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Editorial

Confabulations and related disorders: We've come a long way, but there is still a lot to do!

Gianfranco Dalla Barba [a,b,c,d,*] and Michael Kopelman [e]

[a] INSERM, Paris, France
[b] Institut de la Mémoire et de la Maladie d'Alzheimer (IM2A), Département de Neurologie, Hôpital de la Pitié-Salpêtrière, Sorbonne Universités, Université Pierre et Marie Curie-Paris6, Paris, France
[c] Dipartimento di Scienze della Vita, Università degli Studi di Trieste, Italy
[d] Centro Medico di Foniatria, Padua, Italy
[e] King's College London, Institute of Psychiatry, Psychology and Neuroscience (IoPPN), Academic Unit of Neuropsychiatry, St Thomas's Hospital, London, UK

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1. Introduction

If you search PubMed you find that 30 years ago (1987), only one neuropsychological paper on confabulation was published, the article by Kopelman distinguishing between provoked and spontaneous confabulation (Kopelman, 1987). During the course of 2016, there have been 31 papers on confabulation to date, and in the intervening years 306 publications.

For many years, the study of confabulation occurred within the domain of psychiatry. In his pioneering studies Korsakoff (1889), described his syndrome as a 'psychosis', although he also proposed a temporal context confusion account of confabulation (a confusion of "old recollections with present impressions"). Although neurology was already an active medical discipline, especially within Charcot's group at the Salpêtrière in Paris, the study of confabulation remained firmly anchored within the psychiatric writings of Kraepelin, Bonhoeffer, Pick, Van der Horst, Bleuler, Chaslin, Berlyne, and others. Although we may never know who introduced the term 'confabulation' into medical discourse, potential candidates include Wernicke (Wernicke, 1900) and Bonhoeffer (Bonhoeffer, 1904). In his textbook *Elements of Mental Semiology and Clinics* (1912), the French psychiatrist Chaslin at the Salpêtrière entitled one paragraph on confabulation as 'Perversions of memory. False memories, pseudo-reminiscences (the Germans' Confabulations)'. This brief title both attributes to the Germans the origin of the term and endorses the psychiatric nature of this phenomenon as 'perversions' of memory.

The current neuropsychological interest in confabulation can partly be traced back to Kopelman's paper (Kopelman, 1987) in which, following Bonhoeffer (1904) and Berlyne (1972), the distinction between provoked and spontaneous confabulations was proposed. Since then, a number of theories on the origin of confabulation have been proposed, but the distinction between provoked and spontaneous confabulations, although criticized, remains a notion, which cannot be overlooked.

As far as the mechanisms of confabulation are concerned, four major approaches have been proposed.

Johnson argued that confabulation reflects poor source monitoring, or reality monitoring, i.e., deciding whether a memory is a trace of something that actually happened or is a memory of an imagined event (Johnson, 1991). Damage to frontal/executive functions was postulated to produce an

* *Corresponding author*. INSERM, Paris, France.
E-mail address: michael.kopelman@kcl.ac.uk (M. Kopelman).
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impairment in reality monitoring and, thereby, to stimulate confabulation. This interpretation of confabulation is consistent with the idea that confabulation is a form of source amnesia (Moscovitch, 1989; Schacter, Harbluk, & McLachlan, 1984).

Moscovitch and colleagues (Gilboa, Alain, Stuss, Melo, & Moscovitch, 2006; Moscovitch, 1989, 1995; Moscovitch & Melo, 1997) have proposed that confabulation is the result of a deficit in strategic retrieval. When strategic retrieval is disrupted, following damage to the ventromedial and orbitofrontal cortex, confabulation in both semantic and episodic memory should occur, assuming that the demands of episodic and semantic information on strategic retrieval are matched.

In another approach to the underlying mechanisms, Dalla Barba (2002) argued that a disruption of personal temporality produces confabulation. This hypothesis assumes that, in confabulating patients, knowledge of time is preserved in that they are aware of a past, present and future. However, they are unable to distinguish between personal habits, repeated events and routines, on the one hand, and true episodic memories or true personal plans, on the other. In spite of some important differences, this theory is not incompatible with the theory proposed by Schnider and co-workers which posits that confabulators suffer from a deficit of reality filtering and fail to suppress inappropriate memory traces (Bouzerda-Wahlen, Nahum, Liverani, Guggisberg, & Schnider, 2015).

A fourth approach involves a motivational account, arguing that confabulation results from patients' tendency to embellish their memories in order to protect the 'self', driven by wishful thinking (Conway & Tacchi, 1996; Fotopoulou, Conway, & Solms, 2007).

The idea for this Special Issue was proposed by one of the guest editors (GDB) and was enthusiastically accepted by Sergio Della Sala. Our aim was not to provide answers to all the open questions concerning confabulation, but instead to reflect the theoretical and experimental work of some of the major contributors to this topic. Only one paper on confabulation was published in 1987, 31 in 2016 and 16 in this issue. Confabulation is no longer a psychiatric phenomenon, or a clinical anecdote. It is addressed in neuropsychological and experimental terms; and theorists attempt to understand why individuals, brain damaged or not, make errors in remembering.

2. In this issue

This issue consists of 16 papers covering a wide range of topics and employing different techniques. Here we provide a very brief summary of the papers and then raise some questions for future research.

The involvement of the orbitofrontal cortex in confabulation has repeatedly been proposed. Schnider and co-workers in their article provide a review of their theory, which proposes that what they call 'behaviorally spontaneous confabulation', in which patients tend to act according to their confabulatory beliefs, results from a deficit in 'reality filtering'. This, in turn, is a consequence of a lesion to the orbitofrontal cortex.

Gilboa and Moscovitch suggest that the ventro-medial frontal cortex (vmPFC) establishes context relevant templates, and mediates decision-monitoring processes to ensure that only context-relevant responses are enacted. Using EEG techniques, they provide evidence for this hypothesis in patients with vmPFC damage and confabulation.

Bajo and co-workers investigated the cognitive and emotional factors associated with the presence and clinical course of confabulation with follow-up over 9 months. Their findings show that the severity of memory impairment (especially on autobiographical memory) and errors in executive tests (particularly in making cognitive estimates) are strongly associated with the severity of confabulation scores at baseline and also changes through time. Their findings suggest that confabulation results from executive dysfunction where autobiographical memory is also impaired (compare Johnson, O'Connor, & Cantor, 1997), and it resolves when these impairments subside.

Whilst Bajo et al. found that confabulations in general tend to decline, Dalla Barba et al. found that many confabulations were both consistent and persistent through time, the patients giving the same confabulations to the same questions over time. This was particularly true where habits and repeated personal events were mistaken as specific, unique personal episodes, or as well-known public events where semantic knowledge was implicated. Such errors occur within Temporal Consciousness, in which individuals normally remember their personal past, are oriented in the present world, and predict their personal future.

Turnbull describes the 'emotion dysregulation' hypothesis in the origin of confabulation (compare Conway & Tacchi, 1996; Fotopoulou et al., 2007). He develops the idea that the positive aspects of confabulatory states may have a role in perpetuating the imbalance between cognitive control and emotions. He identifies three main causal factors: that positive emotions are related to more global or schematic forms of cognitive processing; that positive emotions influence the accuracy of memory recollection; and that positive emotions make people more susceptible to false memories.

Coltheart has distinguished between spontaneous confabulation, on the one hand, and two types of provoked confabulation, on the other. The latter he labels as 'memory-recall provoked confabulation' and 'question-provoked confabulation', and he explores the latter in detail. He argues for a broader conception of confabulation, seen also in healthy people as part of "a drive for understanding".

Venneri et al. have compared resting state fMRI in 18 confabulating Alzheimer patients (AD) and 18 non-confabulating AD patients. They found that confabulatory tendencies in early AD are associated with a disconnection between computational hubs in frontal and medio-temporal regions, coupled with up-regulation of frontal activation, especially in the midline and anterior cingulate regions.

Spitzer and co-workers investigated confabulation in autistic adolescents. This is a largely unexplored domain. The authors administered executive tests, and a questionnaire aimed at eliciting confabulation, to their patients and to a group of normal controls. On the basis of their results, the authors suggest the possibility that, in at least some cases,

confabulation in autism may be less related to social factors than to impaired source memory or poor executive function.

McDermott and co-workers investigated a phenomenon related to confabulation, namely false recognition, using the well-known Deese–Roediger–McDermott paradigm, specifically conceived to elicit false recognition. The question addressed was whether novel words, which are subjectively experienced as having been recently studied, would elicit the parietal memory network activation in the same way as 'hits'. Their interesting result is that true old items and false alarms activated similarly the parietal memory network.

Garrison et al. carried out an experiment in healthy participants, finding that source memory was less accurate for auditory stimuli than visual stimuli with a greater rate of externalization than internalization. They argued that the findings were consistent with the greater prevalence of clinical auditory, rather than visual, reality discrimination errors, which they argued were relevant to confabulations, hallucinations, and delusions.

Feinberg and Roane studied 4 patients with delusional misidentification for persons and/or confabulation about 'phantom' persons and prominent 'self-referential' narratives. They compared these cases with 4 cases of delusional misidentification for persons and/or confabulation about 'phantom' persons who lacked self-referential symptoms. They argued for a role of psychological defence in the self-referential cases.

Bertrand et al. have provided a valuable translation of Arnaud's (1900) description of a case of persistent déjà vu, and a very helpful discussion and interpretation of this case in the light of modern neuropsychological findings. They argue that the psychopathology is best understood as a form of memory disorder, a reduplicative paramnesia, best described as 'recollective confabulation'.

Turner et al. have also described deja vecu or recollective confabulation as instances in which the sense of déjà vu is persistent and convincing. Their patient's *deja vecu* experiences were entirely restricted to non-personal events, suggesting that there are differences in the degree to which personal and emotional associations are formed for autobiographical and non-autobiographical episodic memories. They propose a two-factor theory of deja vecu.

In a very different context, Gudjonsson describes a 'memory distrust syndrome', which can lead to false confession which, in turn, can be internalized and described as a 'confabulation'. He has described a case in which solitary confinement and other contextual risk factors, personal vulnerability, and the acute mental state led to the gradual development of a memory distrust syndrome. He warns that this can occur in intellectually able and educated individuals.

We asked Paolo Bartolomeo to write an article on confabulation following right brain damage. He and his colleagues argue that confabulation can occur in patients with right hemisphere damage. They are not 'memory confabulations', but these patients may be unaware of their left hemiplegia, and confabulate about it, denying the ownership of their left limb. The authors review this literature and propose that confabulation in right brain damage might reflect, at least partially, the attempts of the left hemisphere to make sense of inappropriate input received from the damaged right hemisphere.

Finally, Martinaud et al. have also examined bodily 'ownership' in right hemisphere lesions. They have contrasted the visual capture effect in 'ownership' of a rubber hand with disturbance in the sense of somatic ownership. The former they relate to pathology in a fronto-parietal network, and the latter to more posterior lesions including the right temporo-parietal junction and supramarginal gyrus.

3. Issues and controversies

Common themes in this Special Issue have included the assumption of different subtypes of confabulation, the importance of reality monitoring, the critical nature of damage to the orbito-frontal or ventro-medial prefrontal cortex in at least some cases, and the scope for either broadening the concept of confabulation or, at least, investigating related phenomena. However, despite the increasing interest and literature on confabulation, and the effort made in this Special Issue, there are still a number of issues that remain unresolved.

One is how broadly or narrowly we should use the word 'confabulation' itself. Kopelman (2010) argued for a narrow definition, related to false or erroneous memories arising unintentionally in the context of a neurological amnesia. He believed that confabulation should be kept distinct from delusions, delusional memories, or other phenomena such as anosognosia, because incorporating them risks a loss of meaning in conflating what might be rather different phenomena with distinct underlying bases. Others in this volume (e.g., Coltheart) have taken a different view, arguing that confabulation is a general property of human cognition and the 'drive for causal understanding'; and Gudjonsson has related it to the phenomenon of ('internalised') false confession.

A second issue, within the topic of 'memory confabulation' itself, is the question of how many subtypes of confabulation are useful, and are they truly distinct, or should they be viewed as lying along a single dimension? Berlyne (1972) and Kopelman (1987) postulated two types; Coltheart postulates three subtypes (spontaneous and two types of provoked); and Schnider (2008) has argued for four subtypes (intrusions/simple provoked, momentary, fantastic, and behaviorally spontaneous). Whilst others have raised legitimate questions about how distinct these subtypes of confabulation really are, further categories have been offered in this Special Issue (e.g., the notion of 'recollective confabulation'). Should we be thinking in terms of a dimension or continuum of provoked versus spontaneous confabulation, manifest in either thought/recollection and/or action? But what does this do to Kopelman's (and now Coltheart's) underlying assumption of provoked confabulation as a component of normal cognitive processes versus spontaneous confabulation as a phenomenon that is specifically pathological, related to underlying brain disease?

Another issue concerns the quantification of confabulation. At present, it is seldom specified how much the patients described in the literature are confabulating. The result is that patients who may produce one or two confabulations are compared with patients who confabulate across the board on objective measures of confabulation, such as the confabulation battery (Dalla Barba, 1993; Dalla Barba & Decaix, 2009). It is

as if conclusions on the nature of anomia were drawn by comparing patients with one or two anomias in spontaneous speech with patients who have an amnestic aphasia. Can sensible conclusions on the mechanisms of confabulation really be drawn by describing, studying and comparing patients in whom confabulations are not quantified?

A further question concerns what role and weight should be attributed to frontal dysfunction in confabulation. If it is uncontroversial that some patients with frontal lesions (or executive dysfunction) confabulate, it is also the case that confabulations have been observed following lesions in at least other 19 posterior and subcortical sites. Moreover, for confabulation to occur, the hippocampus must be, at least partially, preserved (Dalla Barba & La Corte, 2013). So, are frontal lesions really necessary and sufficient for confabulation to occur, or should other brain lesions, as well as the sparing of the hippocampi, be taken into account in a general theory of confabulation?

These issues and others will continue to be debated, as well as those thrown up by related phenomena, including delusions, *deja vecu*, and anososgnosia. The increasing literature on these issues will, we hope, lead to a better taxonomy and understanding of these phenomena.

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