

Esther Lorente-Rovira¹
German Berrios²
Peter McKenna³
Micaela Moro-Ipola⁴
Jose M^a Villagrán-Moreno⁵

Confabulations (I): Concept, classification and neuropathology

¹U.S.M. Malvarrosa
Hospital Clínico Universitario de Valencia
AVS, CIBERSAM
²Departamento de Psiquiatría de Cambridge
Reino Unido

³Complejo Asistencial Benito Menni
Sant Boi de Llobregat, Barcelona,
CIBERSAM
⁴Departamento de Psicología Básica
Clínica y Psicobiología
Universitat Jaume I, Castellón

⁵Unidad Hospitalización Psiquiátrica Breve
Hospital de Jerez de la Frontera
Cádiz (SAS)
Unidad de Gestión Clínica de Salud Mental

Introduction. A working definition of confabulation could be that of describing them as false memories due to a retrieval problem, where the patient is unaware that he/she is confabulating, and has the belief that the memory is true. Several types of confabulations have been described, according to a broad variety of criteria. Confabulations can be seen in very different neurological conditions, which have lead to a controversy on their pathophysiological mechanisms. Objective: To obtain an updated revision in Spanish of the definitions, types, brain regions involved and neuropsychological correlates of the confabulations.

Development. After reviewing the concept and several types of confabulations, the damaged brain regions associated to two conditions where confabulations occur, such as Korsakoff syndrome and patients with anterior communicating artery aneurysm, are described. The neuropsychological correlates associated to them are then reviewed.

Conclusions. Confabulations are a difficult-to-define complex phenomenon. Probably, the most accepted classification, in accordance with how they appear, would be that which distinguishes spontaneous from provoked confabulations, although the validity of this distinction is not clear. Regarding to crucial cerebral regions involved in the confabulations, it seems that prefrontal cortex lesions, specifically in ventromedial and orbitofrontal areas, are necessary. Neuropsychological evidence suggests the presence in most of the cases of executive dysfunction and at least some degree of memory dysfunction as an underlying mechanism of confabulation. Nevertheless, the specific characteristics of these neuropsychological dysfunctions are not well-known.

Key words:

Confabulations, False memory, Intrusion errors, Memory dysfunction, Executive dysfunction, Prefrontal cortex.

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Correspondence:

Esther Lorente-Rovira
Fax: 963 56 14 98
E-mail: esterlorente@hotmail.com

Confabulaciones (I): Concepto, clasificación y neuropatología

Introducción. Una definición operativa de confabulaciones sería aquella que los describe como falsos recuerdos resultado de un problema de recuperación, de los que el paciente no es consciente, y cuya creencia en la veracidad del recuerdo es genuina. Han sido descritos varios tipos de confabulaciones, utilizando una gran diversidad de criterios. Las confabulaciones pueden llegar a verse en trastornos neurológicos muy distintos, existiendo controversia en torno a sus mecanismos patofisiológicos.

Objetivo. Realizar una revisión actualizada en castellano sobre la definición, tipos, regiones cerebrales implicadas y correlatos neuropsicológicos de las confabulaciones.

Desarrollo. Tras revisar el concepto y los distintos tipos de confabulaciones, se describen las regiones cerebrales dañadas en dos patologías donde pueden aparecer confabulaciones, el síndrome de Korsakoff y pacientes con rupturas de aneurismas de la arteria comunicante anterior. Se revisan posteriormente los correlatos neuropsicológicos asociados a las mismas.

Conclusiones. Las confabulaciones son un fenómeno complejo y de difícil definición. Probablemente la clasificación más aceptada es la que, atendiendo al modo en que aparecen, distingue las confabulaciones espontáneas de las provocadas, aunque no está clara la validez de esta distinción. En cuanto a las regiones cerebrales cruciales implicadas en las confabulaciones, parece que lesiones en el córtex prefrontal, específicamente en áreas ventromediales y orbitofrontales, son necesarias para que el fenómeno aparezca. La evidencia neuropsicológica sugiere la presencia, en la mayoría de los casos, de disfunción ejecutiva y al menos cierto grado de disfunción de memoria como mecanismos subyacentes a las mismas; sin embargo, las características específicas de estas disfunciones neuropsicológicas no son bien conocidas.

Palabras Clave:

Confabulaciones, Córtex prefrontal, Falsos recuerdos, Disfunción ejecutiva, disfunción memoria, intrusiones.

INTRODUCTION

Although the term "confabulation" has traditionally been used to refer to false products of the memory (mnestic confabulations), currently, as pointed out by Schnider,¹ it is also used to refer to false perceptions of body states or the external world (non-mnestic confabulations). The present work will only focus on memory confabulations.

Different definitions have been proposed for the term confabulation. Equally, very different criteria have been used to classify them. Confabulations may appear in very different neurological disorders and in psychiatric disorders, there being controversy regarding their physiopathological mechanisms.

The purpose of the present work is to provide an updated review in Spanish on the definitions, types, brain regions involved and neuropsychological correlates of confabulations in neurological patients.

CONCEPTUAL ASPECTS

What are confabulations?

Korsakoff² described a behavioral disorder, that he called "*pseudo-remiscences*," in which the patients commit verbal errors in the belief that they are being precise or correct. This phenomenon was later called *confabulation* in the works of Bonhoeffer,³ Wernicke,⁴ Pick,⁵ and Kraepelin^{6,7}. Since then, many definitions have been proposed and the confabulations have been linked, on the one hand, to memory disorders and on the other to a type of false narration of fantastic content with little or no mnestic alteration, manifesting the difficulties existing in describing the concept.⁸

For Talland,⁹ for example, confabulation is "a factually incorrect verbal or narrative statement of the point of view from the facts, excluding intentional falsification, fantastic elaboration, random guesses, intrinsically unmeaningful stories and chaotic subjects of delusion and hallucinations, and all the systematic delusions except those that come from the disorientation of the patient in his/her experience of time." Berlyne¹⁰, on his part, considers it as "a falsification of memory occurring in clear consciousness in association with an organically derived amnesia" and suggests, following Bonhoeffer,³ two types, spontaneously or provoked and fantastic or productive. Mercer et al.¹¹ states something similar. More recently, Moscovitch¹² defined confabulation as: "a symptom that accompanies many neurological and some psychiatric disorders, such as schizophrenia. What distinguishes confabulation from lying is that there is

typically no intention to deceive and the patient is unaware of the falsehoods. It is an "honest lie"...(when) the neuronal structures involved in the reconstructive process (memory) are damaged, the memory distortions become prominent and result in confabulations."

Recently Hirstein¹³ indicated seven criteria to make up the meaning of the term confabulation: 1. Does the patient try to deceive? 2. Does the patient have some motive behind his or her response? 3. Is a defective memory involved? 4. Must the confabulation be in response to a question or request? 5. Does the confabulation fill a cognitive gap? 6. Are confabulations necessarily in linguistic form? 7. Are confabulations the result of delusions? This authors indicates that there would be three concepts of confabulation, depending on the aspects we consider essential or control: one *mnemonic* or related with memory, another *linguistic* (in which the core would be the false narrative character) and another *epistemologic* (in which the fundamental is that the subject does not question the ill-grounded and not necessarily linguistic statement, above all).¹³

In any case, serious conceptual problems exist in the definition of the use of confabulation and its application to psychopathology. As Berrios pointed out,⁸ it is likely that the tendency to use "inexact or false statements that aim to transmit information about the world or one's self" is a function normally distributed in the population in the light of certain communicative demands and therefore that the different phenomena we consider confabulation would acquire a new meaning in the light of this premise.

We will finish this section with the working definition proposed by Gilboa and Moscovitch,¹⁴ who indicated four characteristic aspects of confabulations:

- They are false memories within the context of retrieval that often contain false details within its own context as well. Therefore, some confabulations may be real memories poorly situated within time while others seem to lack any real basis.
- The patient is not aware that he/she is confabulating and often is not even aware of the existence of a memory deficit. Thus, the confabulations are not produced intentionally and probably are not the result of compensatory mechanisms.
- The patients may act in consequence with their confabulations, reflecting their genuine belief in the false memory.
- They are more obvious when an autobiographical recollection is requested. However, there is evidence of cases of spontaneous confabulations which are not directly related with the life of the patients. Under

certain conditions of evaluation, they may also appear in semantic memory tasks.

Classifications

Confabulations have been classified using different criteria: content (evaluated in terms of true/false, bizarre/fantastic, positive/negative, plausible/implausible), the way in which they arise (provoked or spontaneous), the *domains* in which they may be manifested (self-biographical, episodic, personal semantics, general semantics) and the *clinical syndromes* or *population* in which they appear (see following section).

The most popular and accepted classification nowadays is probably that of Kopelman.¹⁵ Kopelman considers that the most important criterion is how they arise. He distinguishes between *spontaneous* and *provoked* confabulations. Spontaneous confabulations would be uncommon and would be related with an amnesic syndrome superimposed to a frontal dysfunction while the provoked ones would be frequent in amnesic patients when the memory tests are administered to them. They would be similar to the errors committed by healthy subjects in prolonged retention intervals and could represent a normal response to defective memory.

The backgrounds of this classification began at the onset of the XX century. Bonhoeffer³ was the first to distinguish between "momentary" and "fantastic" confabulations. Kraepelin¹⁶ distinguished between simple and fantastic confabulations. He defined "simple" confabulation as a minor distortion in facts, time or details while "fantastic" confabulations were described as bizarre, florid, exaggerated or implausible verbalizations. Berlyne¹⁰ followed the classification of Bonhoeffer and defined momentary confabulations as fleeting, "invariably" provoked by questions probing the subject's memory, that were short and that were "real" memories displaced in their temporal context. Fantastic confabulations, on their part, would be spontaneous, sustained, of varied thematic, generally grandiose and very evident in the subjects' everyday conversation.

The validity of this distinction is questionable. There are patients who do not clearly fall into one of these two categories.¹⁷ Furthermore, it is not clear if distinguishing between spontaneous and provoked confabulations is useful to determine the etiology or temporal course.^{7, 8} At present, we do not know if provoked and spontaneous confabulations represent two different forms with different neuropathological mechanisms or if they are extremes on a continuum with a single underlying mechanism. The studies that have tried to clarify the subject have not been conclusive. Thus, some authors suggest that spontaneous confabulations

represent a more severe grade of the same disorder than the provoked confabulations¹⁸⁻²¹ while other authors believe they are two different disorders with different neuropsychological mechanisms.^{10, 15, 22}

In fact, Schneider¹ proposes four different types of confabulation, based on possible mechanisms of different production:

- *Intrusions in memory tests*^{15, 22} that could be called simple provoked confabulations, a term that would also include those occasional distortions that appear when a subject is pressured to remember the details of a story. For the author, this type of confabulations does not respond to a specific mechanism.
- *Momentary confabulations*. They describe false verbal statements (more than intrusions or distortions of isolated elements) in a discussion or another situation that urges the patient to make a comment. On the contrary to the fantastic confabulations, momentary ones are conceived and inherently plausible, although they may be inconsistent with the current state of health of the subject and the circumstances they are living. Confabulations may go from a simple statement to an elaborated, completely invented, story. It is the most common type of confabulation. For the author, this type of confabulation is still not sufficiently understood and thus it is not clear if they have their own mechanism.
- *Fantastic confabulations*, which have no base in reality, as described by Kraepelin²³ in patients with paralytic dementia and psychosis. These confabulations lack meaning, are inconceivable from logic and are not accompanied by the corresponding behavior. As occurs with momentary ones, the authors question the independence of this phenomenon.
- *Behaviorally spontaneous confabulations*, that occur within the context of severe amnesia and disorientation. The term stresses the concordances between the patient's spontaneous behavior and verbal expression of their concept of reality. Thus, it would be a syndrome characterized by confabulations, amnesia and disorientation, where, on the contrary to the rest of the other types, the patients behave according to their confabulations. This phenomenon would have a specific mechanism that is different from the other types.

The validity of these subtypes is sometimes questionable, there presently being no evidence to support it. Perhaps a very simple and pragmatic subdivision is that posed by Metcalf et al.¹⁷ who distinguish those confabulations that occur in a natural setting (spontaneously or provoked by a conversation) from those that only occur in the neuropsychological tests.

NEUROPATHOLOGY OF THE CONFABULATIONS

Neuroanatomical bases of the confabulations

There is considerable variety of disorders in which it is possible to observe confabulations¹: *delusion*, ruptured anterior communicating artery aneurysm, cerebrovascular accident, traumatic brain damage, alcoholism and avitaminosis (Korsakoff syndrome), syphilis, encephalitis due to herpes simple, tumors, hypoxia and cardiopulmonary arrest, dementia and schizophrenia, and other psychoses. In addition, it is also possible to observe confabulations in healthy subjects. However, false memories in healthy subjects and false pathological memories (confabulations) would be two different phenomena, not only because of the severity but also because of the different underlying mechanisms. False memories in healthy persons would fundamentally be the result of normal physiological processes at the time of coding (processes that associate the piece of information, constituting the memory of an event) while false pathological memories would be the result of a defective process during evocation of these memories, since most reflect erroneous composition of stored memories before the disease began.¹

As we have just seen, there is considerable heterogeneity of disorders in which confabulations can be observed. Perhaps the two most prototypic disorders would be Korsakoff syndrome and patients with ruptured anterior communicating artery aneurysm,¹⁴ which is why we briefly review the neuropathology of both disorders.

In patients with the Wernicke-Korsakoff syndrome, the mammillary bodies, anterior and dorsomedial nuclei of the thalamus and structures that include the basal prosencephaly and orbitofrontal cortex are more frequently affected.²⁴ In these patients, there are two dysfunctional subsystems. In the first, the lesions would affect the mammillary bodies and the anterior nuclei of the thalamus, which, on receiving afferences out of the hippocampus via the fornix, would be directly related with the severe amnesia that characterizes this disorder. In the second subsystem, it would have a relationship with the dorsomedial nucleus of the thalamus that is connected reciprocally with orbitofrontal and medial areas of the frontal cortex, and receives inputs from cortical and subcortical structures (amygdala and structures of the basal prosencephaly). This second system would be more related with the production of confabulations.¹⁴

In regards to the patients with ruptured anterior communicating artery aneurysm, lesions have been found in different areas in those patients who, in addition to amnesia, had confabulations: septal nucleus of the prosencephaly,²⁵ medial and orbitofrontal damage,²⁶ both frontal lesions and in the basal prosencephaly (or any other area that may cause amnesia).¹⁹

In order to clarify the localization of the lesion associated to the confabulations, Gilboa and Moscovitch¹⁴ reviewed 33 studies, that included a total of 79 cases of spontaneous confabulations, defined according to the criteria of Kopelman:¹⁵ 47 with ruptured anterior communicating artery aneurysm, 14 with traumatic brain damage and the rest with dementia, other cerebrovascular problems (infarctions, arteriovascular malformations, occlusions, posterior communicating artery aneurysms, multiple sclerosis, encephalitis, meningitis, Korsakoff syndrome, etc.). Specifically, the review was aimed at elucidating the involvement of lesions in the frontal and/or basal prosencephaly areas, which, as has been described, have been detected as being critical in the production of confabulations. The authors conclude that the lesions of the ventromedial area of the frontal lobes are sufficient to produce the confabulations, even when the damage in other regions is minimum or absent. Unfortunately, these studies based on lesions were not accompanied by neuropsychological tests on memory and executive functions.

On another part, Schacter et. al.²⁷ observed that both ventromedial frontal damage and amnesia secondary to basal prosencephaly should be present for long-lasting confabulations to occur. Thus, no specific damage alone seems to be sufficient.²⁸ As has been previously stated, although Gilboa and Moscovitch¹⁴ reviewed the existence of damage in the prosencephaly, they did not evaluate the presence of amnesia or mnesic performance of these patients.

An alternative is the proposal by Schnider.^{1, 29, 30} This author proposes the anterior limbic system as a responsible area for the appearance of spontaneous confabulations. These confabulations would be caused by lesions in anterior limbic structures, specifically by a disconnection of the loop connecting the orbitofrontal cortex (through the dorsomedial nucleus) with the amygdala. Gilboa et. al.,³¹ in a study after the previously mentioned review, agreed with Schnider. They concluded, based on the results of his study, that the prefrontal ventromedial cortex damage was always accompanied by confabulations. However, this is a necessary but not sufficient region, since the non-confabulating amnesic patients also have lesions in this area. For the confabulations to appear, there should also be a lesion in the orbitofrontal cortex. Turner et al.,³² however, indicate the inferior medial area of the frontal lobe as a critical anatomical localization.

Neuropsychological correlates of the confabulations

From the neuropsychological point of view, there are basically three hypotheses that can be distinguished to

differentiate the grade in which memory deterioration contributes to confabulation.³³ Classically, memory dysfunction was considered to be the core question. After, the role of the executive functions began to be stressed as being necessary and sufficient in the appearance of the phenomena. The third hypothesis establishes that confabulations are caused by a combination of memory deterioration and executive dysfunction. The first studies did not use standardized executive tests but rather indexes such as monitoring of the responses, perseverations, etc. More recent studies are already using well- standardized tests on both memory and executive functioning. These three hypotheses are explained in the following.

Memory dysfunction

This hypothesis establishes that amnesia is a necessary condition for confabulating. Korsakoff² described confabulations as "a special form of amnesia" and the notion that they are primarily a memory disorder has predominated since then.³³ It is true that confabulations are a prominent clinical characteristic in different neurological disorders that involve memory deterioration, such as the Wernicke-Korsakoff syndrome.⁹ From this model, it is understood that confabulation is a way of filling the gaps left by amnesia.^{3, 10, 34}

Levine et al.³⁵ studied a patient with severe traumatic brain damage associated to a lesion in the right frontal areas who had isolated retrograde amnesia and who suffered confabulations in the initial phases of his recovery. From the neuropsychological point of view, the patient did not have any other deficit in other memory or executive functions. However, evidence has demonstrated that a memory deficit alone cannot explain the phenomenon of confabulations since, for example, in patients with Korsakoff syndrome, the confabulations tend to remit, even when the severe amnesia persists.²⁴ Korsakoff and Kraepelin already acknowledged that defective memory alone cannot explain the appearance of confabulations.³⁶ On the other hand, pictures such as the Capgras syndrome do not include prominent disorders of the memory, even though they manifest confabulations. Finally, in healthy subjects, confabulations are not a usual response to memory deficits.^{37, 38}

Schnider¹ maintains that provoked confabulations are not associated with amnesia because, in fact, they also appear in healthy subjects.³⁹ However, relevant momentary confabulations, fantastic ones produced by patients with schizophrenia, and behaviorally spontaneous ones would always be associated to a variable grade of amnesia, measured through delayed free memory tests. In these confabulating patients, learning and recognition may be preserved. Therefore, the confabulations would not have to

simply be deficiencies in information storage with a gap in memory.

Executive dysfunction

This hypothesis establishes that confabulations are the result of an executive dysfunction. In general, the data provided to support this idea are based on the fact that the confabulations decrease when the executive function improves.^{11, 18, 40-42}

The Mercer et al. study¹¹ provides evidence of covariation between confabulations and executive performance without changes in the memory functioning. These authors divided a group of 10 neurological patients into non-confabulators, moderate confabulators and severe confabulators. The severe confabulators differ from the non-confabulators and moderate ones because they have worse capacity to monitor and correct their responses. In this study, no relationship between the severity of memory deterioration and confabulations was found.

Mention is usually made of the work of Kapur and Coughlan¹⁸ as evidence in favor of the role of executive dysfunction in confabulations. They provided a single case study in which a patient showed very marked confabulations after suffering frontal lobe damage caused by an anterior communicating artery aneurysm. Initially, the patient had both "fantastic" and "momentary" confabulations. However, several months later, the patient only had "momentary" confabulations. This change in the type of confabulation was parallel to improvement in performance on some executive tests (Cognitive Estimations and the Wisconsin Card Sorting Test, WCST, modified, although without changes in others such as verbal fluency). His global functional profile on the memory tests did not change (he performed well on a series of memory tests, such as recognition tests and immediate recall of stories, but very badly on the delayed memory test). The authors concluded that confabulations did not require a global amnesic syndrome to become manifest and that the severity of the frontal lobe dysfunction (executive) determined whether the type of confabulation was more or less fantastic.

Similarly, Papagno and Baddeley⁴⁰ studied a male patient who had clear evidence of confabulating. This fact was associated to frontal lobe damage and to deterioration in performance on a verbal fluency test and on the WCST. On the Wechsler Memory Scale, his performance on the free verbal recall tests and on the paired associated learning was normal. Only delayed recall of short stories and learning outside the spectrum (*supra-span*) showed evidence of abnormal performance, this improving when the confabulations ceased.

But, above all, the patient was not clinically amnesic. The authors concluded that the patient seemed to have a relatively normal memory capacity, but that his capacity to evaluate that which was recovered and to stop the retrieval when the result was not plausible was temporally affected.

Shapiro et al.⁴¹ studied seven neurological patients with different conditions who had confabulations. They administered four tests: the confabulation battery used by Mercer et al.¹¹ in order to elucidate the nature of the confabulations in a structured situation; the confabulation test with cues to be able to test the capacity of the confabulators to use cues; the modified Visual-Verbal test, chosen to evaluate capacity to monitor responses, to inhibit the incorrect responses or the response perseverations, to change the tendencies of response and to use cues to direct the performance and finally, the Cognitive Estimation task. They suggested two groups of confabulations, that is, moderate and severe. The severity of the confabulations was associated with perseverations, deteriorated resources of self-monitoring and failure to inhibit incorrect responses. In fact, in one of the cases of the study, the resolution of the confabulations involved attenuation of these cognitive deficits. This study does not provide information on memory functioning.

Benson et al.⁴² described a patient with acute alcohol-induced Korsakoff amnesia, whose confabulations decreased with improvement of the executive function - according to the neuropsychological evaluation - and the frontal lobe function - according to the functional image - within the context of absence of changes in memory function. Specifically, the Trail Making (A and B), Stroop Test, verbal fluency and Design fluency tests improved when the confabulations disappeared. This correlated with a significant improvement of perfusion in the cingulate and orbitofrontal cortices. The patient's performance on the WCST was normal and consistent with this. His dorsolateral frontal cortex showed normal perfusion in the SPECT. The authors concluded that the confabulations were the result of a dysfunction of the orbital and medial frontal cortex.

Other studies, on the contrary, have not found any evidence of executive involvement in confabulating patients.^{43,44}

Consequently, although most of the literature reviewed has shown some association between confabulations and executive functioning, this is not always true. In the cases where a positive association is found, the question could be posed if they are really providing evidence on the exclusive involvement of executive dysfunction in the appearance of the confabulations. As we have seen, these studies either do not take into account that some memory deficits are also present^{11, 18, 42}, which therefore indicate the involvement to

some degree, at least, of certain memory functions, or they do not evaluate/inform on the memory functioning.⁴¹ All this would lead us to consider the third hypothesis in question.

Dual hypothesis: memory and executive dysfunction

The third hypothesis supports the simultaneous involvement of deficit processes, both memory and executive functioning, in the appearance of the confabulations.

Mention has previously been made of the Mercer et al.¹¹ study to support the idea of executive dysfunction in the confabulations as no evidence has been found on covariation between confabulations and memory deficits. In relationship to memory functioning, we found the mnemonic capacity of the patients without confabulations was not distinguishable from that of moderate confabulations. Severe confabulators had greater deterioration in recent memory and comparatively better performance in remote memory questions. It is true that no relationship was found between the number of correct responses and grade of confabulation, but all of the patients had amnesia. The authors concluded, consequently, that confabulations may be attributed to the coincidence of four factors: 1) The patients' belief that a better response is expected on their part, 2) lack of precise recall of the response, 3) availability of an overlearned or affectively significant response and 4) defective capacity to monitor it or to make self-corrections.

Stuss et al.⁴⁵ suggested a frontal deficit superimposed on a basic deficit in the memory as a possible mechanism of spontaneous and persistent confabulations. The five patients they studied showed, together with amnesia, frontal dysfunction reflected in failure to inhibit responses, inability to monitor behavior, noticeable misuse of environmental cues, tendency to be impulsive and lack of concern about incorrect performance.

Fisher et al.²¹ studied the neuropsychological and neuroanatomical correlates of nine patients in an acute period of recovery after rupture and clipping of aneurysms of the anterior communicating artery. Five of the nine cases had "spontaneous" confabulations and had severe anterograde amnesia, very poor attention and executive functioning and disease negation. The remaining four patients only had "momentary" or "provoked" confabulations and also had severe anterograde amnesia but showed deterioration of the relatively moderate executive functioning.

Baddeley and Wilson⁴⁶ gave a detailed description of an amnesic patient with bilateral damage in the frontal lobe

who confabulated. The patient had severely deteriorated episodic memory both for visual and verbal material, poor autobiographic memory and deterioration of the semantic memory. He also had dysexecutive syndrome, neuropsychologically seen by poor performance in verbal fluency and on the WCST.

DeLuca²⁸ provided a series of three amnesic patients with anterior communicating artery aneurysm with deteriorated performance in measurements of executive functioning (WCST) who had fantastic confabulations. The authors also provided data on three more patients but without amnesia, who also showed deterioration on the WCST. However, these patients did not have confabulations. The author concluded that confabulations require the presence of an amnesic disorder and executive dysfunction.

Cunningham et al.³³ classified 110 patients with different neurological diseases and psychiatric disorders into non-confabulators, mild confabulators, and severe confabulators according to their responses on a standardized memory test. They considered confabulation as a novel intrusion on a prose test. Severe confabulators performed significantly worse on the memory measurements and executive functioning that evaluated sustained attention, mental tracking and set-shifting ability, part A and B of the Trail Making Test. However, there were no differences between the groups in measurements of problem-solving, formation of concepts in verbal fluency (WCST, phonological fluency test, Stroop test). These findings suggest that both memory as well as other aspects, but not all, of executive functioning contribute to confabulations.

Hashimoto et al.⁴⁷ described the case of a 73-year-old woman who developed an amnesic confabulatory syndrome after right focal basal hemorrhage (right focal basal forebrain hemorrhage). The confabulation, in spite of the persistent anterograde amnesia both with verbal and visual material, gradually abated with the improvement of the executive function (modified WCST and part B of Trail Making). This study provides similar results to those provided by Kapur and Couglan¹⁸ and Benson et al.,⁴² but with different conclusions.

Therefore, in relationship to the neuropsychological mechanisms involved in confabulations, we can conclude that both memory as well as executive systems are involved to some degree in the appearance of confabulations, although there is no profile consistent with dysfunction in any of these broad neuropsychological functions. However, as indicated by Schnider,¹ it is true that most of the patients who have a combination of severe memory dysfunction and executive failures (common in patients with traumatic brain damage) do not confabulate markedly. It is also not clear which specific executive dysfunctions are responsible for the confabulations,

whether they are specific, a combination of some, or of a generalized executive failure. It is also not clear if executive dysfunctional differences could give rise to different types of confabulations. The most recent evidence points to a specific executive deficit more than a generalized one associated to confabulations: Fischer et al.²¹ only found an association with tests that measure self-monitoring (*set shifting* and *perseveration*). Cunningham et al.³³ found an association with tests that measure sustained attention, *set shifting* and *mental tracking*, but not with the forming of concepts, problem-solving or verbal fluency. Finally, Nys et al.⁴⁸ reported the disappearance of spontaneous confabulation parallel to improvements in mental flexibility but not in other executive measures.

With the explicit purpose of making an attempt to elucidate the memory and executive deficit involved, and in the case of the latter, which are specifically involved in the production of confabulations, Turner et al.³² studied 57 patients with focal frontal lesions, administering a confabulation battery. This made it possible for them to detect eight patients with high scores on the battery. They administered them a wide battery of neuropsychological test. They confirmed that, in fact, there was a large variety in regards to performance on the memory tests and executive functioning in these patients. They found that while there were significant differences in all of the memory measurements evaluated between the high confabulator's group with regards to the low confabulator's groups in confabulations, these groups differed in only two executive measurements, that is, the Stroop interference test and the verbal fluency test, the deficit being more consistent in the former of the measurements. Consequently, they concluded that some grade of memory dysfunction seems to be necessary, but that performance in the classical measurements of executive functions is less useful when explaining the confabulations. In another work, regarding explanatory models of the confabulations,⁴⁹ this question was approached on reviewing the evidence supporting it.

CONCLUSIONS

Confabulations are false memories within the context of retrieval, in which the patients are not generally aware that they are confabulating, believing that the false memory is genuine (since they can act in consequence of their confabulations). That is, this is an "honest lie." Different types of confabulations have been described using a large diversity of criteria. Probably, the most accepted classification is that which distinguishes spontaneous from provoked confabulations. The validity of this distinction is not clear. In fact, it is not known with certainty if it refers to a continuum of severity or distinct disorders with also different physiopathological mechanisms.

In regards to the crucial cerebral regions involved in the confabulations, it seems that in order for this phenomenon to appear, there must be lesions in the prefrontal cortex, specifically in the ventromedial and orbitofrontal areas. Neuropsychological evidence suggests, in most of the studies, at least a certain grade of memory dysfunction and executive dysfunction as underlying mechanisms to them. However, the specific characteristics of these neuropsychological dysfunctions are not well known.

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