

Chapter 3

- This chapter addresses memory-based confabulations. As in Chapter 1, Hirstein offers a two-stage hypothesis about the generation of such confabulations. The stages here correspond to memory retrieval and execution/monitoring.

One theme of great interest that comes up frequently in the literature is the idea that these types of confabulations might be caused by two separate malfunctions. First, patients have a memory problem, which they share with those with medial temporal lobe damage. But second, they 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, 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. (44)

- One example of an executive-focused account of memory confabulations is Marcia Johnson's theory that such confabulators have a malfunctioning reality monitoring system. On her view, memory-based confabulation arises from a failure to properly distinguish real and imagined events. (45)

- Q: Are many confabulations indiscriminate in content or type of memory involved, or are they focused on particular types of memories (e.g., autobiographical rather than semantic memory)?
- The structure of memory systems is briefly explained in §3.2. Recall the story of H.M.
- §3.3 and §3.4 give us many details about Korsakoff's syndrome and ACoA.

○ Note the 4 cognitive features of ACoA: memory loss, personality changes, executive deficits, and confabulation. (56–57)

● Some theories:

○ Johnson’s reality monitoring theory: In some confabulatory patients memories of imagined events may simply be mistaken for memories of actually experienced events. (62)

○ Schnider’s theory:

Armin Schnider’s research group similarly hypothesizes that the problem in memory confabulations is that the orbitofrontal cortex and its limbic connections are not performing their function of suppressing or inhibiting recalled memories that are not relevant to the current task. (63)

○ Recall that some confabulations are domain specific, and some theories of confabulation might better fit this datum than do others.

Whereas retrieval theories are supported by confabulation restricted to certain memory domains, executive or reality monitoring theories have trouble with domain-specific confabulation because the general understanding of executive processes is that they can be applied in more than one domain. Executive processes may have their own domain specificities, however. (64)

● So, what is the connection between memory problems and confabulation? Hirstein notes that there is definitely a correlation, but memory problems are neither necessary nor sufficient for confabulation. The correlation is very significant, though:

Nevertheless, a correlation exists between memory problems and confabulation. In a study comparing nonamnesic and amnesic patients with ACoA syndrome, only the amnesic patients confabulated (DeLuca 1993). Cunningham et al. (1997) divided 110 patients with “diverse neurologic and psychiatric diagnoses” into high-, low-, and nonconfabulation groups based on the number of confabulations they produced in a story recall test. High-confabulation patients were significantly worse than the low- and nonconfabulation groups in other memory tests. (66)

● Hirstein’s conclusion concerning the most promising underlying cause of confabulation:

If Korsakoff's syndrome involves damage to the thalamic mediodorsal nucleus, and aneurysm of the anterior communicating artery often involves damage to the orbitofrontal cortex, understanding the circuit that connects these two areas is important for understanding confabulation. This seems to be our best clue thus far toward solving the riddle of confabulation. (69)

Chapter 4

- In this chapter Hirstein groups confabulation with sociopathy and disinhibition. This suggests looking at damage to the orbitofrontal cortex as a likely explanation of confabulation.

The sociopath tells lies with an ease and confidence that resembles the act of confabulation, even if the confabulator is not actually lying. Several researchers have pointed to the orbitofrontal cortex as a problem area in sociopathy, and patients have been discovered who became sociopathic after damage to that structure, a condition known as acquired sociopathy . . . We might see confabulations as resulting from disinhibition also; the same sort of damage that loosens the behavior of the disinhibited person loosens the tongue of the confabulator. (71–72)

- We can see liars, sociopaths, and confabulators as belonging to different points along the same spectrum, along which they differ in their awareness of the falsity of their claims. (74)
- §4.2 presents many interesting examples of damage to the orbitofrontal cortex, including the story of Phineas Gage. Such injuries often negatively affect social traits and dispose these victims to risky behavior.
- Confabulation is often accompanied with disinhibition, further supporting the focus on the orbitofrontal cortex. Hirstein goes even further, claiming that confabulation is a species of disinhibition. (81)

Q: But, can disinhibition be the entire, or even primary, explanation of confabulation?

- §4.3 goes into detail concerning the anatomy of the orbitofrontal cortex. I will not discuss this.
- Sociopaths often have abnormal or damaged orbitofrontal cortices, lack social graces, and tend not to respond properly to contradictions in their

speech. These attributes are also associated with confabulation.

- Hirstein claims that sociopaths are often not outright liars, as they often seem to believe what they say (like confabulators).