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CHAPTER 16

Confabulation

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Confabulation

Confabulation is a symptom observed in cases of brain damage and in some psychiatric disorders (e.g., schizophrenia). Various definitions have been given in the literature (e.g., Berlyne, 1972; Talland, 1961), but they all have in common the idea that patients sometimes make statements that are false or engage in behaviors that reflect false memories or beliefs, and that they do this with no deliberate intention to deceive. For example, in a now classic paper, Stuss, Alexander, Lieberman and Levine (1978) described five confabulating patients, two who had had aneurysms of the anterior communicating artery (ACOA), a multiple infarct patient, and two trauma patients. One of the trauma patients sometimes described his injury accurately, but at other times fabricated a story about a drowning accident in which his head was injured as he attempted to rescue one of his children. Another often cited ACOA case was described by Kapur and Coughlin (1980). The patient was a 48 year old man who was disinhibited and confabulated. For example, he would sometimes claim to have been engaged in business appointments during the previous hours when in fact he had been undergoing psychological tests. Damasio, Graff-Radford, Eslinger et al. (1985) reported a 32 year old patient who had had an aneurysm of the anterior cerebral artery (ACA) who claimed to have been a space pirate and believed that Egyptian President Sadat had visited him in the hospital. Dalla Barba, Mantovan, Cappelletti and Denes (1998) examined a 57 year old woman who had had a heart arrest. When asked her age during one interview, she responded '20' and when asked to look in a mirror, said 'Well, I look 50 years old – but it's because I was sick. I fell in a ditch while I was playing with my bothers. That's why I damaged my face.' Here we review studies of such patients and consider confabulation in the context of theoretical accounts of normal memory distortion.

Within cognitive psychology, there is a tradition of studying *accurate* memory (including errors of omission), dating from Ebbinghaus's (1885) foundational work, and a tradition of studying memory *distortions* (errors of commission) that goes back at least to Bartlett (1932) and the Gestalt psychologists (see Riley, 1962 for a review). In neuropsychology, there are traditions of studying *deficits in accurate* memory (errors of omission) that includes Milner's (1966) classic studies of the amnesic patient HM, and a tradition of studying *abnormally frequent or flagrant memory distortions* that goes back to Korsakoff's (1889/1996; Banks, 1996) observations of confabulation in alcoholic patients. Although articles in these various traditions do not always refer to each other as much as one might expect, they offer different views of the same 'elephant'. Together they provide a rich description of

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the types of cognitive processes that account for both true and false memories (e.g., Burgess and Shallice, 1996a; Johnson and Raye, 1981, 1998; Mitchell and Johnson, 2000; Schacter, Norman and Koutstaal, 1998; Roediger, 1996). Modern functional neuroimaging techniques provide powerful new tools to explore more fully the relations among cognitive processes and brain structures that have been suggested by findings from cognitive psychology labs and neuropsychological studies of patients. We now have the opportunity to make observations of memory processes in action in normal and brain damaged individuals. As methodological advances continue, we can expect our future understanding of normal and impaired memory to become both more complex theoretically and increasingly concrete – as component processes engaged in various contexts are dynamically correlated in time with spatially identified activation.

Realizing this future depends to some extent on a clear idea of where we are now. Hence, this chapter discusses cognitive processes involved in identifying the origin of memories (source monitoring), and the evidence from confabulating patients that provides information about some of the brain structures that underlie normal source monitoring processes. In this review, we restricted our literature search to articles and chapters published in English. We included both case studies and group studies and recorded, where available, etiology, type and examples of confabulation, area of brain damage, and type of neuropsychological and experimental tests administered. When a case was discussed in more than one article, any additional information about it was added to the data base but the case was included only once. Here we will report only cases in which some information was provided about etiology or damaged brain regions. We also discuss data from functional neuroimaging studies that have begun to explore source monitoring.

As a theoretical context, we use the source monitoring framework (e.g., Johnson and Raye, 1981; Johnson, 1988; Johnson, Hashtroudi and Lindsay, 1993). Similar ideas are expressed in other models dealing with memory distortion (e.g., Burgess and Shallice, 1996; Moscovitch, 1989, 1995; Schacter et al., 1998). When useful, we also draw upon the more specific component processes of the MEM model (Johnson, 1992). This model provides a mid-level architecture of the subprocesses involved in perception and reflection (Johnson, 1997). It includes processes that underlie working memory (WM) and executive functions (e.g., Baddeley, 1986) and that underlie long-term memory (LTM) encoding and retrieval (e.g., Bower, 1970; Craik and Lockhart, 1972; Tulving, 1983). The MEM model proposes that WM and LTM tasks recruit from a common set of component processes and thus provides a context for characterizing both the similarities and differences in cognitive requirements across WM and LTM tasks.

Theoretical context: source monitoring in neurologically intact individuals

Brain damage does not make a perfect system imperfect. It makes an imperfect system worse. Thus, in a way, brain damage can serve to magnify normal experience. An amnesic is us when we cannot remember and know we can't. A confabulating patient is us when we do not remember accurately and don't know we don't. That is, confabulators, like all of us on occasion, assume a mental experience is an accurate memory or belief when in fact it does not reflect past or current reality. Of course, confabulating patients are much more likely to take whatever comes to mind for memories, and the misattributions they make can be quite bizarre. Thus, at first blush, it may seem easier to see ourselves in the amnesic than in the confabulator, but that may be because we have had many more conscious experiences of forgetting than of misremembering. That is, we more often are aware of our errors of omission than our errors of commission.

Understanding the relation between normal memory distortion and abnormal memory distortion will shed light on each. Cunningham, Pliskin, Cassisi et al. (1997) noted 'the precise mechanisms

involved in the production of confabulation have yet to be adequately characterized' (p. 868). At the same time, the *cognitive* mechanisms involved in normal memory distortion are quite well characterized and, although there is much to learn about the details of their operation and how they combine in various situations, they are, in a general way, understood (e.g., Jacoby and Kelley, 1992; Johnson, 1997; Loftus, 1997; Mitchell and Johnson, 2000; Ross, 1989, 1997). What has not been worked out in detail is the relation between these cognitive mechanisms and specific brain mechanisms.

In considering how progress might be made on this front, consider first the processes involved in normal memory. What information do we ordinarily use to attribute a mental experience to memory? What encoding processes determine whether such information will be available when we need it later? What ordinarily signals us to wonder whether what we take as memories are accurate? What keeps certainty related to accuracy? What processes can be engaged to further evaluate the source of mental experiences and constrain the amount of distortion that occurs? Such questions have been addressed in many studies investigating memory distortions (for reviews see, e.g., Johnson et al., 1993; Mitchell and Johnson, in press; Roediger, 1996). An overview of the processes that produce both true and false memories is provided by the source monitoring framework (SMF, Johnson et al., 1993; Johnson and Raye, 1981; 1998; Mitchell and Johnson, 2000).

According to the SMF, memory records the activities of perceptual and reflective processes (Johnson, 1992). Accurate event (episodic) memories require these processes to have bound features (e.g., color, object, location, person, semantic detail, emotions, etc.) of experience together so that features can cue each other later. When subsequent mental experiences have the qualities of bound feature information (e.g., they are rich in temporal, perceptual and spatial detail, and/or emotional qualities), they will tend to be attributed to past, specific events (e.g., Brewer, 1992; Johnson, et al., 1988; Lindsay, 1993). When they are not rich in these qualities we are more likely to question them and more likely to try to access additional information. Encoding, revival and attribution of information is also influenced by prior knowledge, schemas, stereotypes, and beliefs (e.g., Bartlett, 1932; Bransford and Johnson, 1973; Mather, Johnson and De Leonardis, 1999), all of which are influenced by social/cultural factors (Johnson and Mather, in press). Events that fit with what we know or believe are easier to encode, easier to revive as memories, and less likely to be questioned later.

In normal cognition, encoding, revival and attribution processes are not perfect and hence omission and commission errors will occur. That is, some memory errors can be traced to imperfect initial encoding and some to imperfect reactivation, retrieval and evaluation processes. For example, being distracted or focusing on one's own emotions, thoughts, beliefs, or images at the expense of focusing on features of external events can decrease appropriate feature binding, create inaccurate representations, and increase the likelihood of later source confusion and misattribution (e.g., Johnson, Nolde and De Leonardis, 1996; Mather, Johnson and De Leonardis, 1999). Furthermore, omission and distortions at encoding can be filled in, strengthened, or embellished through rumination. At retrieval, sometimes appropriate criteria are used (i.e., the right features are considered and weighted appropriately), but still a vivid imagination can exceed the threshold and be taken to be a memory of an actual event, for example, when one believes they said or did something that they only thought or imagined saying or doing. For example, the perceptual detail in a mental experience might lead one to conclude it is a memory for a perceptual event when, in fact, the event was imagined or suggested (e.g., Zaragoza, Lane, Ackil and Chambers, 1997; Belli and Loftus, 1994).

Another possible cause of source misattributions is that an individual may use less than optimal or inappropriate evidence when more appropriate evidence could be used (Marsh and Hicks, 1998).

Distraction or impulsivity might produce source attributions based on readily available but potentially poor source information. For example, attributions about source might be made on the basis of familiarity, or semantic, or emotional features even though other specific, perceptual detail that might help identify source has been encoded. In addition, while source attributions are normally influenced by stereotypes and other knowledge and beliefs, the poorer the information of other types, the greater the potential influence of possibly misleading stereotypes, knowledge, and beliefs (Mather et al., 1999). Furthermore, motivation to be accurate may be low. The fact that people sometimes normally adopt less than optimal criteria (e.g., underweight better source diagnostic features) is demonstrated when instructions or test conditions induce them to consult better or additional information and their likelihood of false memories decreases (e.g., Lindsay and Johnson, 1989; Dodson and Johnson, 1993; Mather, Henkel and Johnson, 1997).

The SMF also proposes that processing varies in whether it is more heuristic or systematic. Heuristic processing is 'cognitively inexpensive' and is usually operating any time we are remembering, whether or not we are aware of it; more systematic processing tends to be engaged more selectively and deliberately. Heuristic processing is comprised of fewer component processes and uses readily available information (e.g., familiarity), including qualities (e.g., perceptual detail) and schemas (e.g., world knowledge, stereotypes) activated by a cue. Systematic processing tends to be comprised of more component processes and/or require the retrieval and evaluation of more specific or selective information and/or information such as other memories and knowledge that are not readily activated by the initial cue. For example, a misattribution based on a fast heuristic source decision could be caught by a systematic decision based on plausibility given other knowledge. Systematic processing is more likely to require selective attention, self-initiation of cues, shifting among representations or features of representations, and noting inconsistencies among activated information or between activated features and long-term knowledge. Conditions that might activate systematic processing include mental experiences that are near the criterion for heuristic judgments (ambiguous) and hence create uncertainty or conflict, conditions in which the stakes for accurate memory are high, and so forth.

The terms heuristic and systematic do not imply that there are two discrete sets of processes; rather they are convenient short-hand terms. Important points are that (1) the encoding, retrieval and evaluation processes involved in memory vary in cognitive complexity (i.e., the number or iterations of component processes engaged); (2) the information available and/or used in source monitoring varies in its specificity and appropriateness for the situation; (3) different combinations of component processes are selectively engaged depending on the situation, motivation, etc.; (4) heuristic and systematic processing are both sources of constraints on what will be felt to be veridical; (5) both heuristic and systematic processing are imperfect. Within the SMF, any of the above factors could contribute to memory distortion, and confabulation presumably is the result of severe disruption of the balance among mutually constraining processes. A major challenge is to work out the relation between particular types of brain damage and disruption in particular source memory processes operating at encoding, retrieval and evaluation.

One way to be more specific about the component processes that are engaged during heuristic and systematic remembering is to describe them in terms of the MEM model. Minimally, explicit source judgments require *noting* the match between activated information and the criteria being used. For example, old/new recognition requires a comparison of the familiarity of the activated information and the current familiarity criterion. If the judgment is made more difficult by increasing the similarity of targets and distractors, or by requiring more specific source information, additional processes must be recruited. For example, more specific featural information often takes longer to revive

(and become differentiated) than does familiarity (e.g., Johnson, Kounios and Reeder, 1994). In a source memory laboratory task, if enough source information is not readily available when the memory probe is initially presented, subjects must maintain activated information through *refreshing* and/or *rehearsing* until they can *note* its features (compare them to whatever criteria are being maintained and used). *Refreshing* and *rehearsing* are the types of functions included in the concept of working memory (e.g., Baddeley, 1986). The task may also require the agenda-driven *reactivation* of additional information, or the systematic *retrieval* (through conscious search efforts) of supporting or disconfirming evidence. *Reactivation* and *retrieving* are the types of processes included in accounts of long-term memory (e.g., Tulving, 1983). The *shifting* among aspects of activated information (e.g., between features of representations) or among component processes (e.g., from *noting* to *retrieving*) requires subjects to assess the state of their own mental experiences with respect to *agendas* (goals, tasks, etc.) and to *initiate* additional component processes as required. *Shifting* and *initiating* are the types of processes usually encompassed by the idea of executive function (e.g., Baddeley, 1995). Recall of complex autobiographical memories often involves maintaining an *agenda* and iterations of a number of these various processes – *refreshing* activated information, *initiating* cues, *retrieving* additional information, *noting* whether its qualitative characteristics meet expectations, *discovering* consistencies or inconsistencies with other activated knowledge and beliefs, and so forth. Models of autobiographical recall include similar concepts (e.g., Conway, 1992; Burgess and Shallice, 1996). The component process terms of MEM (e.g., *rehearsing*, *reactivating*) can be thought of as functional labels for patterns of activation in neural networks that mediate transactions among brain regions (e.g., a frontal-parietal circuit for *rehearsing*, see Jonides, Schumacher, Smith et al., 1998).

Source monitoring processes will yield occasional errors even under the best of circumstances and when people are fully attending to the issue of the origin of their mental experiences. More errors will occur when source monitoring is incidental to another task which is more central to the person (e.g., telling an autobiographical memory on a social occasion; generating novel instances in a category generation task, e.g., Brown and Murphy, 1989). And even more errors will be made under conditions of distraction, stress, time pressure or other factors that further disrupt or attenuate the recruitment of specific component processes or affect the information used in making source attributions (e.g., Dodson and Johnson, 1996; Zaragoza et al., 1997). Similarly, in considering how such a complex system for remembering might break down in more profound ways from brain damage, we would expect that damage to the brain regions that are associated with working memory, long-term memory and executive function would compromise a patient's ability to identify the origin of mental experiences because these various tasks recruit from the same underlying set of component processes. However, damage to different areas may disrupt source monitoring in different ways. Next we examine the literature on confabulation with these ideas in mind.

Confabulation in brain damaged patients

Searches of PsycInfo (1887–May 1999) and Medline (1966–May 1999) turned up 389 articles and chapters with 'confab' in the title or abstract. We also included relevant cases that we were aware of but that did not turn up in the computer search (e.g., cases mentioned in articles that were not primarily concerned with confabulation, e.g., Whitty, 1956). Since our primary interest was in cases of presumed or documented focal brain damage where confabulation was observed, we excluded patients with psychiatric disorders, epilepsy and dementia. Also, certain subtypes of confabulation likely to involve specific deficits in perceptual processing (Capgras syndrome, reduplicative paramnesia, Anton's syndrome and confabulation associated with anosognosia for hemiplegia) were

excluded. Of course, damage often is in more than one area and some patients may have both focal and diffuse damage (e.g., Korsakoff patients). Using liberal criteria for what constitutes 'focal' damage, we identified 349 cases with enough information to classify them on factors related to our present purposes.

Etiology

Confabulations have been reported in many types of patients, but the largest number of cases of particular etiology come from cingulectomy patients, Korsakoff patients, tumor patients, and individuals who suffered rupture of an aneurysm of the ACOA, stroke, or trauma. Table 1 lists articles in which patients of various types are reported and Table 2 lists the total number of each patient type.

Damaged brain regions

Laterality and confabulation

Table 2 also shows the number of patients whose damage was primarily in the right or left hemisphere, or was bilateral. In addition, there were 224 cases where no information was provided about which hemisphere was affected; of these, 74 cases were Korsakoff patients, and 14 were ACOA patients. The general pattern shown is Table 2 is the same when only patients were included for whom arteriorgram, CT, MRI, PET, SPECT scan results were reported or who had been autopsied.[1]

There were about an equal number of cases classified as left (29) as right (32) hemisphere damage in the studies reviewed, and overall about as many bilateral (64) as unilateral (61) cases. For this purpose, medial damage, such as the cingulectomy cases, was classified as bilateral unless it was explicitly reported as unilateral. There would be a greater bilateral-unilateral disparity if the Korsakoff patients are considered, assuming it is reasonable to suppose that their damage is likely to be bilateral. ACOA patients are the group for which we

TABLE 1
List of papers reporting confabulating patients by etiology

ACOA aneurysm
Alexander and Freedman, 1984
Baddeley and Wilson, 1986
Beeckmans et al., 1998
Benson and Stuss, 1990
Dalla Barba et al., 1997a
Damasio et al., 1985
Delbecq-Derouesne, 1990
DeLuca, 1993
DeLuca and Cicerone, 1991
DeLuca and Locker, 1996
Downes and Mayes, 1995
Eslinger and Damasio, 1984
Fischer et al., 1995
Johnson, 1997
Johnson et al., 1997
Kapur and Coughlan, 1980
Moscovitch, 1989
Moscovitch and Melo, 1997
Parkin et al., 1988
Phillips et al., 1987
Schnider et al., 1996a
Schnider et al., 1999
Shapiro et al., 1981
Stuss et al., 1978
Talland et al., 1967
Vilkki, 1985

Cingulectomy
Whitty, 1956
Whitty, 1966
Whitty and Lewin, 1957
Whitty and Lewin, 1960

Korsakoff syndrome
Benson and Stuss, 1990
Benson et al., 1996
Berglund et al., 1979
Berlyne, 1972
Clarke et al., 1958
Dalla Barba et al., 1990
Davidson, 1948
Gorman et al., 1950
Hampton, 1947
Kopelman, 1987
Kopelman et al., 1997
Mercer et al., 1977
Moscovitch and Melo, 1997
Schnider et al., 1996a
Tei et al., 1995
Victor et al., 1959
Welch et al., 1997
Wyke and Warrington, 1960

Trauma
Aita, 1948
Baddeley and Wilson, 1986
Conway and Tacchi, 1996
Cunningham et al., 1997
Dalla Barba, 1993a
Malloy et al., 1993
Moscovitch and Melo, 1997
Sabhesan, 1988
Schnider et al., 1996a
Schnider et al., 1999
Shapiro et al., 1981
Stuss et al., 1978
Weinstein, 1996
Weinstein and Lyerly, 1968
Weinstein et al., 1956

Tumor
Cunningham et al., 1997
Luria et al., 1967
Morris et al., 1992
Papagno and Muggia, 1996
Schnider et al., 1996a
Schnider et al., 1999
Sprofkin and Sciarra, 1951
Whitty, 1956
Williams and Pennybacker, 1954

Stroke
Cunningham et al., 1997
Dalla Barba, 1993a
Dalla Barba, 1993b
Dalla Barba et al., 1997b
Damasio et al., 1985
DeLuca and Cicerone, 1991
Feinberg, 1994
Haut et al., 1995
Kopelman et al., 1997
LaPlane et al., 1981
Logue et al., 1968
Moscovitch and Melo, 1997
Papagno and Baddeley, 1997
Sandson et al., 1986
Schnider et al., 1996a
Schnider et al., 1996b
Shapiro et al., 1981
Stuss et al., 1978

Other
Dalla Barba, 1998
Damasio et al., 1985
Downes and Mayes, 1995
Mercer et al., 1977
Moscovitch and Melo, 1997
Shapiro et al., 1981
Thorpe, 1994
Watkins and Oppenheimer, 1962

[1] Of the 349 confabulating cases identified, 156 patients had one or more of the following: CT (100), MRI (20), PET (3), SPECT (2), EEG (24), and/or angiography or anterigrams (16), surgery resulting in known damage (27), or were examined at autopsy (9).

TABLE 2
Etiology and laterality of confabulating patients

Etiology	Left	Right	Bilateral	NS[a]	Total
ACOA	14	15	22	14	65
Cingulectomy	0	0	18	0	18
Korsakoff	0	1	0	74	75
Trauma	0	4	11	82	97
Tumor	2	1	3	12	18
Stroke	9	11	9	37[b]	66
Other	4	0	1	5	10
Grand total	29	32	64	224	349

[a] NS = not specified.
[b] Includes 16 patients who suffered an aneurysm of the ACOA or ACA.

have the largest number of cases and some specific information about region of brain damage. There were about the same number of left (14) and right (15) hemisphere cases and slightly more unilateral (29) than bilateral (22) cases. From these studies, we do not know the base rate of brain damage to each hemisphere in the population of all ACOA patients (confabulating and nonconfabulating), thus we do not know the relative proportions of right versus left hemisphere cases that yield confabulation. However, there is no strong evidence from our subpopulation of ACOA patients that confabulation is more likely from right than left hemisphere damage or vice versa and no evidence that confabulation is more likely from bilateral than unilateral damage. However, it is not possible to control for extent of damage in this analysis. Also, the numbers within any particular cell of Table 2 are quite small and thus the relation between hemispheric locus of lesion, extent of lesion, and likelihood of confabulation remains an open question (see Fischer, Alexander, D'Esposito and Otto, 1995 for an approach to these issues).

Laterality and confabulations associated with perceptual deficits

It has been suggested that confabulation is more common after right than left hemisphere damage (e.g., Belyi, 1988; Joseph, 1986). This observation is most likely to come from studies of patients whose brain damage produces some deficit in perceptual processing. For example, Belyi compared right and left frontal tumor patients on a task requiring them to look at a sequence of cartoon pictures and interpret the story depicted. Right hemisphere patients were somewhat more likely to generate stories based on a few details (suggesting they had not adequately attended to or perceived most of the pictures) whereas left hemisphere patients were somewhat more likely to mention more pictures or elements but not incorporate them into a coherent story, and were more likely to comment on the difficulty they were having. Belyi suggested that the right hemisphere patients' more embellished stories were confabulations. However, because the subjects were explicitly asked to 'tell the plot' for (i.e., interpret) the pictures, the pattern of results could reflect better ability to comply with task demands by right hemisphere patients, suggesting that an undamaged left hemisphere was more critical for interpretation (e.g., Gazzaniga, 1995), even in the face of poor perceptual information, than an undamaged right hemisphere (which might result in better visual information but poorer ability to weave it into a story). In addition, with more perceptual information, there is more information that an impaired left hemisphere can deal with and there is more detail to contradict any particular story out of a set of possible stories. With right hemisphere damage, there may be less information to contradict any particular story and thus less awareness of a difficulty in interpretation with right hemisphere damage. Because of the ambiguity about whether these represent confabulations of the sort characteristic of the patients of most interest here, these cases are not included in Tables 1–6.

In any case, there are several specific subtypes of confabulation that appear to reflect deficits in some aspect of perceptual processing. These include Capgras syndrome, reduplicative paramnesia, Anton's syndrome and confabulation associated with anosognosia for hemiplegia (e.g.,

Bisiach and Geminiani, 1991; Ellis and de Pauw, 1994). These cases often involve more posterior brain damage, sometimes in combination with frontal damage. It should be noted that whereas the overall incidence of confabulations in papers reviewed in the present chapter does not appear to be strongly right or left lateralized, right hemisphere damage appears to be more likely than left to produce confabulations related to perceptual deficits.

Particular brain structures and confabulation

Table 3 provides information about the specific brain regions damaged, without regard to hemisphere. There were 131 cases where brain region was indicated. There were an additional 103 cases where the regions of damage might be inferred, including 73 Korsakoff patients and six ACOA patients (see note, Table 3). As can be seen in Table 3, 87% of the confabulating cases where information about areas of damage was provided involved or were likely to involve damage in one or more of the following areas: anterior cingulate, basal forebrain,[2] orbitofrontal, frontal, or thalamus (see Fig. 1); 89% fall in these categories if only the patients for whom there was CT, MRI, PET, SPECT, or autopsy evidence were noted (see footnote 1). For Korsakoff patients, likely specific sites of damage are the dorso-medial thalamic nucleus and the mamillary bodies, with possibly generally reduced gray matter volumes in the anterior diencephalon structures, orbitofrontal cortex, and the medial temporal lobe (Parkin and Leng, 1993). There is considerable variation in the neuropathological outcome of ACOA aneurysms, but more frequent sites of damage, based on the *confabulating* ACOA patients represented in Table 3 for whom we have information, include the medial and orbitofrontal regions, other unspecified frontal areas, basal forebrain, and striatum (particularly the head of the caudate); other areas reported for at least one ACOA patient include polar frontal, anterior cingulate, anterior corpus callosum, anterior commissure, and frontal-parietal.[3]

TABLE 3
Region of brain damage in confabulating patients

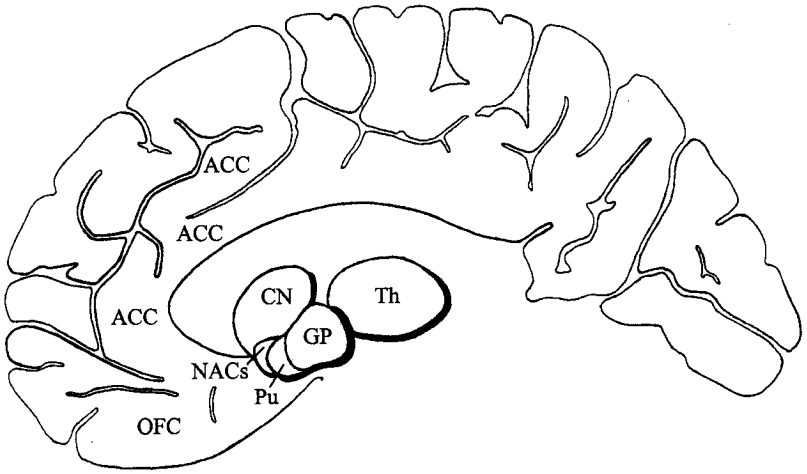
Region of brain damage	Number of confabulating patients
Anterior cingulate	18
Basal forebrain	10
Orbitofrontal	7
Basal forebrain and orbito-frontal	1
Frontal	40
Frontal and other regions	29
Thalamus	10
Internal capsule	1
Ventricle	7
Temporal	2
Parietal-temporal	2
Occipital	1
Mesencephalon	2
Parietal	1
Not specified[a]	218
Grand total	349

[a] The 'Not specified' category includes 73 Korsakoff patients, six ACOA patients, one ACA patient, 22 ACA or ACOA aneurysm patients, and one carbon monoxide poisoning patient.

Korsakoff and ACOA patients have provided much of the evidence about confabulation (e.g., Kopelman, 1987; DeLuca and Cicerone, 1991). However, useful information comes from other types of cases, especially those in which controlled surgical intervention provides some additional confidence about the nature and extent of the brain damage. For example, Watkins and Oppenheimer (1962) presented a detailed account of a 57 year

[2] Authors differ in their use of the term *basal forebrain*. Basal forebrain used to indicate a general area of the brain refers to the ventral cerebral hemisphere areas forward of the hypothalamus (approximately Talairach $y = -10$). This general use somewhat variably includes some of the following: amygdala, basal ganglia, septal nuclei, nucleus accumbens, nucleus basalis, ventral anterior temporal lobe and ventral frontal lobe and associated pathways (e.g., see Parkin and Leng, 1993). Basal forebrain used more specifically, often in reference to lesions affecting cholinergic neurons and pathways, includes the septal nuclei, nucleus accumbens, nucleus basalis of Meynert, and diagonal band of Broca (see Damasio et al., 1985).

[3] Structures supplied by the anterior communicating artery include the basal forebrain, anterior cingulate cortex, anterior hypothalamus, anterior fornix, septal nuclei, anterior commissure, and genu of the corpus callosum (Parkin and Leng, 1993).



Abbreviation	Region	Displaced from medial section
ACC	Anterior Cingulate Cortex	5 mm
CN	Caudate Nucleus	5 mm
GP	Globus Pallidus	13 mm
NACs	Nucleus Accumbens and Septal Nuclei	4 mm
OFC	Orbitofrontal Cortex	5 mm
Pu	Putamen	13 mm
Th	Thalamus	5 mm

Notes: basal ganglia (CN, Pu, GP); striatum (CN and Pu); basal forebrain (NACs, nucleus basalis of Meynart, and diagonal band of Broca, but see footnote 2).

Fig. 1. Schematic representation of some of the brain regions discussed in text (adapted from Talairach and Tournoux, 1988).

old man who had lesion of the left thalamus as a consequence of a surgical procedure (thalamolysis) for treatment of Parkinsonian tremor. The operation involved placing a balloon in the ventrolateral nuclear mass of the thalamus. When the balloon was inflated, the patient 'immediately shouted 'Stand by, everyone in the aircraft' and from that moment was hallucinated, irrational, and inaccessible' (p. 243). For several weeks the patient's dominant mood was 'jocular expansiveness' and he exhibited gross amnesia for recent events. Subsequently, his confusion cleared and memory improved but he became anxious and depressed and 9 months after the operation, committed suicide. Examination of his brain was reported in detail. The track of the cannula included the left middle frontal gyrus, the genu of the internal capsule, the internal capsule and the medial tip of the globus pallidus. The 'brunt of the damage was sustained by the lateral and ventrolateral nuclei of the thalamus, and a portion of the internal capsule just behind the genu'. Whitty (1956, Case 5) reported an equally interesting case of a patient whose confabulations came and went as the cystic part of a tumor involving the third ventricle and the left thalamus was repeatedly tapped and filled.

Information about the consequences of damage to the anterior cingulate come from a remarkable series of papers describing patients who had undergone an anterior cingulectomy, in most cases to treat obsessive compulsive disorder (Whitty and Lewin, 1957, 1960; Whitty, 1966). The operation consisted of a bilateral cortical ablation in BA 24, approximately 1 cm wide and 1 cm deep, extending

back from the anterior end of the genu of the corpus callosum for 3–4 cm. Postoperatively, for several days, the patients reported especially vivid experiences that they were not sure were dreams or reality. For example, one patient reported having what seemed to him like waking dreams. When asked how he slept the night before he said 'I was on the go all the time: friends coming in to talk to me'. When the experimenter asked if the Sister (nurse) had let them in, the patient said 'No, they weren't really there, of course, but that's what I mean (referring to earlier questions about his thinking). My thoughts seem to be out of control, they go off on their own – so vivid. I am not sure half the time if I just thought it or it really happened' (Whitty and Lewin, 1957, p. 73). Generally, the patients exhibited awareness of their problem and recognized inconsistencies in their memories (Whitty and Lewin, 1960). Whitty and Lewin believed that most of these experiences were real events displaced in time. Temporal confusions are, of course, a type of source misattribution.

We will return to a discussion of brain regions after considering the characteristics of confabulations and the performance of confabulating patients on neuropsychological and other cognitive tasks.

Duration

Although it is frequently noted that confabulation often clears, there is little systematic evidence about how long confabulation lasts. Comparing across patients with different types of brain damage, the surgical lesions of anterior cingulectomy patients typically produced temporary confabulations lasting several days (Whitty and Lewin, 1957, 1960), damage to the basal forebrain may last weeks to months (e.g., Damasio et al., 1985), and damage to various other frontal areas may last months to years (Stuss et al., 1978). Within the category of basal forebrain damage, Schnider, Ptak and Remonda (1999) noted that their patients with pure orbitofrontal lesions recovered first; other basal forebrain lesions were associated with confabulations for months; confabulation lasted longer in a patient with bilateral damage (a combined lesion of the amygdala on one side and the perirhinal cortex on the other side) and in another patient with an orbitofrontal lesion which presumably destroyed all connections to the dorsolateral prefrontal cortex. More reports tracking the time course of confabulation for patients of various etiologies would be useful (e.g., Schnider et al., 1999).

The nature of confabulations

In ordinary, everyday cognition, monitoring processes are constantly engaged as people make attributions about the veridicality of perceptions, memories, beliefs, and knowledge about the past and present and as they make predictions about the future (Johnson, 1988; Johnson and Sherman, 1990). Because these processes are imperfect, reality and source monitoring distortions can occur in all these domains. However, we would not necessarily expect any particular individual to show distortions equally across all domains. The information considered and amount required, the relevant knowledge that is available and brought to bear, and the reflective processes engaged very likely change across domains and situations within domains. Motivational factors are not necessarily constant within individuals across domains. Furthermore, some individual differences such as imaging ability may be more likely to have an impact in some domains (e.g., episodic memory) than others (e.g., semantic memory). People may be more suggestible (e.g., Loftus, 1979) about some topics than others. We would not expect such individual differences to disappear under conditions of brain damage. In fact, their effects might be exaggerated (cf. Weinstein, 1996).

Like normal reality monitoring distortions, confabulations can be about autobiographical events, beliefs, or semantic knowledge; they can refer to the more remote past, the more recent past, the present, or the future (e.g., Dalla Barba, Cappelletti, Signorini et al., 1997a; Moscovitch and Melo, 1997). There might be differences in the degree of

distortion shown for these various types of confabulations both within and across brain damaged individuals (e.g., Dalla Barba, 1993a). For example, as we might expect in neurologically intact individuals, some patients might require less evidence to confabulate about a personal event than to confabulate about general knowledge or vice versa. Or, assuming that confabulations occur partly because cues activate bits of information around which a confabulation can be constructed, fragmented activation may be more likely for autobiographical than semantic memory in some patients, and perhaps the reverse in others. More standardized measures of confabulation that attempt to assess various domains and categorize confabulations will be necessary to begin to explore these issues.

Important steps toward more systematic collection of information about confabulation include procedures used by Dalla Barba (1993a,b) and by Moscovitch and Melo (1997). Dalla Barba developed a confabulation battery that asks questions about personal semantic memory (age, current address, number of children), specific personal events, orientation in time and place, general semantic memory (famous facts and people), and linguistic semantic memory (vocabulary test). The test includes several questions that normal subjects would be likely to answer with 'I don't know' – some semantic (e.g., What did Marilyn Monroe's father do?) and some episodic (e.g., Do you remember what you did on 13 March, 1985?) (see also Schnider, von Daniken and Gutbrod, 1996a). Moscovitch and Melo expanded the Crovitz test, in which subjects are asked to describe an autobiographical memory in response to a cue word (e.g., 'letter'), to include questions asking subjects to describe an historical event.

An approach for comparing the features of confabulated and nonconfabulated memories uses a memory characteristics questionnaire (MCQ, e.g., Johnson, Foley, Suengas and Raye, 1988) which either the subject or an experimenter can complete and that assesses qualitative characteristics of memories such as perceptual and spatial detail, emotion, amount of confidence in the memory, etc. (e.g., Johnson et al., 1997). An MCQ could also be used for comparing the qualities of confabulations observed in different patient groups or for assessing the changes in confabulation across time in the same patient. The MCQ, perhaps in combination with Dalla Barba's confabulation battery, might further help systematize the collection and characterization of confabulations.

Another approach to assessing confabulation compares patients on the extent to which they introduce intrusions in story or list recall (Kopelman, 1987; Cunningham, Pliskin, Cassisi et al., 1997; Schnider et al., 1996a). Standardizing the materials (e.g., which stories), procedure, and scoring method (e.g., number of intrusions, ratio of intrusions to ideas reported, etc.) used for this would be useful in characterizing patients, especially in combination with scores on a battery such as Dalla Barba's. Assessing confabulation for both naturally occurring memories and knowledge and for experimental materials is important because not all false memories and beliefs are produced by the same mechanisms (e.g., Johnson, 1988; Johnson et al., 1993) and thus one type of confabulation may not necessarily predict another (Schnider et al., 1996a).

The lack of a standard battery makes it difficult to compare content, frequency, or severity of confabulations across studies. The criteria or classification scheme used by individual researchers varied considerably. For example, investigators have distinguished between reactive and fantastic confabulations or between provoked and spontaneous confabulations (Berlyne, 1972; Kopelman, 1987). Reactive/provoked confabulations tend to be minor embellishments, or elements of true memories displaced in time; these are often produced in response to questions. Fantastic/spontaneous tend to be confabulations of entire events, not necessarily of actual events (although they may reflect dreams, information seen on TV, etc.), or actual events greatly displaced in time; sometimes they are implausible or even bizarre. Such confabulations may be offered in response to questions, but may also occur at other times without obvious

prompting. These classifications cannot simply be mapped onto each other because provoked confabulations could be realistic or fantastic, as could spontaneous confabulations. Frequently, in published papers, insufficient detail was provided to determine which of these four categories was appropriate. Another problem is that what might seem fantastic to the experimenter could in fact be real and vice versa (e.g., Benson and Stuss, 1990). Also, the scheme used depends on the range of confabulations observed in a particular study. A patient showing a 'high' level of confabulation assessed by intrusions in story recall in one study might have been classified as 'mild' if compared to patients classified on the basis of a clinical interview in another study. The task is further complicated by the fact that a patient may show more than one type of confabulation and/or the nature of their confabulations may change over the course of recovery.

Nevertheless, we attempted to classify patients in terms of the nature of the confabulation reported in the papers under review. In order to obtain at least a coarse-cut summary of evidence across reports, we classified as many of the cases as we could as either *mild* or *severe*. (We do not mean to imply that confabulations differ only in degree; indeed, as discussed above, the SMF proposes that there are a number of different ways that source misattributions can come about.) The severe category included any patient(s) the authors designated as fantastic, wild, bizarre, spontaneous, free, severe, profound, marked, atypical, persistent, extended, or high. The mild category included any patient(s) the authors designated as mild, moderate, momentary, typical, provoked, realistic, or low. We classified several other patients based on other information provided by the authors. In these cases, severe was used for cases in which the patient, at any time during observation, exhibited fantastic confabulations or realistic but implausible confabulations, or memories that were greatly jumbled or temporally disordered. The mild category was used for minor gap-filling of a realistic sort (e.g., in story recall) and autobiographical memories showing mild temporal displacement involving days or months. Of the 349 cases, 133 could not be classified. Table 4 shows the number of patients classified as mild or severe broken down by etiology. Approximately 69% of the reported cases of ACOA patients were classified as severe, followed by approximately 50% of tumor cases, 44% of anterior cingulate cases, 23% of the stroke cases, 21% of the trauma cases, and 16% of Korsakoff patients – consistent with previous reports that fantastic confabulation in Korsakoff patients is relatively rare (Kopelman, 1987; Parkin and Leng, 1993). The large percentage of ACOA patients classified as severe points to the importance in normal source monitoring processes of the brain structures affected by this type of aneurysm.

Table 5 shows the relation between hemisphere of damage (left, right, bilateral) and classification as mild or severe confabulation in the patients reviewed here. The percentages of left, right, and bilateral hemisphere patients that were severe as opposed to mild were 66%, 59%, and 59%, respectively.

Executive function, memory deficits and confabulation

Confabulation was described as a clinical symptom sometimes associated with alcohol abuse by Kor-

TABLE 4
Etiology and type of confabulation

Number of confabulating patients	Type of confabulation			
Etiology	Mild	Severe	NPD[a]	Grand total
ACOA	14	45	6	65
Cingulectomy	10	8	0	18
Korsakoff	26	12	37	75
Trauma	14	20	63	97
Tumor	7	9	2	18
Stroke	30	15	21	66
Other	2	4	4	10
Grand total	103	113	133	349

[a] NPD = not possible to determine the type of confabulation from the information given in the article.

TABLE 5
Laterality and type of confabulation

Number of confabulating patients	Type of confabulation			
Damaged hemisphere	Mild	Severe	NPD[a]	Total
Left	5	19	5	29
Right	12	19	1	32
Bilateral	17	38	9	64
Not specified	69[b]	37[c]	118[d]	224
Grand total	103	113	133	349

[a] NPD = not possible to determine the type of confabulation from the information given in the article.
[b] Includes six ACOA aneurysm patients and 26 Korsakoff patients.
[c] Includes four ACOA aneurysm patients and 10 Korsakoff patients.
[d] Includes one ACOA aneurysm patients and 37 Korsakoff patients.

sakoff (1889/1996, see also Talland, 1965) at about the same time that Ebbinghaus was reporting his groundbreaking studies of memory. Korsakoff thought of confabulation as being on a continuum of severity and associated it with a 'peculiar form of amnesia'. Korsakoff observed that memory deficit alone was not enough to produce confabulation and that confabulation was relatively uncommon in the population of all patients with memory deficits. A clear characterization of the ways in which confabulating patients' memory performance does and does not resemble nonconfabulating patients with memory disorders would be useful, but there are surprisingly few studies designed to explicitly compare confabulating with similar nonconfabulating patients (e.g., DeLuca, 1993; Johnson, O'Connor, and Cantor, 1997; Schnider, von Daniken and Gutbrod, 1996a).

Memory in brain damaged patients is typically assessed with the WMS, CLVT or other standard neuropsychological tests. Profound disruptions in memory (recognition, recall) result from damage to medial temporal lobe/diencephalic structures (especially the hippocampus, entorhinal cortex, parahippocampal cortex and perirhinal cortex, Squire and Knowlton, 1995). Memory deficits in medial-temporal amnesics typically are reflected in poor recall (errors of omission), poor recognition, slow or no learning with repeated exposures, and rapid forgetting. Executive function is typically assessed with tasks that minimize explicit long-term memory requirements and which measure problem solving (e.g., Tower of London), categorization (Wisconsin Card Sorting Task – WCST), verbal fluency (give words starting with 'f'), ability to overcome response competition (e.g., Stroop), and task switching (e.g., trails – B) (e.g., Shimamura, 1995; Stuss and Benson, 1986). Because patients with frontal damage often score poorly on these latter tests, 'frontal' function is often used as a synonym for executive function but various reflective processes recruited may involve a number of brain regions (e.g., prefrontal cortex, anterior cingulate, basal ganglia; see below). In general, the distinction between memory and executive function may be somewhat misleading because reflective processes are critical for memory and vice versa. For example, although the hippocampal formation may bind features together into complex memories (e.g., Cohen and Eichenbaum, 1993; Squire and Zola-Morgan, 1991), frontal/executive processes greatly influence which features enjoy the opportunity for binding (e.g., Goldman-Rakic, Selemon and Schwartz, 1984; Johnson, 1992). The relative frequency of use of different neuropsychological tests to assess confabulating patients is shown in Table 6. From Table 6 it is clear that scores on a common set of standard neuropsychological tests are not available for most confabulating patients.

Clinically, confabulating patients, especially those showing more severe forms, tend initially to be confused, disoriented, and to show memory disturbances, although not necessarily the most profound forms of amnesia. In contrast, patients with medial temporal damage alone may be profoundly amnesic but are rarely reported to confabulate. Confabulating patients also are likely to show deficits on many (although not necessarily all) tests of executive function (e.g., Dalla Barba, Mantovan, Cappelletti and Denes, 1998). As patients recover from the initial trauma (head injury, operated tu-

TABLE 6
The number of confabulating patients who received various neuropsychological tests

Neuropsychological test	Number of patients
WMS-LM	100
WCST	83
WAIS-R	79
Fluency	74
CVLT	68
Trailmaking test	64
WMS-R	51
Stroop	49
Digit span	47
Cowat	41
Rey figure copying	40
WMS-PAL	32
Boston naming test	30
WMS-mental control	30
WMS-VR	28
WMS-general	27
WMS-delayed	27
WMS-orientation	22
Confabulation battery	21
Boston diagnostic aphasia exam	20
WMS-info	20
Rey auditory verbal learning	16
Boston famous faces test	14
WRMT-faces	14
NART	14
WRMT-words	13
Cognitive estimates	10

mor, aneurysm, etc.), they tend to confabulate less. Sometimes this is accompanied by improved executive function scores (Kapur and Coughlin, 1980), but not always (Schnider et al., 1999). Because memory often does not improve, it appears that confabulation results from executive dysfunction superimposed on memory deficits. (A notable exception was a head injury patient reported by Stuss et al. (1978) whose memory improved over a period when his confabulation did not.) Nevertheless, across the cases reported in the literature, it is clear that although amnesia alone is not necessarily accompanied by confabulation, confabulating patients are quite likely to show memory deficits (e.g., DeLuca, 1993; Stuss et al., 1978). Although confabulating patients are also likely to show deficits on frontal tasks, which frontal tasks are administered and how patients perform on them is quite variable (at least in part because standard tests of executive function vary in the component reflective processes they recruit). As Schnider et al. (1996a, p. 1365) note, the dysexecutive syndrome 'represents a broad class of cognitive failures following prefrontal damage or disconnection ... thus, this does not disclose the specific mechanism of confabulations'. That is, it is important to attempt to identify the particular source memory processes that are disrupted in various patients in order to understand the different types of memory deficits, including various types of confabulation, that are observed.

There have been surprisingly few confabulating patients tested on cognitive tasks other than the standard neuropsychological tests shown in Table 6. In addition to the investigations of autobiographical recall and comparisons of autobiographical and semantic memory already mentioned, there have been a few studies directly investigating confabulating patients' source memory. We briefly summarize these next.

Schnider et al. (1996a) gave patients a continuous yes/no recognition task in which repeated targets (line drawings) were intermixed with distractors. A second list was given an hour later in which the distractors from the first list became targets and first-list targets became distractors. Compared to other amnesics, a group of confabulating patients had particular difficulty when the targets and distractors were switched, suggesting they had a deficit in identifying the source of familiarity based on temporal cues and/or inhibiting responses to the most familiar items from the first test. That this is not a deficit limited to temporal memory is suggested by the results of a more detailed study of one of the patients from this same group (Schnider et al., 1996b); the patient showed poor source memory both for which experimenter taught her which words and for which room she had learned the words in (relative to two age-matched normal controls).

Dalla Barba et al. (1997a) tested an ACOA patient in two source tasks. In one, the patient (GA) was asked to discriminate actual episodes from her life ('today for lunch you had boiled meat')

from some confabulations she had produced in previous testing sessions ('today for lunch you cooked spaghetti'). She was more likely to endorse confabulations (86%) than actual events (43%). Dalla Barba et al. suggested she was likely to be influenced by her 'personal semantic information'. Based on the SMF, such schema-based responding should increase when other source evidence is lacking (Mather et al., 1999). In a second source identification task, she was severely impaired, relative to normal controls, in discriminating names of objects referring to pictures she had seen from the names of objects she had been asked to imagine. Her old/new recognition was also very poor. Similarly, Johnson, Grande and Milberg (reported in Johnson, 1997) tested a confabulating ACOA patient (WL) in a similar task and found poor recognition memory and poor source memory. Interestingly, WL showed an overwhelming tendency to call any item recognized as old 'picture' whereas Dalla Barba et al.'s patient GA made source errors equally in both directions. Both asymmetrical and symmetrical patterns reflect source memory deficits; however, it would be interesting to know whether WL's and GA's source errors were accompanied by similar or different subjective experience as assessed, for example, by an MCQ.

Johnson et al. (1997) investigated source memory in another confabulating ACOA patient, GS. On a speaker identification task, GS showed a clear deficit in source identification relative to normal controls, but it was about the same magnitude as the source deficit of three other, nonconfabulating frontal patients. On a temporal order discrimination task (identifying items from lists presented 30 min apart), GS was superior to the other frontal patients in source accuracy and did not differ from the normal controls. However, GS did show clinical signs of temporal confusion about when autobiographical events had occurred. GS also had somewhat richer memories for imagined than perceived events (e.g., experimental tasks such as imagining himself hammering versus actually typing), a pattern not found for normal or frontal controls. This may be similar to Dalla Barba et al.'s (1997a) finding that GA recognized more of her confabulations than actual events as true. As suggested by Johnson et al., memories for vivid imaginations (or personal semantic knowledge) may be especially compelling against a background of generally impoverished memories.

Thus, overall, available evidence about source monitoring in confabulating patients indicates that they generally show deficits in source identification tasks relative to normals. However, amnesic (Chalfonte, Verfaellie, Johnson and Reiss, 1996; Schnider, von Daniken and Gutbrod, 1996c) and frontal (Janowsky, Shimamura and Squire, 1989; Shimamura, 1995) patients who do not confabulate show source memory deficits as well. The underlying mechanisms for source deficits are not likely exactly the same for medial-temporal amnesics and frontal patients (Johnson et al., 1993). First, at encoding, medial-temporal damage may disrupt feature binding processes (e.g., Murray and Bussey, 1999; Chalfonte et al., 1996; Damasio et al., 1985; Eichenbaum and Bunsey, 1995; Schacter and Wagner, 1999); frontal damage may disrupt processes that facilitate feature binding (e.g., noting, refreshing, and rehearsing conjunctions of features) (e.g., Rao, Rainer and Miller, 1997), as part of a frontal/medial-temporal circuit (e.g., Mitchell, Druzgal, Raye et al., 1999). Second, during remembering, medial-temporal damage may disrupt reactivation of bound features whereas frontal damage may disrupt more systematic retrieval and evaluation processes (e.g., initiating cues, setting appropriate criteria, etc.) (e.g., Damasio et al., 1985; Johnson, 1997; Moscovitch and Melo, 1997).

According to the SMF, any disruption in the ability to encode and/or retrieve feature information would show up, of course, on an explicit source memory task. But such patients would not necessarily confabulate. Poor feature information may increase the opportunity for confabulation, but confabulation requires more than a deficit in feature information or in bound feature information. It requires a failure in source attribution processes that evaluate the quality of the information (i.e., that make one aware that information is

poor) and that link belief or action to the quality of the information. For example, confabulating patients may have particular difficulty engaging or maintaining an agenda to evaluate mental experiences. Cognitively, self-monitoring requires transactions between subsets of reflective processes (e.g., taking thoughts as the objects of evaluation or reflecting on reflection, Johnson and Reeder, 1997), and is subserved by frontally mediated interactions (Stuss, 1991), perhaps typically involving both hemispheres (cf. Johnson, 1997; Johnson and Reeder, 1997). Deficits in processes that monitor more specific qualitative characteristics of thoughts may result in overresponsiveness to nonsource diagnostic cues such as familiarity. The impact of disruption in these various factors on the source memory processes of different confabulating patients remains to be sorted out.

In short, the specific reflective processes that are disrupted in confabulating patients and that account for their memory distortions need more focal investigation to assess their relative contribution (see also Moscovitch and Melo, 1997). Suggestions regarding the anatomical regions that subserve these processes have included several ideas: (1) confabulation results from damage in ventromedial frontal cortex (e.g., Moscovitch and Melo, 1997); (2) confabulation results from amnesia from, for example, basal forebrain (septal nuclei, nucleus accumbens, nucleus basalis of Meynart, and diagonal band of Broca, see Damasio et al., 1985) damage, in combination with frontal damage (DeLuca, 1993); another variant of this is that both mild and severe confabulation involve damage to septal areas, with severe confabulators also having bilateral or medial frontal damage and damage to the striatum, specifically the head of the caudate (Fischer et al., 1995); (3) confabulation (at least the more spontaneous, acted out variety) involves a disconnection of prefrontal, probably orbitofrontal areas, from a circuit that includes the dorsomedial nucleus of the thalamus and the amygdala (Schnider et al., 1996a); (4) confabulations of increasing durations are associated with, respectively, lesions in anterior cingulate, basal forebrain, and frontal areas (Johnson, 1991). Thus based on the picture from confabulating patients alone, candidate brain regions involved in normal source monitoring include ventromedial and orbitofrontal regions, structures in the basal forebrain, the caudate nucleus, and the anterior cingulate (see Fig. 1).

In the next section we consider three anatomical circuits (Cummings, 1993) and their potential usefulness for understanding the relation between the brain damage seen in confabulating patients and the cognitive processes involved in source monitoring. We will suggest that these or similar circuits, in combination with ideas about the processes involved in memory derived from the SMF, provide a way of integrating these various hypotheses about brain damage and confabulation.

Segregated frontal-subcortical circuits

Cummings (1993) outlined five frontal-subcortical circuits, three of which are of particular interest with respect to source memory (see Fig. 2): a dorsolateral prefrontal circuit originating in the convexity of the frontal lobe (BA 9 and 10), a lateral orbitofrontal circuit originating in inferolateral PFC (BA 10) and a circuit originating in the anterior cingulate (BA 24). The first two circuits project from PFC (dorsolateral and lateral orbital, respectively) to the caudate, then globus pallidus and then thalamus and back to PFC. The third projects from the anterior cingulate to the nucleus accumbens, then globus pallidus, then thalamus and back. The specific subregions of the caudate, globus pallidus and thalamus depend on the circuit. What is interesting about these circuits is that disruption in each is associated clinically with different syndromes each of which might be expected to have some impact on source monitoring. Disruption of the *dorsolateral prefrontal circuit* is associated with poor performance on neuropsychological tests assessing fluency, motor planning, memory retrieval, set-shifting, learning, and problem solving. Disruption of the *lateral orbitofrontal circuit* is associated with disinhibition (e.g., inabil-

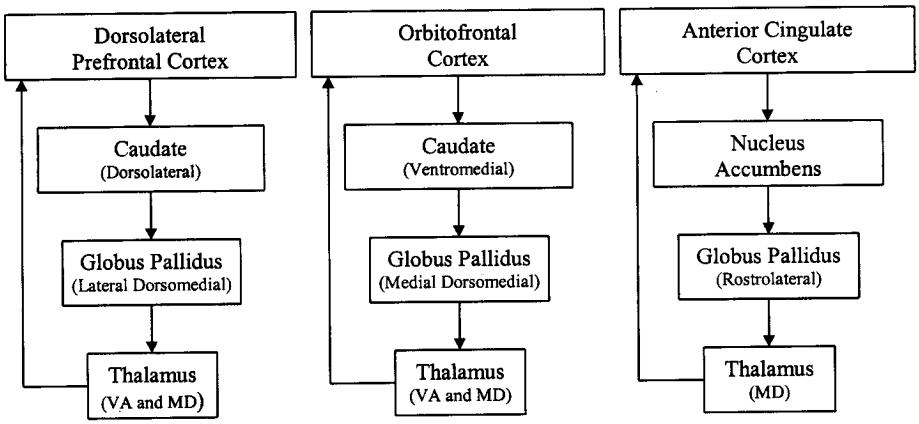


Fig. 2. Organization of three frontal-subcortical circuits (VA indicates ventral anterior; MD, medial dorsal). The indirect circuits and connections of the substantia nigra and the subthalamic nucleus are not shown (adapted from Cummings, 1993; Manning and Cummings, 1997).

ity to suppress responses to irrelevant stimuli). Disruption of *the medial frontal-anterior cingulate circuit* is associated with diminished initiative (Cummings, 1993).

As can be seen in Table 3, most of the cases of confabulation reviewed here may have involved damage to one or more of these three circuits. Furthermore, various hypotheses that have been offered about the relation of brain damage to confabulation have emphasized the importance of one or more structures in these circuits and thus can be roughly classified by circuit with the best match in structures emphasized: (1) dorsolateral prefrontal circuit (Fischer et al., 1995); (2) orbitofrontal circuit (Fisher et al., 1995; Schnider et al., 1999); (3) anterior cingulate circuit (Damasio et al., 1985, DeLuca, 1993; Johnson, 1991). As discussed next, there is converging evidence from neuroimaging of normal subjects for the importance of two of the three circuits during source monitoring (dorsolateral and anterior cingulate).

Evidence from neuroimaging about brain regions active during source monitoring

Activations in dorsolateral PFC frequently have been found during tests of episodic memory (e.g., Tulving et al., 1994; Nolde, Johnson and Raye, 1998). Many of the same areas are also active during WM tasks (D'Esposito, Aguirre, Zarahn et al., 1998), supporting the idea that both LTM and WM tasks draw from a common set of component processes (Johnson, 1997). Furthermore, the evidence from both neuroimaging and event-related potential (ERP) studies is generally consistent with the idea that PFC is even more important in source identification than in old/new recognition (e.g., Johnson, Kounios and Nolde, 1996; Wilding and Rugg, 1996; Ranganath and Paller, 1999). In addition, source identification tasks (pictures vs. words, Nolde, Johnson and D'Esposito, 1998) show greater left PFC activity than old/new recognition judgments which do not explicitly require subjects to retrieve or evaluate specific feature information. Similarly, left PFC activity has been reported for other simple feature source judgments – temporal (Zorilla, Aquirre, Zarahn et al. 1996) and location (Nyberg, McIntosh, Houle et al., 1996; see also Ranganath and Paller, 1999). Finally, as noted above, remembering autobiographical events often requires a type of iterative source monitoring in which cues are self-generated, the resulting retrieved information is evaluated, new cues are generated, etc. (see also Burgess and Shallice, 1996; Conway, 1992, 1996). A neuroimaging study by Conway, Turk, Miller et al. (1999) reports

increases in left PFC activity during autobiographical recall.[4]

A recent neuroimaging study explicitly investigating complexity of source monitoring by Raye, Nolde, Mitchell et al. (1999) found activation that may reflect two of the three circuits described by Cummings (1993) – the dorsolateral PFC circuit and the anterior cingulate circuit. Subjects saw pictures and words and heard other words and were later required to make three different sorts of source attributions as words were presented on a screen: old/new (yes to all old items), picture (yes to items previously seen as pictures and no to all other items), picture/heard (yes to items previously seen as pictures and items previously heard and no to all other items). These three conditions were selected to vary in the complexity of the source monitoring processes involved. Old/new recognition can be based on familiarity, whereas both P and PH conditions require more specific source information. The PH relative to the P condition was, in addition, expected to require more shifting between qualities (visual/pictorial and auditory) of representations during evaluation, and perhaps the need to maintain and adjust separate criteria for the two dimensions.

Areas with activations that varied with condition included left dorsolateral PFC near the intersections of BA 9/10/46. This finding is consistent with a recent review of neuroimaging research that found that left PFC is likely to become engaged as more systematic processes are required during episodic long-term memory tasks (Nolde et al., 1998a). This area showed the pattern ON < P = PH. Because P and PH did not differ here, it suggests this particular activation may reflect processes required for more selective (specific) reactivation or retrieval of feature information. Alternatively, it may reflect the greater demands to keep information active while it is being evaluated during source identification than during old/new recognition.

With respect to the third circuit described by Cummings (1993), activation in anterior cingulate cortex (ACC) is also often found in episodic memory studies (Nyberg, 1998). For example, we examined the studies reviewed by Nolde et al. (1998a), and found that approximately 75% reported activity in either BA 32 or both BA 24 and 32 during episodic recognition or recall. There were two regions of BA 32 activation in the Raye et al. (1999) study, one more inferior and one more superior, showing the pattern ON < P < PH. The superior ACC activation was very close to areas reported by Barch, Braver, Nystrom et al. (1997) and Carter, Braver, Barch et al. (1998). Barch et al. found increased ACC activity here with increased task difficulty, and Carter et al. found increased ACC activity here with increased response competition even when responses were correct. They suggested that ACC is sensitive to conditions likely to lead to er-

[4] Table 5 shows a somewhat higher percentage of severe than mild confabulation in left than right hemisphere patients, but the percentage of severe confabulations in bilateral patients was not greater than in right hemisphere patients. In combination with the neuroimaging results, these findings support two hypotheses worth pursuing. (1) The more systematic processes engaged in remembering do not depend on left hemisphere activity alone, but right and left working together – thus damage in either hemisphere (or both) may have a similar impact with respect to severity of confabulation. A related possibility is that confabulating patients who show unilateral damage on a scan may have bilateral dysfunction initially, perhaps as a consequence of disruption in neurotransmitters (dopamine, acetylcholine) subsequent to trauma, which accounts for their confabulation; when their condition stabilizes, we see reductions in their confabulations and a poor correlation between primary hemisphere of structural damage and type of confabulation. (2) The level of damage in the left PFC may be related to the quality of the episodic memory that the patient can encode and/or retrieve, but without damage to other regions, this would not necessarily alone result in confabulation because the patient is aware of the poor or ambiguous quality of what is remembered and has appropriate doubts about its veridicality. This is consistent with the suggestion below that the disruption of the different circuits in Fig. 1 would be differentially associated with errors of omission and errors of commission. Alternatively, there might indeed be different kinds of confabulations associated with right, left, and bilateral hemispheric damage but this is not apparent from comparing across studies using different criteria for characterizing confabulations and without more specific evidence regarding the particular region(s) of damage (e.g., some areas of damage may produce more lateralized effects than others). Evaluating these various alternatives depends on collecting more systematic evidence about both the nature of confabulations and regions of damage.

rors and sensitive to response competition. Activations similar to both our inferior and superior ACC areas were reported in D'Esposito, Detre, Alsop et al. (1995). D'Esposito et al. found greater AC activity in these two areas during dual than single tasks and suggested that ACC is involved in task switching. One possibility is that both ideas are correct and that different regions of ACC participate in different but related functions (cf. Paus, Koski, Caramanos et al., 1998). Thus both of these ACC activations may be part of an anterior cingulate circuit shown in Fig. 2; however, they may reflect different component processes within that circuit. Activity in inferior ACC may reflect *shifting* attention among different features of information, or sensitivity to task conflict that initiates a shift in attention. Activation in superior ACC may reflect sensitivity to other types of competition, for example, among similar representations, or uncertainty in the outcome of the noting or match process, and its outputs may signal (*initiate*) the need for further processing, more systematic processing (e.g., retrieval), or a change in processing (e.g., a need to adjust criteria).

With respect to the second circuit described by Cummings, based on the neuropsychological evidence presented in Table 3, we might expect different levels of activation in basal forebrain/orbitofrontal regions during source identification compared to old/new recognition. Neither Nolde et al. (1998b) nor Raye et al. (1999) identified activity in this region, however it can be difficult to obtain good functional data in this region. Further studies of source memory directed at this area would be useful. One possibility consistent with Cummings' description is that the orbitofrontal circuit participates in the inhibition of non agenda-related activation (or perhaps helps maintain an agenda) which reduces interference and facilitates ongoing processing in dorsolateral PFC and anterior cingulate circuits. Damage to the orbitofrontal region might therefore result in interference that could impair the functioning of other processes that might otherwise be intact (and that would allow for higher performance under conditions of reduced interference). For example, poor performance in patients with orbitofrontal lesions when previously familiar targets became distractors (Schnider et al., 1996a) would be consistent with this idea. That is, orbitofrontal cortex may help mediate the extent to which initial responses (e.g., based on recency, familiarity, perceptually salient stimuli, etc.) are withheld or suppressed until more information is considered.

Results from a study by Benson, Djenderedjian, Miller et al. (1995) of a confabulating Korsakoff patient provides intriguing evidence consistent with this general picture. Using SPECT, they found hypoperfusion in the medial orbital frontal area and cingulate during the period when the patient was confabulating, which improved to normal on a subsequent scan at 4 months. This patient's confabulations were prominent initially but absent at 6 weeks. Over the same time period, the patient improved on neuropsychological tests of frontal function, but not of memory. The area of most change in blood supply overlaps with the inferior ACC area reported by Raye et al.

Conclusions and future directions

A sense of remembering depends on mental experiences with rich detail that are taken to be memories. Having experiences of remembering that reflect reality is tied to a number of factors: binding and consolidating the features of experience, reactivating and retrieving these features together later, evaluating them with respect to appropriate criteria about what constitutes a memory, and distinguishing them from other mental experiences. All of these processes are inherently constructive and reconstructive – they are influenced by prior knowledge, beliefs, expectations, and motives. To sort out specific, actual events from the merely known, imagined, associated, or fleetingly brought to mind by environmental cues (or to distinguish reasonable from unreasonable beliefs) requires self-monitoring and cognitive control (which include taking mental experiences as objects of reflection; Johnson and Reeder, 1997). The impor-

tance of the experience of doubt and ambiguity should not be underestimated – doubt often is a clue that heuristic monitoring is ongoing; doubt often initiates more systematic monitoring; and doubt is the normal appropriate subjective experience when evidence is poor. Such encoding, revival and evaluation processes are described at a cognitive level in the source monitoring framework. Based on the combined evidence from brain damage and from neuroimaging of normal subjects, such episodic remembering is likely to involve all three of the circuits shown in Fig. 2 or similar such circuits. Thus we would expect memory to be disrupted by damage to any of them.

We cannot, as yet, with certainty tie particular processes to particular circuits or parts of circuits, but some working hypotheses are suggested by the available evidence. Damage to the dorsolateral circuit may reduce subjects' ability to encode the relations between features of events because, presumably, this would disrupt the working memory processes that help sustain the activation of conjunctions of feature information through refreshing, rehearsing and noting. Damage to this circuit would also affect the systematic retrieval of information required by complex tasks, including autobiographical recall. Damage to the anterior cingulate circuit may disrupt a person's ability to detect conflict or inconsistencies in sources of information, initiate processes, and shift among types of processes or representations (and produce the cognitive operations cues that signal the self as the origin of information). Damage to the orbitofrontal circuit may disrupt processes (either inhibitory or excitatory agenda maintenance processes) that help foreground stimuli related to current agendas and the information that is most relevant to task goals, or may disrupt processes that withhold responses (e.g., based on familiarity) until better information revives and/or can be evaluated (e.g., Johnson et al., 1994). Thus damage to the dorsolateral circuit may be more likely to produce errors of omission than of commission, whereas damage to either the anterior cingulate or the orbitofrontal circuit may be more likely to produce errors of commission because these regions may be critical to a person's ability to self-monitor – that is, to take mental experiences (or behavior) as objects of reflection. Disruption of the orbitofrontal circuit may be particularly likely to result in spontaneous, disorganized cognition that is most strikingly seen in patients in confused or confabulatory states. The fact that confabulation is associated with damage to medial structures where there is a relatively high probability that damage will disrupt, if only temporarily, normal interhemispheric communication is consistent with the idea that such self-monitoring ordinarily often involves interactions between hemispheres (e.g., Johnson, 1997). Also, it should be kept in mind that these circuits (and probably others) very likely work together. Furthermore, a process defined in cognitive terms may involve more than one of the circuits outlined in Fig. 1 (e.g., maintaining an agenda, especially in the face of interference, may require both orbitofrontal and dorsolateral circuits). And, of course, brain damage may often affect more than one circuit.

Much information has been gained about confabulation using clinical assessment of confabulation, standard neuropsychological tests, and structural neuroimaging. But additional progress requires more than a structural scan and administering standard neuropsychological tests (e.g., WCST), or testing patients in only one version of a laboratory task (e.g., list discrimination). Also, because confabulating patients are relatively rare, developing practices that would make it easier to accumulate evidence across studies would be useful. With this in mind, we have several recommendations: (1) use of consistent abbreviations or patient numbers across reports so that individual patients can be identified; (2) schematic representation of results of scans (e.g., Schnider, 1999) and reports of individual scores on neuropsychological tasks (e.g., Beeckmans et al., 1998; Fisher et al., 1995) so that patients can be compared across studies; (3) standardized assessments of confabulation, which provide the opportunity for both episodic and semantic confabulation (e.g., Dalla Barba,

1993a; Schnider, 1996a) and which have been equated for difficulty in normal subjects (Moscovitch and Melo, 1997); (4) the development of coding procedures for summarizing the qualitative characteristics of memories and beliefs (e.g., Johnson et al., 1997), both those informally observed in everyday situations and those elicited during interviews and testing; (5) where possible, comparisons of confabulating patients with other patients (e.g., DeLuca, 1993) who differ in some key point of interest (e.g., unilateral vs. bilateral damage; orbitomedial damage vs. dorsolateral PFC damage; (6) use of a broader range of experimental tasks; rather than test only one type of feature (e.g., temporal memory), test several in order to assess both generality and specificity in disruption (e.g., source memory for different features such as speaker, time, modality, e.g., Johnson et al., 1997); (7) attempt to vary the extent to which specific processes are recruited. With respect to this last approach, particularly promising is the possibility of varying processing demands (e.g., complexity) and observing correlated brain activity in both normal (e.g., Raye et al., 1999) and brain damaged patients; (8) consider the possibility that individual differences (e.g., in anxiety, imagery ability, suggestibility, etc.) that affect normal memory affect patients' memory as well (e.g., Weinstein, 1996).

We are far from having a dynamic, real-time mapping of specific brain processes to specific cognitive processes in normal source monitoring, and even farther from understanding how such processes are disrupted and the consequences of disruption of any one type of process for the functioning of others. Nevertheless, building on evidence and ideas that have come before, we can also see the rough outlines of a future in which just such questions can be addressed.

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