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Confabulation: Honest Lying

WHAT IS CONFABULATION?

We are all wrong. Philosophers, scientists, writers, poets, even mathematicians make huge, sometime devastating errors. We make errors in our daily life — errors of judgment, of planning, of memory, among others. Much of the time, we are not aware of our errors, we feel that we are right and we keep maintaining our position regardless of any evidence in contrast with it. In some cases, however, our mistakes in judging and remembering are so evident that we need other names to distinguish them from ''normal'' errors. Delusion and confabulation are the names we use to indicate ''abnormal'' errors in judgment and remembering.

So starts a recent paper (Dalla Barba and Boisse 2010) on a study on confabulation.

The term "confabulation" has been used to denote a wide range of errors in memory as well as distortions in other cognitive domains. Despite confabulation having been studied for well over a century, there is very little on which researchers agree, even when it comes to the most basic question of how confabulating should be defined, or how many types of confabulation there are.

At its simplest, people who confabulate provide information, or act based on information, that is obviously false. These people are ***genuinely unaware*** that the information is wrong.

A number of disorders can cause confabulations, including Korsakoff's syndrome, ruptured aneurisms of the anterior or posterior communicating artery, subarachnoid hemorrhage, encephalitis, traumatic brain injuries, brain tumors, Binswanger's encephalopathy, multiple sclerosis, and psychotic disorders. Confabulation is often seen in dementia patients, although it has not been extensively studied in this particular group; and to my knowledge, the studies that have been done have focused exclusively on early-stage dementia.

Studies on all disorders are typically lumped together when trying to develop clues on what causes confabulation and how it is expressed. However, there may be distinct differences in the ways and frequencies with which patients with different disorders, with damage to different parts of the brain, or with different types of damage to the same part(s) of the brain, confabulate. And it is quite possible that

the results from studies done on early-stage dementia may result in a skewed concept of when and why and how moderately- and severely-demented patients confabulate.

Different types of confabulations

The most recent papers I found generally agree on the core characteristics of all forms of confabulation, i.e.:

(i) Confabulation is an account based in memory that is false with respect to the context (e.g., time, place, etc.) in which the event is placed, and may contain false or grossly inaccurate details within its own context.

(ii) Patients typically exhibit anosognosia for their memory problems and are unaware of the fact that they are confabulating. Thus, confabulations are not intentionally produced and are probably not the result of compensatory mechanisms. However, the content of a confabulation may be emotionally biased, e.g., may reflect unintentional motivations and drives.

(iii) Patients may act upon their confabulation, reflecting their genuine belief in the false memory.

(iv) Confabulations are most frequent in the autobiographical domain (e.g., personal past experiences or plans for the future), and autobiographical confabulations are usually associated with the strongest confidence in their veracity.

Various attempts have been made to distinguish among the different kinds of confabulation. One common distinction is between "semantic" and "episodic" confabulations. ***Semantic memory*** refers to a person's store of conceptual and factual knowledge that is not related to any specific memory, such as the color of broccoli or what a fork is used for. ***Episodic memory*** involves the ability to learn, store, and retrieve information about unique personal experiences that occur in daily life, such as the details of a recent office meeting or a special holiday gathering that took place several years earlier. Episodic memories typically include information about the time and place of an event, as well as detailed information about the event itself. These memories may be drawn upon to envision future events. Planning and anticipation are similar to recalling personal memories and use similar areas of the brain. ***Semantic confabulations***, then, are false statements associated with knowledge of generally known facts. ***Episodic confabulations*** are false statements associated with memories of personal past episodes or experiences, or personal plans for the future.

Another very common distinction is between "provoked" and "spontaneous" confabulations. ***Provoked confabulations*** are typically described as plausible minor memory distortions in response to direct questioning — reflecting the need to fill a memory gap when questioned or in a test situation — whereas ***spontaneous confabulations*** are unprovoked, often implausible, memories. Provoked confabulation resembles errors produced by healthy persons on tests of memory following prolonged retention

intervals, and may reflect a normal strategy to compensate for memory deficits. Spontaneous confabulation, on the other hand, is thought to be due to impaired source memory (i.e., a deficit in remembering contextual information about an event) and temporal confusion (i.e., the difficulty in distinguishing irrelevant and old memory traces from relevant and new traces referring to the ongoing reality).

However, it can be difficult to draw the line between provoked and spontaneous confabulations. Moreover, several researchers have noted cases in which spontaneous confabulations were plausible and provoked confabulations were bizarre and implausible. In fact, a recent study (Dalla Barba and Boisse 2010) concluded that the great majority of confabulations do not clearly fall in either category. Instead, they consisted of "general memories, habits, and misplacements", i.e., either true episodes misplaced in time and place, or personal habits and routines that the patient "remembers" as specific events. When asked what they did today or what they will do tomorrow, for example, confabulating patients may reply with well-established memories from their pasts, however irrelevant these memories may be to their present situations.

Accordingly, Dalla Barba and Boisse also suggested distinguishing between semantically anomalous and semantically appropriate confabulations. A confabulation that is ***semantically anomalous*** is one that is inconsistent with knowledge and information shared by the members of society. However, it may be made of autobiographical elements put together in an inappropriate semantic structure. An example of this is a statement made by a patient suffering from traumatic brain injury. He said that he had won a running race the previous day and was awarded with a piece of meat that was placed on his right knee. It is unlikely that anyone would have such an experience (semantically anomalous). However, elements of the statement were autobiographical: the patient had spent much of his free time running races earlier in his life, and he fell during a race, incurring severe head trauma and an open wound on his right knee. A ***semantically appropriate*** confabulation, on the other hand, might sound perfectly plausible to someone who does not know the patient. An example might be asking a dementia patient how she celebrated Christmas last year, and the patient responding with a description of family traditions even though the previous Christmas was spent doing something entirely new and different. Unless the questioner actually knew what the patient had done that year, her answer would have sounded factual. An example of a semantically appropriate *personal plan* for the future might be asking a dementia patient what he intends to do tomorrow, and the dementia patient responding that he intends to give a piano lesson. This would sound perfectly reasonable to anyone who did not know that the patient had stopped playing the piano or giving piano lessons two years earlier.

Confabulation versus delusions

Patients with confabulation will sometimes cling to their false memories even when confronted with the truth or despite being aware of contradictory evidence. Accordingly, confabulation needs to be distinguished from other types of false ideas, most notably delusions.

There are at least superficial similarities between confabulation and delusions; e.g., both involve the production of unintentional false statements, both are resistant to contradictory evidence, and both have been shown to be influenced by emotion and motivation. Some researchers hold that confabulation is a form of delusion, when delusions are more broadly defined.

Others contend that a distinction can be made based on the context in which the two disorders occur (neurological vs. psychiatric) and the fact that confabulation is a memory-related phenomenon whereas delusion is a belief formation disorder. Several clinical characteristics distinguish the two, most notably which delusions tend to be more systematic and pervasive, whereas confabulations are more isolated, polythematic, and fleeting in nature. Recent papers have suggested that, if delusions have a memory component at all, it appears to be related to biased input, encoding, and integration of novel information.

Alzheimer's patients can exhibit delusions as well as confabulations. The delusions that are frequently observed in Alzheimer's patients include beliefs about theft; the patient's house not being his home, Capgras syndrome (someone close to the patient, such as a spouse, is an impostor), belief an intruder is in the house, abandonment, spousal infidelity, and paranoia.

An Alzheimer's patient who spontaneously confabulates forms a "memory" for an event that did not occur, but is merely consistent with current information. For example, the patient may not remember that she has rearranged the furniture in the living room. Upon seeing the furniture in locations that do not match her memory, she may create a new "memory" that involves someone breaking into the house and moving the items. This confabulation fades with time. A patient with delusions of theft, on the other hand, may become convinced that a particular individual has repeatedly robbed him, even in the absence of readily understood triggers such as a lost billfold.

WHAT CAUSES CONFABULATION?

To discuss what causes confabulation, one must first understand memory — at least, to the extent that memory is currently "understood" — and the ways in which Alzheimer's affects memory.

Memory in Alzheimer's

There are thought to be a number of different memory systems, including episodic memory, semantic memory, autonomic simple classical conditioning, motoric simple classical conditioning, procedural memory, perceptual priming, conceptual priming, and working memory. Each of these depends on different parts of the brain. Some of these memory systems are severely impaired in Alzheimer's patients, while others, such as procedural memory (cognitive and behavioral skills that operate at an automatic and unconscious level, such as learning to ride a bicycle or to play the piano) are relatively preserved — at least, in the earlier stages.

Here, the focus will be on episodic and semantic memory, since they are thought to be the most relevant to confabulating.

The medial temporal lobes (including the hippocampus, parahippocampus, presubiculum, subiculum, and amydala) constitute the core of the ***episodic memory*** system. The medial temporal lobes interact extensively with other brain regions (e.g., the anterior thalamus nucleus, mammillary bodies, fornix, and prefrontal cortex), some of which are associated with "Papez's circuit". A lesion in any one of these structures may cause the pattern of impairment that is characteristic of episodic memory dysfunction, i.e., the greatest disruption is in the ability to learn new information, moderate disruption is seen in the ability to recall recently learned information, and the ability to recall remotely learned information is generally intact.

How the medial temporal lobes *store* memories is not yet understood, but is thought to involve collecting and combining information from multiple cortical streams, such as the sights, sounds, smells, tastes, emotions, and thoughts during a given episode (e.g., eating a special holiday meal.) This information is transferred first to the parahippocampal region, then to the hippocampus proper, and then to the entorhinal cortex. It is processed in the dentate gyrus, and then transferred to the CA3 region of the hippocampus, where the critically important hippocampal index is assigned, allowing the memory to be stored in a unique way so that it can later be recalled.

Typically, memories are *retrieved* when a cue from the environment matches a part of the stored memory. For example, years later, the individual bites into a little cake that tastes remarkably like the one he had previously eaten at that special holiday meal. This sensory cue is transferred from the cortex to the parahippocampal region, the hippocampus, and then the entorhinal cortex. From there, it goes directly to the CA3 region where the original hippocampal index is found. The cue activates the hippocampal index associated with the index of the original stored memory — not the memory itself — in the CA3 region. This activation leads to the reinstatement of much of the neural pattern of activity associated with the original event in the CA1 region of the hippocampus, the subiculum and various cortical regions — leading to the experience of 'remembering' all of the sights, sounds, smells, tastes, emotions, and thoughts of the original holiday meal.

The hippocampus remains critical for memory retrieval until a process known as consolidation occurs. This process is still not understood, but it is thought that once a memory is consolidated, the distributed pattern of cortical neural activity is directly linked together, such that when a cue is encountered, the memory may be retrieved directly from cortical-cortical connections, without the need for the hippocampus.

In addition to the medial temporal lobes and Papez's circuit, the *frontal lobes* are also important for episodic memory. Whereas the medial temporal lobes are critical for the retention of information, the frontal lobes play key roles in the acquisition and encoding of information; retrieval of information

without contextual and other cues; recollection of the source of information; and assessment of the temporal sequence and recentness of events. One important reason why the frontal lobes are important for encoding episodic memory is that they enable the individual to focus his attention on the information to be remembered and to engage the medial temporal lobes. Dysfunction of the frontal lobes may cause a variety of memory problems, including distortions of episodic memory and false memories, such as when information becomes associated with the wrong context or incorrect specific details.

A simple analogy has been used to help conceptualize the differences between deficits in episodic memory that occur because of damage to the medial temporal lobes (and the Papez circuit) and those that occur because of damage to the frontal lobes. The frontal lobes are analogous to the "file clerk" of the episodic memory system, the medial temporal lobes to the "recent memory file cabinet," and other cortical regions to the "remote memory file cabinet". Thus, if the frontal lobes are impaired, it is difficult — but not impossible — to get information in and out of storage. However, the information may be distorted due to "improper filing" that leads to an inaccurate source, context, or sequence. Getting information into storage may require stronger encoding, and getting information out of storage may require stronger cues from the environment.

If, on the other hand, the medial temporal lobes are impaired, it may be impossible for recent information to be stored. This will often lead to the impaired person asking for the same information again and again and again. Older information that has been consolidated over months to years is likely stored in other cortical regions — the "remote memory file cabinet" — and will therefore be available for retrieval even when the medial temporal lobes or Papez's circuit are damaged.

Of the six major memory systems, episodic memory is the most clinically relevant for Alzheimer's patients. Very early in the course of the disease, there is prominent medial temporal lobe pathology and pathologic involvement of the lateral temporoparietal and medial parietal cortex, as well as a lesser (and more variable) degree of pathology in lateral and medial prefrontal cortex. These cause disruptions to the episodic memory system that are among the earliest signs and symptoms of Alzheimer's. Initially, they result in minor memory lapses such as misplaced keys, missed appointments and late bills that are brushed off as forgetfulness due to fatigue, distraction or "senior moments". More critical lapses, such as failing to remember whether the stove has been turned off or medications have been taken, often precipitate the initial visit to a doctor to diagnose the problem. Episodic memory deficits continue to represent one of the most significant functional problems as a patient progresses through the mild and moderate stages of Alzheimer's.

As mentioned above, damage to structures associated with episodic memory results in a characteristic pattern in which recent memories are more vulnerable to decay than remote memories. Accordingly, as episodic memory abilities continue to decline, events from the distant past are better remembered than events that occurred after or shortly before the onset of the disease. Vivid remote memories may sometimes be confused with psychotic delusions or hallucinations, such as claims to have recently seen

and interacted with a long-dead friend or family member. Inevitably, the inability to remember recent events or learn new information leads to functional deficits that are devastating.

One common assumption is that episodic memory is primarily or entirely concerned with the past. However, a growing number of investigators have begun to approach episodic memory in a broader context, one that emphasizes both the ability of individuals to re-experience episodes from the past and also imagine or pre-experience episodes that may occur in the ***future*** — e.g., planning and anticipation. Evidence for this close linkage of past and future events comes from studies of patients with episodic memory deficits. For example, studies on amnesia patients have found that they not only cannot consciously remember their pasts, but also cannot imagine their personal futures.

Neuroimaging has provided supportive evidence that the same regions of the brain are involved. For example, a recent neuroimaging study was designed to examine the neural regions involved in the construction (i.e., the search and reconstruction of a past event or the creation of a future event) and subsequent elaboration (i.e., retrieving or imagining supplementary details) of past and future events. The results showed that the left hippocampus and posterior visuospatial regions were involved in both past and future event construction. Elaboration was characterized by a remarkable overlap of activity in regions comprising the autobiographical memory retrieval network, including self-referential processing, contextual and episodic imagery. This striking neural overlap is consistent with findings that amnesic patients exhibit deficits in both past and future thinking, and confirms that the episodic system contributes importantly to imagining the future.

However, remembering the past and imagining the future differ, at least with respect to temporal orientation, and some unique cognitive processes and neural regions should therefore be associated with each. And indeed, this same study found that, during the construction phase, future events recruited regions involved in prospective thinking and generation processes, i.e., right frontopolar cortex and left ventrolateral prefrontal cortex, respectively; and also uniquely engaged the right hippocampus, possibly as a response to the novelty of these events. Unexpectedly, there was no evidence of any regions of the brain engaged uniquely by past events. Every region engaged by the construction and elaboration of past events was also engaged by future events either to a similar or significantly higher level.

Accordingly, it has been suggested that these results raise questions about the adaptive significance of the episodic system. Although the function of the episodic system is typically conceived of as retrieval of past events, it is possible that the primary role of this system is not reminiscence, but rather, future thinking. As such, the ability to retrieve episodic information would exist primarily for the purpose of simulating possible future scenarios and outcomes, and anticipating future needs. And, although I have not seen this suggested in any of the papers I've read so far, it also goes a long way toward explaining why so many of our loved ones seem so unafraid of the future … and also why they often see no need to

consult doctors, take medicines, or take other steps to try to slow down the progression of their dementia.

From this viewpoint, damage to the episodic "memory" system is even more clinically relevant for Alzheimer's patients.

Semantic memory involves memory for factual knowledge that has been learned, but for which specific "time and place" information about the source of the original experience is typically not known. Encyclopedic knowledge of information such as the features of objects (e.g., apples are usually red), categories (e.g., oranges and bananas are both types of fruit); historical events, mathematical tables, and similar types of information are considered to be stored in semantic memory systems of the brain. Semantic memories may also include "autobiographical" information, i.e., personal facts, such as one's place and date of birth, or the names of family members. Evidence that the semantic memory system is different from episodic memory comes from neuroimaging studies, and the fact that previously acquired semantic memory is spared in patients who have severe impairment of the episodic memory system, such as with disruption of the Papez circuit (e.g., in Korsakoff's syndrome) or surgical removal of the medial temporal lobes.

The anterior and inferolateral temporal lobes are important in the naming and categorization tasks by which semantic memory is usually assessed. However, in the broadest sense, semantic memory includes all of the person's knowledge of the world not related to any specific episodic memory. It could therefore be argued that semantic memory resides in multiple cortical areas throughout the brain. For example, there is evidence that visual images are stored in nearby visual association areas.

Alzheimer's is the most common clinical disorder that disrupts semantic memory. This disruption may be due to pathology in the anterior and inferolateral temporal lobes, and/or to pathology in the frontal cortex leading to poor activation and retrieval of semantic information.

In Alzheimer's disease, episodic and semantic memory decline independently of each other, supporting the idea that two separate memory systems are impaired in this disorder.

Interestingly, the content of autobiographical memories shifts from episodic to semantic with aging in healthy adults. Alzheimer's disease enhances this pattern. Very recent neuroimaging studies showed that, as hippocampal volume decreased in Alzheimer's patients, the left inferior frontal gyrus and the ventromedial prefrontal cortex (vmPFC) were activated. The linking function of the hippocampus is related to vivid, episodic retrieval and the linking function of the vmPFC is related to semantic retrieval. The researchers speculated that the linking function of the degraded hippocampus is taken over by the vmPFC, resulting in the shift to semantisation.

Not all dementias are alike in the memory systems that are affected. For example, patients with semantic dementia (the temporal variant of frontotemporal dementia) exhibit deficits in all functions of

semantic memory, such as naming, single-word comprehension, and impaired general knowledge (such as the color of common items) early in the course of the disease. Other aspects of cognition, however, are relatively preserved, including components of speech, perceptual and nonverbal problem-solving skills, and episodic memory.

Brain damage associated with confabulation in dementia patients

There is a general consensus of opinion that confabulation is the result of damage to the brain … but questions still remain as to what types of damage, and where, are needed for confabulation to occur. I get the distinct impression that one possible reason for the controversy is that the data on all types of brain damage, producing all types of confabulation, tend to be lumped together, which may confuse more than clarify. In particular, I wonder how much confabulation in Alzheimer's patients has in common with confabulation in patients who have sharply-localized damage to a single site in the brain. Alzheimer's is a multidomain disorder, including not only memory loss, but also executive dysfunction (e.g., impaired ability to plan ahead, prioritize, stop and start activities, shift from one activity to another activity, and to monitor one's own behavior) and varying degrees of visuospatial and language deficits.

Accordingly, I have done my best to focus on studies specifically done on Alzheimer's and related dementias — although it is often difficult to determine whether researchers derived some of their conclusions about Alzheimer's patients from studies on non-demented persons. Moreover, please note that a 2006 paper commented, "Previous studies that have investigated confabulation in AD have failed to take into account the characteristics of the disease…"

Studies that couple brain imaging techniques (e.g., MRI, PET, and SPECT) with tests on cognitive function (e.g., the MMSE and CASI*) have found that, in *early-stage* Alzheimer's patients tested about autobiographical memory (personal semantic and episodic information):

- Alzheimer's patients produce more confabulations in response to episodic than to personal semantic memory questions, probably reflecting the much more marked impairment of episodic memory characteristic of early-stage Alzheimer's.

- There is a correlation between the degree of cognitive impairment (defined by MMSE and CASI scores) and personal semantic confabulation, but not between cognitive impairment and episodic confabulation, or between cognitive impairment and temporality. (Remember, this is in early-stage Alzheimer's. Remote memory, including personal semantic memory, is relatively preserved in early-stage Alzheimer's.)

- Semantic confabulation (and MMSE and CASI scores) correlates with atrophy in the anterior cingulate, bilateral medial temporal, and right middle temporal gyrus.

- Confabulation scores for remembering the past and planning the future (i.e., confabulations involving the episodic memory system) are strongly correlated, and are higher in patients who also exhibit delusion and/or aggression.

- Lower blood flow ("hypoperfusion") in the frontal and/or temporal regions of the brain, including Brodmann's area 9 which plays a role in episodic memory retrieval, is associated with delusion in Alzheimer's patients. Lower metabolism ("hypometabolism") has also been reported in frontal or temporal regions of the brain in delusional Alzheimer's patients.

- Delusional Alzheimer's patients exhibit more episodic confabulations and have lower blood flow in the prefrontal cortex than nondelusional Alzheimer's patients.

From these studies, the following conclusions were drawn:

- Different mechanisms are involved in personal semantic and episodic confabulations in Alzheimer's patients.

- Episodic confabulation is affected by delusions related to prefrontal hypoperfusion.

- Semantic confabulation (at least, personal semantic confabulation) is associated with cognitive impairment.

[*The Cognitive Abilities Screening Instrument (CASI) includes subscales related to frontal lobe function -- i.e., attention and list-generating fluency -- as well as subscales related to concentration and mental manipulation, orientation, short-term memory, long-term memory, language, visual construction, and abstraction and judgment.]

Progressive neurodegenerative disorders such as Alzheimer's inevitably go on to develop damage in more and more regions of the brain; and so the characteristics of confabulation seen in early-stage dementia may be considerably different from those seen in later-stage patients. However, confabulation in later stage patients has rarely, if ever, been studied.

Possible mechanisms associated with confabulation

Researchers have come up with a number of hypotheses to explain why confabulators "remember" a past that is different from the "real" past. One of the earliest hypotheses was that confabulations occurred as a compensatory mechanism for memory loss (i.e., the patient produces confabulations to fill in memory gaps and avoid embarrassment). However, this hypothesis has been vigorously challenged and is no longer in favor. Current hypotheses fall into two main classes, i.e., temporality/source confusion, and strategic retrieval. (These hypotheses have been developed to cover all types of confabulations associated with all types of brain damage, even though it is becoming apparent that different types of confabulations are associated with different mechanisms. The majority of the studies

have focused on provoked confabulations, for the very simple reason that they are much easier to study. Personal experience leads me to believe that spontaneous confabulating may play a much more important role in the later stages of Alzheimer's, which apparently has never been studied.)

Temporality/source confusion hypotheses posit that confabulations are true memories displaced in time, caused by impaired search processes of the long-term memory storage system. Cues that are necessary for the retrieval of memories might match and activate stored experiences other than the episode that is sought. In the "temporality and consciousness" model, the three dimensions of temporality — past, present, and future — map onto three types of confabulation that are expressed in the context of past episodic memory, current time-place disorientation, and future plans, respectively. The model distinguishes a "knowing" consciousness (expressed in the form of habits and semantics) and a "temporal" consciousness (related to unique personal events, specified in time.) Studies have shown that dementia patients tend to retrieve habitual, generic, well-learned information and to mistake it for specific events. The model proposes that this tendency reflects a disruption in temporal consciousness, leading the patient to rely on "knowing" consciousness instead.

Strategic retrieval hypotheses suggest a general retrieval failure of which temporal confusion is a common symptom. They note that since confabulation can concern experiences encoded and stored before the onset of brain damage, it seems to be associated more with memory retrieval rather than memory encoding or storage difficulties. According to these models, when memories are not elicited directly or automatically by a cue, the target memory needs to be recovered through strategic search processes akin to problem solving. Strategic retrieval processes operate at *input* to frame the memory problem and initiate a search, to constrain the search, and to guide it toward local, proximal cues that can activate associative memory processes. Then, once a memory is recovered, strategic processes operate at *output* to monitor whether the recovered memory is consistent with the goals of the memory task and whether it is consistent with other knowledge, thereby verifying whether the recovered memory is likely true or false. Memory-related confabulation represents a failure of one or more of these strategic processes.

Proponents of strategic retrieval have suggested a pair of mental systems that can help explain the process of the acceptance or rejection of an experience or a memory as true. The first is an intuitive "feeling of rightness" (FOR) that allows healthy individuals to reject memories that simply do not make sense. FOR is impenetrable to rational influences, suggesting it is formed outside of conscious awareness and may be affectively (emotionally) laden. FOR is thought to be defective in people who confabulate. It precedes the second system, which is a more advanced system that cross references FOR-validated material with the body of previously accumulated knowledge. Because confabulators do not have good memory retrieval, this second system will not filter out incorrect statements. All the confabulators are left with is the feeling of rightness response. FOR is hypothesized to be one function of the ventromedial

PFC (vmPFC). Recall that neuroimaging studies showed that, as hippocampal volume decreased in Alzheimer's patients; the vmPFC was increasingly activated, resulting in a shift to semantisation.

Although some models favor a disruption of frontal/executive functions involved in the control of retrieval from long-term memory, this involvement is controversial. Several studies on Alzheimer's patients did not find any correlation between confabulation and performance on executive tasks, the ability to discriminate the origin of information, and/or working memory. Temporality models were developed, in part, in response to such findings. Similarly, studies comparing Alzheimer's and FTD patients failed to provide any evidence of a correlation between the performance on frontal/executive tasks and the tendency to confabulate. Patients with FTD were found to confabulate more on both episodic memory and personal future planning tasks than patients with Alzheimer's. It was suggested that "frontal executive" tasks are not able to discriminate between patients with primary frontal pathology (FTD patients) and patients with secondary frontal dysfunction (Alzheimer's patients); and that the reason FTD patients are more prone to confabulate may be due to a more severe disruption of personal temporality in FTD.

Another area of controversy surrounds whether and/or how ***emotional*** mechanisms influence the presence or the content of confabulations. Initial evidence that confabulations may reflect unintentional motivations and drives that are positively biased led some researchers to coin the terms "motivated" or "self-enhancing" confabulation. This was not seen as an exaggeration of psychological motivation *per se*. Instead, it was conceptualized as the direct outcome of reduced executive control over memory. When irrelevant memory representations are not inhibited and memories are not retrieved in an appropriate manner, motivational factors may acquire a greater role in determining which memories are selected for retrieval and accepted as true. One study concluded that confabulations about current circumstances showed the positive bias, whereas confabulations about the past did not. More recent studies have found that, while an emotional bias may exist, it is not necessarily a positive one. Patients with various memory disorders had an enhanced tendency to produce confabulations with affective content (pleasant and unpleasant), and the affective content appeared to relate, at least in part, to the patient's current mood-state.

The proposal that emotions might play a key role in the affective content of confabulations has received very little attention, even though it is consistent with what is now known about the anatomical basis of confabulating, notably its association with damage to key emotion-related structures in the medial frontal and anterior limbic areas. Memories with affective content (pleasant or unpleasant) are better remembered than neutral events. Emotional enhancement of episodic memory has been linked to the amygdala, a group of nuclei located in the medial temporal lobe. One hypothesis is that emotional arousal activates the amygdala and that such activation results in the modulation of memory storage or consolidation occurring in other brain regions, regulating the strength of memories in relation to their emotional significance. The amygdala is important in the creation of biases and in decision making. There

is extensive evidence that the amygdala is crucially involved in regulating stress effects on memory. Stress hormones and stress-activated neurotransmitters enhance the consolidation of memory for emotionally arousing experiences through actions involving the amygdala. Such amygdala activation strengthens the storage of different kinds of information through the amygdala's widespread network of efferent projections to other brain regions.

It is unlikely that regions of the brain that remain structurally intact are functionally insulated from the effects of damage elsewhere in the brain. In neurodegenerative diseases, such as Alzheimer's, disruption of the functional integrity within individual regions, such as the hippocampus and amygdala, as well as connections between these regions, influences the functional milieu of the remaining network. One consequence of this breakdown is a decline in performance, such as the changes seen on tests of short-term episodic memory. At the same time, there is the possibility of *compensatory* changes that allow performance to remain above what would be expected given the extent of structural damage to the hippocampus and related regions. For example, the amygdala is not normally recruited in healthy older adults to any greater extent during memory tasks. A recent PET study of Alzheimer's patients engaged in a delayed match-to-sample face recognition task, however, revealed that increased activity within the amygdala was found to be associated with better task performance during longer memory delays. Memory networks may be redirected towards the more primitive circuitry involving the amygdala and its connectivity with related brain structures as a buffer for episodic memory decline following degeneration of other medial temporal lobe regions, even though the amygdala as well as the hippocampus frequently undergoes significant pathological changes in the early stages of Alzheimer's.

HOW COMMON IS CONFABULATION IN DEMENTIA PATIENTS?

Several papers written by researchers who study confabulation commented that confabulation is "often" seen in dementia patients, but I did not find any papers which specifically studied the prevalence of confabulation in Alzheimer's. What I did find was a handful of studies which compared the frequency with which various types of confabulations were observed in different dementias.

For example, one study compared Alzheimer's and FTD patients using questions about temporality (personal past, orientation, and future planning). Patients in both groups had mild dementia, and were equally impaired on tests of executive function. Both groups confabulated across all three categories of confabulation, and produced significantly more confabulations in episodic memory than in semantic memory or personal future. However, FTD patients confabulated significantly more than Alzheimer's patients on both episodic memory and personal future. The dementia groups produced fewer correct responses on the confabulation battery in comparison with normal controls, confirming that both a memory impairment and a deficit in personal future planning tasks are a part of the clinical picture of both Alzheimer's and FTD. Interestingly, although FTD patients confabulated more frequently than Alzheimer's patients on episodic memory and personal future, there was no evidence that there were

any qualitative differences (i.e., differences in the content of the confabulations) between the two groups. Both patient groups produced the same type of "semantically appropriate confabulation".

A case history suggested that at least some patients with behavioral variant frontotemporal dementia (bvFTD) may actually be demonstrating "fantastic thinking" (vividly experienced imagination) rather than delusions or spontaneous confabulations, and actually be aware that the false statements are imaginary.

One study compared Alzheimer's with Lewy body dementia (LBD). The Alzheimer's and LBD groups of patients were matched for age, illness duration, nature and severity of cognitive deficits, and regional blood flow distribution on SPECT. Confabulatory responses (as well as inattention, visual distractibility, impairments in establishing and shifting mental set, incoherence, perseveration, and intrusions) were significantly more common in LBD than in Alzheimer's. However, it appears that the LBD group included patients who actually had Parkinson's disease dementia (PDD) rather than LBD per se. When the data from these PDD patients were removed from the comparisons, the pattern of findings remained essentially unchanged, except that the differences in confabulation and memory interference no longer reached significance.

Another reported that confabulation is rare in patients who have MCI. (Note, please, that MCI is not a dementia *per se*, although about a third of patients with MCI do go on to develop a dementia.) There did not appear to be any differences in the prevalence of confabulating among patients diagnosed with amnestic MCI (deficits in memory), non-amnestic MCI (cognitive impairment is restricted to non-memory domains) and multiple MCI (both memory and non-memory domains of cognition are impaired).

What I found increasingly bizarre, the more I read, is that researchers who study confabulation don't seem to be aware that dementia patients exhibit spontaneous confabulation. Confabulation is often described as an "infrequent disorder". Spontaneous confabulation is repeatedly described as "rare", in comparison with provoked confabulation. And yet, Alzheimer's is now the sixth leading cause of death in the United States … and surely all of those who die from Alzheimer's had exhibited spontaneous confabulation for quite some time before they died.

Statements by researchers who study confabulation that seem wildly incongruous to an Alzheimer's caregiver:

"Most patients with spontaneous confabulation eventually stop confabulating."

"Confabulators may occasionally act upon their confabulation." ("Occasionally"? Later-stage Alzheimer's patients persistently and repeatedly act upon the belief their childhood memories are relevant to their present circumstances.)

"Confabulations are usually limited in time; they relate to the recent past, the present, and the future."

In sharp contrast, a recent study conducted by *dementia* experts to identify patterns of symptoms that can be used to diagnose dementias — i.e., a study involving patients who are still in the early stages of dementia –found that *spontaneous* confabulation was a symptom in roughly a third of patients with Alzheimer's or mixed dementia, and a quarter of patients with vascular dementia. Earlier studies had found that spontaneous confabulation is significantly more common in FTD than other dementias, although this particular study only observed it in ~15% of FTD patients.

WHAT CAN BE DONE TO TREAT OR MINIMIZE CONFABULATION?

There is very little mention in the scientific literature of attempting to "treat" or minimize confabulations, even for patients with non-degenerative disorders. One recent paper commented, "Few reports of rehabilitation or management of confabulation exist in the literature, possibly because it is so difficult. Asking the patient to use a stricter criterion at retrieval, or to reason through a series of logical steps to see that his confabulation cannot be true, may do little to convince him, especially in the long run."

Some researchers have had modest success asking the (non-neurodegenerative) patient to keep a "memory book" in which all activities are recorded, with date and time, and used as an aid in temporal and spatial reorientation. This may, in fact, be helpful in minimizing spontaneous confabulations on which early-stage Alzheimer's patients act, since members of discussion forums have reported relying on similar types of memory devices to help them maintain their independence. Other measures to help the Alzheimer's loved one remain oriented with respect to time include, e.g., clocks that show the day and date as well as the time, with a face large enough that all information is readily seen; a calendar to keep track of time and to remember important dates, kept it where it will be seen often; a newspaper delivered daily, and develop the habit of comparing its date with the calendar. Note, please, that orientation is not something to be forced on the loved one but, rather, something to be offered, to the extent the loved one can understand and appreciate it.

One study on early-stage Alzheimer's patients confirmed that poor encoding can play a significant role in provoked confabulations. My take-away lesson is that distractions must be eliminated, to the greatest extent possible, when attempting to communicate with an Alzheimer's patient. Turn off the TV and radio, make sure the loved one is focused on you, and do not expect (or allow) the loved one to try to do anything else while the two of you are talking. This may help minimize provoked confabulations … in the earlier stages.

While struggling to find hints helpful for the Alzheimer's caregiver, I was struck by the discussion in one paper (Schnider 2003) that began, "Spontaneous confabulation is a pervasive disorder that represents a great challenge to any rehabilitation team." The patients under consideration had suffered non-

degenerative brain damage of some sort, and the general approach was to hope that the symptom would eventually resolve itself, and to support the patient in the meantime. The authors went on to say, "Early clinicians proposed avoiding memory training — such as repeated questions about orientation — with patients and engaging them in common everyday activities, accepting their false interpretation of reality as much as possible. Our studies support such an approach. Knowing that any cue can activate a memory and provoke a presently inappropriate action, patients should receive information about their hospitalization, but their false ideas about current reality should not constantly be corrected."

This is, of course, advice that Alzheimer's caregivers frequently share with each other.

In fact, the more I read this paper — one of the very few that exist on spontaneous confabulation — the more I thought I recognized my husband (stage 6 Alzheimer's), as well as the loved ones of many caregivers who frequents discussion forums.

And so, I finally decided that, although I don't recall having read any discussion of spontaneous confabulation in the Alzheimer's patient … that is exactly what our loved ones do, more and more, as the disease progresses and they live more and more in the past. And while *provoked* confabulations are a major annoyance in the early stages — when friends, family, and the medical community take everything our loved ones say at face value, no matter how false we know their statements to be — *spontaneous* confabulations become a far greater concern in the later stages, because spontaneous confabulations are much more likely to be acted upon by the loved one.

Approaches that can be used to cope with spontaneous confabulation, and ease the confusion, frustration, and fear for the loved one, can be found in resources such as:

Jennifer Ghent-Fuller's paper "Understanding the Dementia Experience"

Jolene Brackey's book, *Creating Moments of Joy*

Naomi Feil's "validation therapy"

The Savvy Caregiver training program

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Alzheimer's

Layers of memories separated in time
Photographic double exposures

You are you
but also
a long dead sister

or a half-remembered husband
maybe partially a grandkid

Or maybe you are mostly
the long dead sister.

She is at home
or maybe in another place –
Her last home or
one from many years ago

She is a child
or there are children
to be taken care of
One of them may be you

A Kaleidoscope of images
from whole lives
Jumbled together

~ Anonymous Caregiver

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