g., Kapur and Coughlan, 1980; Papagno and Baddeley, 1997). In addition, confabulation has been shown to be associated with decreased perfusion of the orbitofrontal cortex bilaterally, with improvements in confabulation related to increased frontal lobe perfusion (Mentis, Weinstein, Murphy, McIntosh, and Pietrini, 1995; Benson et al., 1996). Importantly, while improvements in frontal/executive measures are associated with diminished confabulation, the severity of amnesia remains unchanged.

A major problem for the executive dysfunction hypothesis of confabulation is that not all patients with frontal/executive dysfunction confabulate. For instance, Vikki (1985) showed that only ACoA amnesics with frontal lesions were confabulators.

However, the executive model of confabulation also suggests that a memory disorder is not required for confabulation to be manifested (Johnson, 1991). The model contends that executive dysfunction alone is sufficient to result in confabulation. In general, there are few studies that support this claim, and those that do are problematic. For instance, several studies have cited Kapur and Coughlan's (1980) case report in support of the notion that a memory disorder is not required for confabulation. Kapur and Coughlan (1980) state that confabulation is not "dependent upon global amnesia." Similarly, Papagno and Baddeley (1997) state that confabulation can occur without "unequivocal evidence of amnesia." However, close examination of both of these reports indeed reveal significantly compromised memory performance in these patients, not on immediate recall, but on delayed recall. For instance, on the only test provided by Papagno and Baddeley examining delayed recall, patient MM could not recall anything about the story, despite immediate recall close to normal limits. The exact same finding of significantly impaired delayed recall was observed in the case by Kapur and Coughlan (1980). Many ACoA patients show relatively intact immediate recall but significantly compromised delayed recall (c.f., De-

Luca and Diamond, 1995). As such, a look at the few cases in the literature that claim that impaired memory is not required for confabulation provide weak evidence, if any, against the dual-lesion hypothesis of confabulation (both impaired memory and executive functions are necessary, see below).

Most authors who contend that impaired memory is not necessary for confabulation cite the work of Whitty and Lewin (1957, 1960) in support of this claim. Whitty and Lewin reported several cases of confabulation following anterior cingulectomy for treatment of severe obsessional neurosis. The confabulation identified by Whitty and Lewin (1957, 1960) was transient, lasting 24 hours to 3 days, and took place while subjects were disoriented to time. One feature emphasized by these authors was the vivid dreamlike experiences of these patients. However, such experiences are not uncommon in acute confusional states (Lipowski, 1990). Impaired memory and disorientation are other features associated with an acute confusional state (Lipowski, 1990). The case examples provided by Whitty and Lewin include examples of impaired memory and disorientation, a point clearly acknowledged by Whitty and Lewin (1960). As such, it is more likely that what was observed by Whitty and Lewin (1957, 1960) was more a reflection of an acute confusional state, rather than confabulation from the primary cerebral structures that underlie a more general phenomenon. DeLuca and Cicerone (1991) showed that while confabulation was not uncommon in patients who are disoriented, only ACoA patients continued to confabulate with the return of orientation, suggesting a different neuropathological substrate in the ACoA patients compared to a heterogeneous group of hemorrhagic stroke patients. Therefore, citations of Whitty and Lewin's patients as evidence against the importance of memory in confabulation are relatively weak.

For several reasons, it is important to be cautious in citing work from so long ago as definitive. First, if the anterior cingulate is critical for confabulation, why hasn't more recent work confirmed this very early relationship? Second, relative to today's standards, neurosurgical techniques were relatively crude five decades ago. As such, precise lesion characterizations in these patients could not be made with the precision offered today. Lastly, anterior cingulate involvement is often observed in patients with ACoA aneurysm (DeLuca and Diamond, 1995; Johnson, Hayes, D'Esposito, and Raye, in press). Despite decades of ACoA research, few have specifically implicated the anterior cingulate alone as critical for confabulation. Thus, while the anterior cingulate area may play a role in confabulation, it is also possible that due to its proximity to regions that have been shown by ACoA patients to be critical for confabulation (e.g., basal forebrain and ventromedial frontal lobes, see discussion below), the anterior cingulate may have little or no direct role in confabulation.

The third and most recent model is that confabulation (particularly spontaneous confabulation) requires both amnesia and executive dysfunction (dual-lesion hypothesis) in order to be expressed (Stuss, Alexander, Lieberman, and Levine, 1978; DeLuca, 1993; DeLuca and Diamond, 1995; Fischer et al., 1995; Schnider, von Daniken, and Gutbrod, 1996; Moscovitch and Mello, 1997; Wheatly and McGrath, 1997; Cunningham, et al., 1997; Ptak and Schnider, 1999). DeLuca (1993) compared a group of amnesic ACoA patients with a group of nonamnesic ACoA patients, all of whom demonstrated psychometric and neuroimaging evidence of frontal/executive dysfunction. DeLuca found that only the amnesic ACoA group confabulated, which supports the dual-lesion hypothesis of spontaneous confabulation. Numerous other authors have also supported this dual-lesion hypothesis that requires both executive and memory impairment for the expression of confabulation.

Cunningham et al. (1997) showed that "confabulation results from any general neurologic disturbance that produces defects in memory and executive function, rather than a specific pathology or neurologic disorder" (p. 875). Wheatly and McCarthy (1997) showed that while the basal forebrain may be the critical site of memory impairment in ACoA patients, what is critical for confabulation is impaired memory, not impaired basal forebrain. As such, they presented a case of confabulation in a non-ACoA patient with executive dysfunction and impaired memory secondary to a diencephalic lesion. Kopelman (1987) showed that spontaneous confabulation results from superimposing a "frontal" dysexecutive dysfunction on an organic amnesia, while provoked confabulation was a "normal response to a faulty memory."

Ptak and Schnider (1999) reported amnesia and confabulation in an ACoA patient with bilateral orbitofrontal lesions with extensive bilateral basal forebrain damage, which included the septal region. Ptak and Schnider (1996) suggest that the orbitofrontal cortex is the core structure in the maintenance of temporal order in memory by "distinguishing between presently ongoing and previously encountered information." Moscovitch and Melo (1997) showed that the degree of memory impairment did not differ between confab-

ulating and nonconfabulating amnesics, indicating that amnesia is not sufficient, but may be necessary for confabulation to be observed. Interestingly, these authors found that when similar retrieval demands are made for both episodic and semantic memory (i.e., typically everyday demands for semantic retrieval are easier to fulfill than episodic demands), confabulation was observed among both episodic and semantic memories. These data do not support the prevailing view that confabulation involves distortions primarily of episodic but not semantic memory (Dalla Barba, 1993).

Fischer et al. (1995) and DeLuca and Cicerone (1991) reported an association between confabulation and executive dysfunction. Fischer et al. (1995) divided nine acute ACoA patients into two groups, "spontaneous" confabulators and "provoked" confabulators. The authors concluded that the type and severity of confabulation noted is largely dependent upon both impairments in memory and the extent of executive system compromise. They suggested that "spontaneous" confabulation required disruption of both the basal forebrain and frontal systems, supporting the dual-lesion hypothesis. More restricted lesions to either the basal forebrain or orbital frontal structures can result in "provoked" or transient confabulatory responses. However, Schnider, von Daniken, and Gutbrod (1996) found that executive problems did not differentiate between spontaneous and nonconfabulating amnesics. Some have suggested that confabulation was associated more with lesions of the right hemisphere (e.g., Joseph, 1986). However, an extensive review by Johnson et al. (in press) found no evidence for this contention.

In summary, it appears that the dual-lesion hypothesis, which states that memory confabulation requires both a lesion resulting in significant amnesia (i.e., to either basal forebrain, diencephalic or mesial temporal structures) as well as damage to the frontal–executive system, provides the most convincing explanation for confabulation. The ventromedial portion of the frontal lobes (i.e., in the distribution of the anterior cerebral artery) appears to be the key structure within the frontal lobes.

### *Cognitive Explanations of Confabulation*

Numerous authors have concluded that poor self-monitoring is a primary element in the expression of confabulation (Johnson et al., 1993; Benson et al., 1996; Schnider, von Daniken, and Gutbrod, 1996; Papagno and Baddeley, 1997; Moscovitch and Melo, 1997; Ptak and Schnider, 1999; Johnson et al., in press). However, Ptak and Schnider cautioned that "poor self-monitoring . . . may serve as a descriptive explanation" and Schnider, von Daniken, and Gutbrod (1996) continue that such an executive dysfunction "does not disclose the specific mechanism of confabulations." However, recent work has shed some light on the cognitive mechanisms that may be responsible for confabulation.

Moscovitch and Melo (1997) note that there is general agreement that confabulation is more of a problem in retrieval rather than encoding, consolidation, or storage. These authors postulate that it is impaired strategic retrieval (i.e., an active, self-initiated, goal-directed, and an effortful systematic memory search) that is primarily responsible for confabulation due to damage to ventromedial frontal structures, as opposed to associative retrieval (passive, automatic search through memory). As mentioned above, several investigators suggest that spontaneous confabulation is a result of a difficulty in recognizing the temporal order of events during the retrieval of stored information. Moscovitch and Melo (1997) suggest that defective temporal order processing is not the cause of confabulation, but a symptom of a more fundamental problem in the strategic retrieval of the memory trace and defective self-monitoring of this trace. Specifically, Moscovitch and Melo (1997) state that the key deficit that results in confabulation is not simply the severity of the impaired strategic search at input (i.e., searching for the memory trace), but an impairment in the monitoring at output (i.e., monitoring what was retrieved). Hence, confabulation occurs when the outcome of a disturbed strategic search is faulty and a response is emitted without proper monitoring, evaluation, and verification of the recovered memory trace (Rapcsak, Kaszniak, Reminger, Glisky, Glisky, and Comer, 1998).

Johnson and colleagues (Johnson, 1991; Johnson, O'Connor, and Cantor, 1997; Johnson et al., in press) suggest that different types of confabulation would result from different combinations of cognitive difficulties. Johnson discusses confabulation within the context of difficulties in reality monitoring or source monitoring. For instance, confabulation could result from difficulties in the encoding of information. Yet, confabulation may also result from poor retrieval, or reduced motivation. Johnson et al. (1997) concluded that confabulation in an ACoA case study (patient GS) was based on a confluence of factors including: (1) deficits in the systematic retrieval of autobiographical

information; (2) source monitoring deficit; and (3) a propensity toward detailed imaginations. Johnson et al. (1997) conclude that what specific combination of cognitive deficits are observed in a particular patient will dictate the type and severity of confabulation observed.

In summary, cognitive theories on the mechanism of confabulation stress impaired self-monitoring and impaired retrieval mechanisms (i.e., strategic retrieval) as critical elements responsible for confabulation.

## Confabulation and Awareness

The relationship between confabulation and disturbed awareness has long been recognized. For instance, several studies have shown that confabulation decreases as awareness increases (Williams and Rupp, 1938, cited in McGlynn and Schacter, 1989; Wyke and Warrington, 1960; Mercer et al., 1977; Stuss et al., 1978; Shapiro et al., 1981; DeLuca, 1992). Benson et al. (1996) believe that confabulation and defective self-awareness represent the same basic functional disorder (p. 1243). Other evidence to support this contention comes from work showing that confabulation diminishes as performance on tests of frontal/executive improves (Papagno and Baddeley, 1997; Kapur and Coughlan, 1980), and that confabulation diminishes with improvements in frontal lobe perfusion on SPECT scans of the brain (Benson et al., 1996). However, unawareness syndromes do not always lead to confabulation. As described above, a major problem with confabulation is its broad-based definition, which often confuses the picture rather than providing clarity. As such, many have associated responses resulting from deficits of unawareness with confabulation. After a brief introduction to anosognosia and denial, a brief description is provided of unawareness syndromes that have been referred to as confabulation in the classical literature.

The syndrome of unawareness following brain damage is called *anosognosia.* It was first described by von Monakow (1885) and Anton (1896). The indifference that is sometimes associated with anosognosia has been called anosodiphoria (Heilman, 1991). Anosognosia is seen in many behavioral syndromes following brain damage, including Wernicke's aphasia, Anton's syndrome, and left hemiplegia (see Heilman, 1991, for a brief review), each of which is described briefly below.

First, however, anosognosia needs to be differentiated from "denial of illness" (c.f., Prigatano and Schacter, 1991; Prigatano and Klonoff, 1998). Psychiatric definitions of "denial" have been described more as altered self-awareness rather than unawareness. The American Psychiatric Association (1994) defines denial as a defense mechanism where the "individual deals with emotional conflict or internal or external stressors by *refusing to acknowledge* some painful aspect of external reality or subjective experience that would be apparent to others" (p. 755). Prigatano and Klonoff (1998) explain that "denial after brain injury . . . reflects the individual's attempt to use previous coping strategies to deal with impairments that are only partially recognized" (p. 57). Using these definitions, "gap filling" in confabulation could be considered denial while displaced temporal context, or the lack of information access due to a disconnection syndrome (e.g., the Interpreter) could reflect an anosognosia, or unawareness of knowledge (Schacter, 1991).

Wernicke's aphasia is a well-known language disorder in which patients produce fluent speech marked by neologism and semantic and paraphasic errors. These errors have been viewed as confabulations. While comprehension is severely compromised, such aphasics cannot always express themselves. Importantly, these patients are unaware that the person with whom they are speaking does not understand them. It has been suggested that the anosognosia associated with Wernicke's aphasia is one of defective monitoring. Such patients have lost their neural representation of word sounds and are then unable to match their speech output with an intact representation or template. This lack of internal feedback results in the anosognosia (Heilman, 1991).

Despite the inability to demonstrate sight, patients with Anton's syndrome "deny" cortical blindness (Heilman, 1991). They will confabulate responses (e.g., provide a response when asked how many fingers they can see during confrontation) and will make excuses when confronted with their errors ("I'm not wearing my glasses"). While several attempts have been made to explain this phenomenon, it is likely that the explanation for Anton's syndrome is similar to that of Wernicke's aphasia: impaired monitoring. Some have hypothesized that the visual association cortex becomes disconnected from the speech–language areas, leading to decreased monitoring and confabulation. Others have hypothesized that such patients continue to receive input via the "second visual system"; a pathway mediated via the superior

colliculus. This may result in input to a visual monitor or speech–language region that may be disconnected from the geniculocalcerine system (the primary visual system). While impaired memory is usually associated with Anton's syndrome, its significance is not clear.

Patients with right hemisphere lesions (usually involving the parietal and frontal regions) often display left-sided hemiplegia, but also may "deny" that anything is wrong with their left side (e.g., denying that their own hand is theirs, confabulating a response when confronted). Many theories have been posited to explain the unawareness, indifference, and confabulation associated with this behavioral syndrome, ranging from premorbid personality features (Weinstein and Kahn, 1955, 1996) to disconnection syndromes and impaired monitoring of somatosensory input (Heilman, 1991).

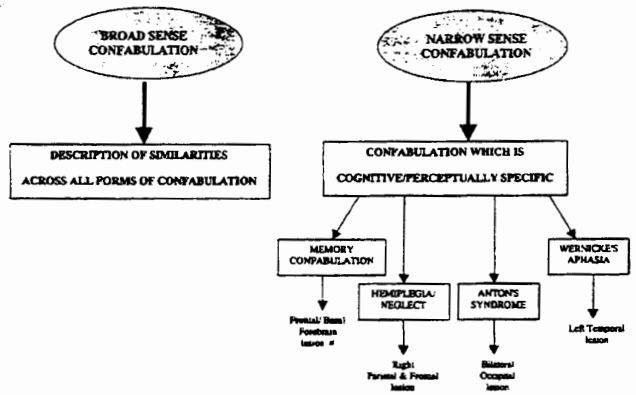
One important feature that is different between these three neurobehavioral syndromes that lead to confabulation, versus those often evident from ACoA aneurysm or Korsakoff's syndrome, is the depth of the confabulation itself. While ACoA confabulation can be very elaborate and lengthy, confabulation associated with Wernicke's aphasia, Anton's syndrome, and anosognosia for hemiplegia is usually targeted (i.e., on the left-hemiplegia or blindness) and limited in content.

An aspect that appears common to all of these clinical entities is an unawareness syndrome (anosognosia). This unawareness may result from different cerebral mechanisms, but the behavioral effect is the same. For instance, with the Interpreter described above, the information within the right hemisphere is not available to the left hemisphere in split-brain patients (i.e., disconnection syndrome) resulting in confabulation from an intelligent verbal hemisphere trying to make logical sense of cues in the environment. However, this mechanism is different from, for instance, the compromised neural representation of word sounds that prevents Wernicke's aphasics from being aware of their own jargon speech. Lastly, memory confabulators (e.g., ACoA patients) can produce a long verbal discourse about seemingly inaccurate events, not being able to self-monitor the temporal and often illogical relationships within their own statements. While the specific behavioral feature of these examples differ, the anosognosia associated with the behavior is the common denominator.

## A Proposed Model of Confabulation

Are neologisms truly confabulations? Are examples of "denying" cortical blindness, where the patient attempts to make seemingly logical inferences (e.g., "I don't have my glasses"), the same as profuse, spontaneous, and fantastic verbal confabulation? At some level of argument, one can conceptualize these broadly as confabulations. At another level, however, they are indeed different. This lack of a clear understanding burdens a greater conceptual understanding of confabulation. What is needed is a model of confabulation that outlines not only the similarities, but also the differences among these various examples of confabulation.

Given the various general forms of cognitive/perceptual distortions that have been referred to as confabulation, can any systematic understanding of confabulation be made? The answer must be "yes," in order to move our understanding of confabulation beyond what was generally conceptualized at the end of the nineteenth century. A proposed model of confabulation is illustrated in Figure 1. First, it is proposed that two definitions of confabulation are needed: *Broad sense confabulation,* which can be defined in a more broad or all encompassing sense, and *Narrow sense confabulation,* which is more perceptually specific. Broad sense confabulation simply refers to the general conceptualization of distortion; that is, what is common across various forms of confabulation is that information is perceptually distorted (e.g., one can "see" with Anton's syndrome, one can use one's left extremities despite hemiplegia, one can displace actual memories temporally). As such, understanding broad sense confabulation means focusing on features that are similar across the various manifestations of confabulatory behavior. It is simply descriptive in nature. It is unlikely that broad sense confabulation will have an anatomic locus, nor should




[a]Lesions resulting in memory disorder may include basal forebrain, diencephalic, or mesial temporal structures

**Figure 1.** Broad and Narrow Sense Confabulation

one be forced upon it. Broad sense confabulation can thus be defined as follows: *statements or actions that involve apparently unintentional but obvious distortions.*

Narrow sense confabulation involves the specific cognitive–perceptual system that is being confabulated. Narrow sense confabulation can be viewed as confabulation that is a manifestation of damage to a specific cognitive–perceptual mechanism that is impaired, each with its own identifiable lesion (see Figure 1).

Because confabulation is not a unitary behavioral construct, the need for narrow sense confabulation becomes critical once one begins to discuss mechanism or neuropathology. For instance, the hypothesis of impaired strategic retrieval for memory confabulation may not apply to explanations of confabulation associated with left hemiplegia. Narrow sense confabulation allows for a discussion of specific mechanisms responsible for a particular form of confabulation, without having to provide a more global explanation for all forms of confabulation.

While the dichotomy between broad and narrow sense confabulation may seem simplistic and "obvious" to researchers and students of confabulation, the inappropriate use of the term in the literature and medical textbooks cited throughout this paper speaks volumes for the need for a more refined terminology when confabulation is discussed.

## Conclusions

In many ways, much has been learned about confabulation during the past century. However, in other very significant ways we have progressed little from what was known 100 years ago. Korsakoff believed that memory confabulation consisted of actual events displaced temporally of which patients were unaware. In contrast, Bonhoffer (1901) felt that confabulations were an active attempt to cover up a disorder of which the patient was consciously aware. Despite the lack of empirical support for the active gap filling hypothesis, these two opposing viewpoints remain prominent in today's literature and thinking. Little progress has been made in 100 years of research in identifying whether memory confabulation represents a single entity, differing only in degree, as hypothesized by Korsakoff, versus having two (or more) distinct forms of confabulation, each with its own neuropathologic mechanism, as initially outlined by Kraepelin. It is believed that much of the difficulty in understanding factors such as these stems from the lack of a universally accepted definition or model of confabulation. It is hoped that the model proposed in the present paper can serve as a starting point, eventually resolving into an accepted conceptual framework from which to study confabulation.

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