10 Confabulations about Personal Memories, Normal and Abnormal

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Certain types of neurological patients, and normal people in certain situations, will confabulate—sincerely claim to remember events that did not actually happen. The word *confabulate* is derived from the Latin verb *confabulari,* meaning "to talk with," derived in turn from the Latin noun *fabula*, meaning "tale" or "fable." It was perhaps this original root that the German neurologists Karl Bonhoeffer, Arnold Pick, and Carl Wernicke had in mind when they began referring to false memory reports made by their amnesic patients as "*Konfabulationen.*" Most of these patients suffered from what later came to be known as "Korsakoff's syndrome." When asked what they did the day before, typical Korsakoff's patients have no memory at all but, instead of admitting ignorance, will confidently report events that either did not happen (at least not to them) or happened to them long ago. A male patient, for example, might claim that he was finishing up the year-end inventory at his supermarket, when in fact he had been in bed at the hospital the whole time. This chapter examines two neurological syndromes that gave rise to the concept of confabulation: Korsakoff's syndrome and aneurysm of the anterior communicating artery. These syndromes will then be compared with false memory syndrome, which can affect normal children and adults. The connections between the neurological phenomenon of confabulation and normal memory errors can provide insights into the complex functions of memory.

A full understanding of confabulation in both normal people and neurological patients will require contributions from psychology, neuroscience, and philosophy as well as classical neurology. Recently, the psychological investigation of the functional dynamics of memory has merged with the neuroscientific investigation of the brain processes responsible for them in the new field of cognitive neuropsychology. Philosophy

can also play a helpful role here in several ways. Epistemology, the philosophical investigation of knowledge, contains detailed theories of what formally constitutes knowledge and how to assess knowledge claims. Confabulations about memories are flawed knowledge claims generated by brain processes that are malfunctioning (temporarily in the case of normal people; chronically in the neurological patients). There is also a long tradition in philosophical thought, dating back at least to Locke, on the relationship between our memory and our identity over time. Confabulation may be an attempt to maintain a coherent identity over time by linking our current self to previous actions or events, to present our self to others as a unified being, aware of and responsible for our past actions.

Implicit and Explicit Memory

The brain's many memory systems can be divided into two main types: implicit and explicit. Explicit memory presents information to consciousness in the form of thoughts or images, whereas implicit memory largely bypasses consciousness. Procedural memory, a type of implicit memory, allows us to acquire skills, such as how to play the piano or ski. It functions largely without consciousness; indeed, conscious awareness can interfere with its workings, as in the case of the trick sometimes played on fellow golfers: asking them whether they inhale or exhale when they swing. Simply considering the answer can cause the intricate pattern of muscle activations to fall completely apart. Classical conditioning, of the type discovered by Pavlov, is also a form of implicit memory. The focus here will be on a type of explicit memory known as "episodic" or "autobiographical memory."

Can you remember what you had for breakfast this morning? To do this, you need to employ your *autobiographical memory*. A record of our personal experiences, usually from our point of view, autobiographical memory is fragmentary—we can forget whole hours, days, weeks, and even years. It is an especially individual form of memory, not only because it records most indelibly those things of greatest importance to us, but also because losing it means losing a sense of our self, as anyone who has ever watched someone succumb to Alzheimer's can testify. The Alzheimer's patient eventually forgets you and may claim that you are someone else, or a stranger. This calls to mind another function of autobiographical memory: it records

information about other people, places, and things that are significant to us and thus allows us to build lasting social relationships.

With some of the things we know, the knowledge of when and where we first acquired that information is long gone. We know that cats have claws, but we most likely have no idea when or where we learned this. Other information brings with it what researchers call "source memory": a type of episodic memory about when and where a memory was acquired. Source memory is a fragile thing, and we are all prone to characteristic errors in source memory tasks. We may remember an interesting medical fact but misremember which television program we learned it from, or we may remember a mildly interesting piece of gossip, but misremember whom we learned it from. The prefrontal lobes are important for retrieving source memories. In one study, normal people and hospital patients with frontal cortical lesions learned the answers to a set of trivia questions. When they were tested a week later, the frontal patients had normal memory of the answers themselves, but showed poor source memory, often claiming they had learned the answer at some earlier point in life.[1]

Another type of explicit memory, one that most often comes without a source memory tag, is called "semantic memory" and involves knowledge of impersonal facts, such as that the Eiffel Tower is in Paris, that Truman was a U.S. president, and so on. Thus far, researchers have been unable to clearly separate the neural loci of semantic and episodic memory, and perhaps for good reason: the two memory systems interact in several ways, and some have suggested they are merely different levels of categorization in the same memory store.[2] Autobiographical memories do seem to aggregate into semantic memories, as when we learn on several occasions that Lincoln gave the Gettysburg Address. We forget the occasions, but remember the core fact. Semantic and autobiographical memories would also need to interact when confabulations are produced about autobiographical events that also involve semantic knowledge, such as a "memory" of being present at some historical event. Such interactions might lead to wholesale changes in the memory system and the creation of a type of fictional autobiography, complete with surrounding (fictional) history.

Autobiographical memory is a function of the medial temporal lobe memory system, which includes the hippocampus and the adjacent parahippocampal and perirhinal cortices. The hippocampus is not where the content itself of memories is stored, but rather appears to contain a set of

neural links to the content, which is distributed widely throughout the cortex. Memories of an episode in our life typically contain information from more than one modality: sight, hearing, and even taste, touch, and smell. Each of these components is stored in a unimodal sensory area; for example, the visual components of an episodic memory are stored in the visual cortices in the occipital and inferior temporal lobes whereas the auditory components are stored in the auditory cortex in the superior temporal lobes. These distributed representations are linked to a central index in the hippocampus. When recent episodes are retrieved, the index is reactivated, causing activation to spread to each of the associated unimodal areas. Once a representation of an episode has been fully consolidated, activation can spread between the separate features themselves, so that hippocampal activation is no longer needed.

Neuroscientists are beginning to understand which brain areas make up the frontal components of the medial temporal lobe memory system. The medial temporal and hippocampal regions tend to be more involved in spatial context memory, whereas the frontocortical region, the diencephalon, and the temporal lobes are involved in temporal context memory. Much has also been learned about the neural bases of short-term memory systems located in the frontal lobes. Psychologists have not been able, however, to determine whether there is one type of short-term memory or several and exactly what time span is involved, although "short term" is typically thought to mean several seconds. In the 1980s, neuroscientists began exploring a large area in the dorsolateral portion of the prefrontal lobes that seems to be responsible for what has been called "working memory," which at least overlaps with the psychologist's concept of short-term memory.[3] This prefrontal area appears to monitor and manipulate representations contained in posterior cortical areas.[4]

In the late 1950s, surgeons removed much of a man's temporal lobes (including most of the hippocampus, the parahippocampal gyrus, and the amygdala) in an attempt to reduce the severity of his epileptic seizures.[5] This patient, known as "HM," developed a severe amnesia for autobiographical events, but retained his basic intelligence and his personality. Researchers also observed that HM could retain information for a short time and could also acquire new motor skills such as mirror writing, solving puzzles, or tracing mazes, without knowing that he was doing so, a form of procedural memory.

Korsakoff's Syndrome

Confabulation was among the symptoms Sergei Korsakoff observed in a group of alcoholic patients he was treating in 1887. Other symptoms included memory loss, anxiety, fear, depression, and general irritability. It has since been learned that the syndrome is caused by a lack of vitamin B_1, or thiamine, and not directly by alcohol itself. Korsakoff's syndrome can come on quickly, after an alcoholic coma, or it can progress slowly over many years. And although it occurs primarily in alcoholics, it may also occur in nonalcoholic patients whose digestive systems fail to absorb B_1 for other reasons (malabsorption syndrome, regional enteritis, cancer of the stomach).[6] Alcohol is known to interfere with transport of thiamine in the gastrointestinal tract, and chronic liver disease, a common consequence of alcoholism, can affect the liver's ability to store thiamine. Because chemicals derived from it are involved in the synthesis of neurotransmitters, particularly acetylcholine, as well as GABA, thiamine plays an important role in the proper functioning of the brain.

Memory loss in Korsakoff's is anterograde—patients are unable to form new memories. As with HM, their procedural memories are left intact (they can still drive a car, for example). Korsakoff's patients tend to underestimate both the time they have spent in the hospital and their own ages. Korsakoff himself successfully traced the memory reports of his patients to actual experiences but found that the memories had been displaced in time by the patients. In the early phase of their illness, the confabulations of Korsakoff's patients are typically internally consistent accounts about themselves. The contents of these accounts are drawn fully or principally from the patients' recollections of their actual experiences, including their thoughts in the past.

Aneurysm of the Anterior Communicating Artery

Confabulation can also result from aneurysm of a critical brain artery—the anterior communicating artery (ACoA), which distributes blood to portions of the ventromedial lobe (including parts of the orbitofrontal lobes) and related structures. Aneurysms occur when the walls of blood vessels, weakened by infection or degenerative illness, bulge abnormally. Unless properly treated, the aneurysm may rupture, causing a hemorrhage and

destruction of the surrounding tissue. Although small, the anterior communicating artery feeds a variety of brain areas and organs (portions of the ventromedial frontal lobes, the basal forebrain, fornix, septum, anterior cingulate gyrus, and corpus callosum), and damage to it may also seriously affect blood flow in one or both of the anterior cerebral arteries.

The important cognitive features of the classical ACoA syndrome are:

1. *Memory loss* Patients show both anterograde and retrograde amnesia, the latter often for several years preceding the aneurysm. As in Korsakoff's, short-term memory appears to be intact. In tests of recognition memory, patients can often correctly recognize famous people, for example, at a normal level, but they can exhibit something called "pathological false recognition," that is, cases where they claim to recognize a stimulus they are actually seeing for the first time.

2. *Changes in personality* Like Korsakoff's patients, ACoA aneurysm patients undergo personality changes—manifested as impulsivity, impatience, disinhibition, emotional lability, depression, problems in decision making, and poor judgment in social situations—that compromise their ability to socially interact.

3. *Executive deficits* These include perseveration, poor concept formation, problems with set shifting, reduced verbal fluency, and impairments in cognitive estimation.

4. *Confabulation* Appearing as implausible and "spontaneous" in the acute phase right after the aneurysm, confabulation quite often persists, only as more plausible and "provoked," in the chronic phase.[7]

The memory deficits caused by aneurysm of the anterior communicating artery and by lifelong drinking in Korsakoff's syndrome hold a special interest for memory researchers, indicating as they do important frontal components to the memory system. Neuroanatomists have confirmed that the areas constituting the medial temporal lobe memory system have strong, reciprocal connections to at least two frontal areas. The sites of lesion in Korsakoff's and ACoA amnesia are clearly different from those involved in medial temporal lobe amnesia. There are corresponding differences between the temporal and frontal amnesic patients, the most important being that medial temporal lobe amnesics do not confabulate, will admit their memory deficits, and will pursue compensatory strategies. Indeed, medial temporal lobe amnesics have been found to be *less* likely

than normal people to produce false memories on tasks specifically designed to elicit them (see below). They show much higher latencies in giving their answers and make many more self-corrections than confabulating frontal memory patients in memory tasks, which suggests that their intact executive processes are struggling to correct degraded memories.

False Memories in Normal People

Many of the memory and confabulation problems seen in neurological patients are simply extreme versions of those affecting all of us every day. We sometimes remember what we intended to say or do, rather than what we actually said or did. We frequently displace events in time upon recalling them. And we mistake events that we merely dreamed of for real events or, less often, vice versa. Recent trends in memory research have strongly confirmed what memory researchers have always known, that memorizing something is not at all like recording it and that recalling something is not at all like replaying a recording. Memory is a selective and reconstructive process, which can go wrong in several ways.

The phrase "false memory" is somewhat of a contradiction in terms, given that we cannot be said to truly remember something that never happened, but the phrase's meaning is clear enough. False memories can easily be produced in children by asking them leading questions. In one telling experiment, children were presented with a deck of cards, each of which described an event. Some of the events had actually happened to the children, whereas others had not. When they were repeatedly asked whether the false events had happened to them, a majority of the children eventually agreed that they had, and many of them embellished the events with confabulated details.[8] Apparently, our memory systems have a baseline accuracy level, and we use different frontal checking procedures to increase this level. As already noted, our normal correct memories are rational reconstructions, in that the reconstruction process is guided by what seems rational to us. This can be seen in certain patterns of error in false memories, where we misremember something odd in an event as something more normal or rational.

It is interesting that young children exhibit some of the same memory problems that frontal patients show. This may be because the frontal lobes are among the last cortical areas to mature. Most of the development of

the frontal lobes occurs between ages 5 and 10, and they do not fully mature until the teenage years. Perhaps nature's plan is that the checking processes described above will be instilled after birth, during the long training period we humans require, principally by our parents. What begins as an external loop is made internal: children naturally confabulate, parents correct, and the children change what they said. As we mature, we internalize these corrections, so that the loop runs completely within our brains, although it shares some of the same dynamics: there is still a candidate claim, and there is still a check that has the power to correct the claim or inhibit it from being made.

Adults are also prone to false memories in certain circumstances. The "misinformation effect" is a way to induce false memories in adults in laboratory settings. In a typical experiment, subjects will be first shown a video depicting a staged crime, and then exposed to false information designed to interfere with their memories of the event. When asked later to recount the event, subjects show a strong tendency to incorporate this false information. A number of researchers including Elizabeth Loftus and colleagues have also shown that exposure to prejudicial information after having witnessed an event can influence the subject's later recall of that event.[9] Maryanne Garry's research group has found that both imagining events that never happened and paraphrasing descriptions of such events can make us more likely to later report that those events actually happened.[10] In another type of experiment, normal subjects were presented with a list of words related to sleep, excluding the word *sleep* itself: *bed, rest, awake, tired, dream, wake, snooze,* and so on. When they were later tested, between 30% and 40% of the subjects claimed that they had seen the word *sleep*.[11] Researchers who observed the brains of normal subjects using PET as they performed tasks in which they first heard a list of related words, then were tested for memory of the words were able to successfully differentiate correct from incorrect memories by their different patterns of activation.[12] Subjects of hypnosis may also confabulate when they are asked to recall information associated with crimes, causing researchers to warn criminologists about the dangers of obtaining information from hypnotized subjects. There are also anecdotal reports of hypnotized subjects confabulating when asked why they did something in accord with their hypnotic suggestion. For instance, a hypnotized man is given the suggestion that he will wave his hands whenever he hears the word *money*.

When asked later why is he is waving his hands, he replies, "Oh, I just felt like stretching."

Studying patients with brain damage may be an easier route to understanding confabulation than studying normal people since the patients' site of damage and the known functions of that area provide an obvious starting point. When resulting from frontal brain injuries (such as aneurysms or strokes), confabulations are about past events in patients' lives that either did not happen (or not to them) or did not happen to them when the patients believe they did. With the increasing information available about how our memory systems work, the discussion of memory-based confabulation has grown increasingly sophisticated. One theme of great interest that comes up frequently in the literature is that these types of confabulations might be caused by two separate malfunctions. First, frontal lobe patients have a memory problem, which they share with medial temporal lobe patients. And second, the patients have what is typically referred to as an "executive problem," which is responsible for the failure to realize that the memories they are reporting are fictitious. In a particular case of confabulation, the two problems manifest as two *phases*: first, a false memory is produced, but then, frontal areas fail to perform functions that would allow the person to realize the falsity of the memory. This succession implies that the thoughts that give rise to confabulations exist as genuine beliefs in the patients' mind, as opposed to the patients merely finding certain claims coming out of their mouths, without their actually believing them. It seems, therefore, that the patients' confabulations are accurately reporting their (disordered or ill-grounded) conscious experiences.

We can now turn to a definition of *confabulation,* which involves six individually testable criteria. A subject (S) confabulates in claiming that a proposition (p) is true if and only if (1) S claims that p is true; (2) S thinks that p is true; (3) S's thought that p is true is ill-grounded; (4) S does not know that S's thought is ill-grounded; (5) S should know that S's thought is ill-grounded; and (6) S is confident that p is true.[13] The concept of *claiming* (rather than, for instance, *saying* or *asserting*) is broad enough to cover a wide variety of responses by subjects, including nonverbal responses, such as drawing and pointing. The second criterion captures the sincerity of confabulators. If explicitly asked, "Do you think that p is true?" they invariably answer yes. The third criterion refers to the problem

that caused the flawed response to be generated: processes within the relevant knowledge domain were malfunctioning. The fourth criterion refers to a cognitive failure at a second executive phase, the failure to check and reject the flawed response. The fifth criterion captures a normative element in our concept of confabulation: if the confabulator's brain were functioning properly, the confabulator would know that the claim is ill-grounded, and not make it. The claims made are about things any normal person would easily get right. The sixth and last criterion refers to another important characteristic of confabulators observed in the clinic, the serene certainty they have in their claims, even in the face of obvious disbelief by their listeners. This epistemic approach eliminates a problem endemic to the falsity criterion in the original definition, proposed by memory researchers such as Korsakoff, according to which confabulations are false memory reports: Subjects might answer correctly out of luck. The problem is not so much the falsity of the subjects' claims but rather their ill-groundedness and consequent unreliability, at least in the affected domain, for example, autobiographical memory. In short then, in this epistemic view, to confabulate is to confidently make an ill-grounded claim that we should, but do not, know is ill grounded.

Theories about memory confabulation divide into two categories, depending on which of the two problems is emphasized:

1. *Retrieval theories* Confabulation is caused by a deficit in the "strategic retrieval" of memories, which in turn causes a loss of our sense of the temporal order of our memories, and of their *sources*—the places and times they represent. Theories of this type can be traced all the way back to Korsakoff.

2. *Executive theories* Confabulating amnesics are to be differentiated from their nonconfabulating counterparts by their additional frontal damage. Confabulation reflects two different damaged processes: a memory process and an executive or "monitoring" process. The executive process fails to correct the false memory.

Cognition requires both representations and executive processes for manipulating those representations. Representations are expensive to produce, update, and maintain. Their primary purpose is to allow us to understand and affect the things they represent. Executive processes in the brain's prefrontal lobes perform different operations on our

representations when we decide, weigh, reason, infer, examine, resolve—processes we commonly call by the collective name "thinking." Our memory is itself an immense collection of representations. Executive processes, typically centered in the prefrontal lobes, must control the search and reconstruction processes that take place when we remember. As an example of an executive theory, Marcia Johnson attributes confabulation to a deficit in a more general executive function she calls "reality monitoring," the ability to distinguish real from imagined events.[14] Normal people are able to differentiate real from imagined information at high rates of success. This seems to be a learned, or at least a developed, ability. Real memories, according to Johnson, can often be distinguished from mere imaginings by the amount of perceptual detail they contain and by the presence of supporting memories—source memories—about where and when the remembered event occurred. It may be, however, that retrieval theories and executive theories are merely directed at different parts of the confabulation process, whose first phase involves the production of a false memory, and whose second phase involves failure to notice and correct the falsity. Retrieval theories focus on the failure to access the correct memories; executive theories, on the failure to correct false ones. Executive theorists typically attribute confabulation to a failure in what they call "self-monitoring" or "self-awareness."

Reality Monitoring

Confabulation may be due to a broader failure to test representations, whether they are from memory or not. According to Johnson, episodic memories of an event bind together elements of several different types, some of which represent impersonal features of the event, while others represent personal features, for example, our thoughts or emotions in reaction to witnessing the event. These different features include colors, sounds, tastes, emotions, objects, and locations, as well as information contained in semantic memory. Recall of any one of these features is often enough to draw the entire autobiographical memory back into our awareness. When thoughts presenting themselves as memories are so rich in detail often they are regarded as being genuine. Because of this, if we have a vivid and detailed imagination, we can mistake memories of our

imaginings for memories of actual events, for example, when we believe we did something we only imagined doing.

In reality monitoring, there are further checks we can make to separate real from imagined memories. We can check the consistency of the candidate memory with our set of beliefs, noting any inconsistencies between representations currently in our consciousness or between those and our long-term knowledge. Confabulation patients tend not to notice or worry when they contradict themselves. One male patient, for example, contradicted himself in the same sentence; saying first that he had just visited a store he formerly owned, then that the store no longer existed. As early as 1915, Arnold Pick noted that Korsakoff's patients also feel no need to correct their contradictions.

We can intentionally tighten our monitoring standards when motivated to do so. Researchers often report that simply admonishing memory patients to be more careful can work to increase the accuracy level of their reported memories. It is interesting to note that we tend not to consciously or intentionally loosen our standards; rather, we do so unconsciously and spontaneously. Johnson and her colleagues distinguish between *heuristic* checking of candidate memories, which usually operates automatically when we are remembering, and *systematic* checking, which is intentional. Heuristic processing consists of fewer component processes and uses readily available information, such as familiarity, perceptual detail, and schemas (e.g., world knowledge, stereotypes), typically activated by a cue. Systematic processing is made up of more component processes and may also involve the retrieval of other memories and knowledge that are not initially activated.

Systematic processing requires selective attention: we must explicitly attend to the candidate memory. It also includes self-provided memory cues. We often cue our own memories: when we want to remember someone's name, for example, we may imagine that person's face, producing a cue for our memory system to use in retrieving the name. We can then monitor any representations the cue gives rise to. We may need to use other information to reject candidate names that come up. Often, we may need to use this cuing process several times to reconstruct the memory correctly. As to the neural locus of these monitoring processes, researchers point to bifrontal areas.

The Suppression of Irrelevant Memories

Armin Schnider's research group has advanced a similar hypothesis: memory confabulation results from the orbitofrontal cortex and its limbic connections not performing their function of suppressing or inhibiting recalled memories irrelevant to the current task.[15] Schnider and colleagues argue that the posterior medial orbitofrontal cortex sorts out the mental associations that pertain to ongoing reality by suppressing memory traces that have no current relevance. Schnider claims that lesion of an orbitofrontal-mediodorsal-amygdala circuit produces spontaneous confabulation, which appears to emanate from interruption of the loop connecting the posterior orbitofrontal cortex directly and indirectly (via the mediodorsal thalamus) with the amygdala. Connecting confabulation in ACoA patients with that found in Korsakoff's patients, Schnider points out that the basal forebrain lesions seen in the former group often include damage to the posterior medial orbitofrontal cortex, present in the latter. Schnider's localization is supported by two of his findings. First, patients with lesions involving the posterior medial orbitofrontal cortex and basal forebrain confabulate for much longer periods (several months) than patients with anterior medial orbitofrontal lesions. And, second, there is posterior medial orbitofrontal cortex activation in normal subjects performing a memory task that requires them to carefully separate relevant from similar but irrelevant memories.

If memory confabulation results from two independent lesions, this indicates that there are two types of patients:

1. Those who sustained the memory system lesion first. Such patients should admit their memory deficit until the executive deficit develops, at which point he should deny it and commence confabulating.

2. Those who sustained the executive system lesion first. The course of the disease among such patients may be rather subtle. We also need to allow for people who simply do not develop the executive processes needed to check memory reports——who make do with their memories alone and tolerate a high rate of errors. Thus some Korsakoff's patients are confabulatory, having lost the ability to check their thoughts or candidate memories, *before* losing their memory. Their deficit may pass unnoticed

because they are substantially correct in what they say. But once the amnesia sets in, the problem becomes painfully obvious.

The brain's many types of different memory systems testify to the value evolutionary development places on learning from the past. Several questions remain. Do memory confabulations belong to the larger set of completion phenomena, such as the filling in of the visual blind spot? The brain's executive processes, located in the prefrontal lobes, require clear, unambiguous information in order to achieve their primary task, the creation of effective actions. We typically do not have the time to spend examining gaps in our perceptions and memories. Quite often in real life, when memories occur, we make a quick plausibility check, sort out any obvious contradictions or impossibilities in the memory, and move forward with the belief that the memory is correct. Thus confabulation might be seen as a type of completion phenomenon occurring at a higher social level. We respond to a question about our past with a coherent, reasonable answer, in order to create a coherent, gap-free account of our own life, and present it to others.

The philosopher Daniel Dennett sees confabulation as a type of self-creating activity, in which our confabulations—stories—tend to depict us in favorable ways. Taken together, they constitute a narrative we create and tell to others, about the sort of person we are.[16] Typically the stories that make up this narrative depict us as intelligent, in command of the situation and its relevant facts, and fully aware of the reasons and intentions behind our actions. The stories are all about the same person, the one referred to with that special word *I*. But if we collect all the I-claims, do we find a unified brain system playing a crucial causal role in the making such claims? Perhaps not, since some of the claims will be about our bodies, some about our current actions, others about our past experiences, while still others will be about our semantic knowledge of ourselves. Each of these types of knowledge is accomplished by a different brain system. This can tend to make our sense of self look as if it is produced by a motley collection of processes, cobbled together for various motives and conveniences, and then—protected and patched up by confabulation—presented to others.

Confabulation may be telling us something important about the human mind and about human nature. The ability to create narratives and the ability to check them for truth or at least plausibility seem to be separate

in the human brain—confabulatory patients retain the first ability, but have lost the second. One of the characters in their inner dialogue has fallen silent, and the other prattles on unchecked. Without this second character, however, they have lost the ability to admit ignorance. We see mild versions of this in normal people. We are all familiar with people who seem unable to say, "I don't know," and who will quickly produce some sort of plausible-sounding response to whatever they are asked. A friend once described such a person as "a know-it-all who doesn't know anything."

Those who have lost both their memories and their awareness that they once possessed those memories are untroubled by the loss and move forward in life with what mental abilities remain. They may confabulate when asked about their pasts, and these confabulations are satisfying to them—but not to their friends, relatives, and doctors. Each false claim they make causes us to doubt whether they can continue to relate to us in a normal way. Perhaps one reason why clinical confabulation is so fascinating is that we see a bit of ourselves in the neurological patients. We are aware, at some level, that the difference between us and them is only a matter of degree.

Notes

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13. William Hirstein, *Brain Fiction: Self-Deception and the Riddle of Confabulation.* (Cambridge, MA: MIT Press, 2005). See chapter 8.

14. See Marcia K. Johnson and Carol L. Raye, "False Memories and Confabulation," *Trends in Cognitive Sciences* 2 (1998): 137–145. See also Marcia K. Johnson, "Reality Monitoring: Evidence from Confabulation in Organic Brain Disease Patients," in *Awareness of Deficit after Brain Injury: Clinical and Theoretical Issues,* ed. George P. Prigatano and Daniel L. Schacter (Oxford: Oxford University Press, 1991), 176–197.

15. See Armin Schnider, "Spontaneous Confabulation, Reality Monitoring, and the Limbic System: A Review," *Brain Research Reviews* 36 (2001): 150–160.

16. See Daniel C. Dennett, *Consciousness Explained* (Boston: Little, Brown, 1991).