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AUTOBIOGRAPHICAL MEMORY IN AMNESIA

Michael D. Kopelman, Laura Marsh

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Autobiographical memory in amnesia

Mémoire autobiographique dans l'amnésie

Michael D. Kopelman, Laura Marsh

King's College London
Academic Neuropsychiatry,
3rd Floor, Block D, South Wing,
St Thomas's Hospital,
Westminster Bridge Rd,
London SE1 7EH, UK
<michael.kopelman@kcl.ac.uk>

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Abstract

The term 'autobiographical memory' refers to the recollection of personal facts, episodes and incidents. It can be affected in various ways in clinical disorders, both neurological and psychological. In this paper, examples of 'anomalies' in autobiographical memory are discussed in the context of current explanatory theories. Retrograde amnesia (RA) is a fascinating phenomenon, referring to loss of memories for autobiographical episodes and/or personal semantic facts which occurred before the onset of a brain disease or injury. The severity of RA is only loosely associated with the severity of anterograde amnesia, suggesting different underlying mechanisms. There are various theories of how and why RA occurs. These will be reviewed, and it will be suggested that they all have their limitations. A retrieval deficit, with an age-related encoding factor, giving rise to a temporal gradient, will be suggested. Spontaneous confabulation refers to the unprovoked flow of erroneous memories, seen in some neurological patients, now thought to relate to damage in the ventro-medial and orbito-frontal regions of the frontal lobes. A recent study will be presented which tested between alternative theories of confabulation, finding damage to autobiographical memory and executive systems to be most critical to the 'rise and fall' of confabulation. Psychogenic amnesia intrigues the media! It can be 'global' or 'situation-specific'. A recently published study of 53 cases has highlighted 4 different types of syndrome (or subgroups). The study emphasised the psychosocial circumstances in which the amnesia occurs, and demonstrated different patterns of autobiographical memory loss across the subgroups, and with differential patterns of outcome.

Key words: autobiographical memory • retrograde amnesia • temporal gradient • confabulations • psychogenic amnesia

Résumé

La mémoire autobiographique, impliquée dans la recollection de faits personnels, d'événements et d'incidents, est altérée dans de nombreuses pathologies neurologiques et psychologiques. Cet article propose, en lien avec les conceptions théoriques actuelles, d'illustrer des exemples d'« anomalies » de la mémoire autobiographique : l'amnésie rétrograde, les confabulations et l'amnésie psychogène. L'amnésie rétrograde consiste en une altération de la mémoire autobiographique affectant la mémoire épisodique autobiographique et/ou la sémantique personnelle pour les faits ou événements antérieurs au début de la maladie. Les différentes théories explicatives de cette amnésie rétrograde, ainsi que leurs limites, sont présentées dans cet article. Un déficit de récupération associé à un effet de l'âge sur les processus d'encodage est suggéré pour expliquer les observations de gradient temporel. Les confabulations spontanées font référence à la production non provoquée (spontanée) de souvenirs erronés. Une étude récente, visant à tester les différentes théories explicatives des confabulations, est présentée. Les résultats suggèrent un lien entre mémoire autobiographique, système exécutif et confabulations. Enfin, l'article présente différentes formes d'amnésie psychogène en soulignant le rôle des facteurs psychosociaux dans son développement. Le profil des troubles de mémoire autobiographique et leur évolution sont également décrits selon le type d'amnésie psychogène.

Mots clés : mémoire autobiographique • amnésie rétrograde • gradient temporel • confabulations • amnésie psychogène

Correspondance :
M.D. Kopelman

Introduction

In this paper, we will discuss three topics:

- (i) retrograde amnesia;
- (ii) a recent study of confabulation;
- (iii) and a study of psychogenic amnesia.

Taken together, these could be described as 'anomalies' of autobiographical memory.

Retrograde amnesia

Ribot [1] proposed that 'The progressive destruction of memory follows a logical order—a law... it begins with the most recent recollections which, being ... rarely repeated and ... having no permanent associations, represent organisation in its feeblest form.' In other words, there is a relative sparing of early memories, a so-called 'temporal gradient'.

Kopelman [2] demonstrated these temporal ('Ribot') gradients across differing remote memory tasks. As *figure 1* shows, both Alzheimers and, to a greater degree, Korsakoff patients exhibited a significant temporal gradient in the recall of autobiographical incidents, personal semantic facts, and famous news events. Relative to controls, both patient groups performed significantly less well on recent memories than on more remote memories across all three tasks, with Korsakoff patients showing the steeper temporal gradient. Rensen *et al.* [3] have recently shown that, in Korsakoff patients, the same pattern is produced whether the Autobiographical Memory Interview (AMI) [4] or the Autobiographical Interview (AI) [5] is administered, with significant group by time-period interactions on both tasks. Barnabe *et al.* [6] found the same pattern in Alzheimer patients; the two methods of autobiographical memory assessment gave essentially the same results.

There are several current theories purporting to explain retrograde amnesia and the occurrence of this temporal gradient. The first is classical consolidation theory [7, 8] which states that there is consolidation and structural re-allocation such that early memories eventually become independent of the hippocampi and the medial temporal lobes, and thereby spared from the effects of medial temporal damage. Secondly, there is episodic-to-semantic shift (semanticisation) theory [9], which proposes that episodic memories gradually acquire a more 'semantic' form as they get older, thereby protecting them against the effects of brain damage. This process may be associated with structural re-allocation. Thirdly, multiple trace theory [10] suggests that the hippocampi and medial temporal lobes are always involved in the storage, retrieval, and reactivation of memories. Proponents of this theory consider that the relative sparing of early memories is related to the number of traces that have been laid down. This leads to two predictions: first, that the extent of hippocampal damage should be predictive of the severity of retrograde amnesia; and second, that extensive hippocampal damage should give a flat gradient in episodic memory, and a steeper gradient in semantic memory. Fourthly, memory transformation theory, proposed by Winocur and Moscovitch in 2011 [11], suggests that episodic memories become more semantic, or 'gist-like', with age. This is really very similar to Cermak's [9] semanticisation theory.

However, in our view, none of these theories works terribly well. First, in consolidation theory, it is unclear how long the consolidation is proposed to last, whether years or decades. With regard to theories of memory semanticisation or memory transformation, there is no unequivocal evidence of an episodic-to-semantic shift. Further, it is not entirely clear what the predictions of memory

Figure 1. Temporal gradients across different remote memory tasks: From Kopelman, *Neuropsychologia*, 1989.

transformation theory should be. The conventional wisdom suggests that, in patients with retrograde amnesia, episodic memories will have a flat gradient across all earlier time-periods, while semantic memories will have a steeper temporal gradient. However, if episodic memories are being semanticised, you might expect a crossover effect, with episodic memories showing the opposite gradient to semantic. Finally, in multiple trace theory, experimental findings do not always match the predictions proposed in this theory. First, the correlation between the extent of the retrograde loss and the degree of hippocampal atrophy can be weak, and frontal and lateral temporal changes can be critical [12]. In medial temporal patients, Bright *et al.* [13] found that medial temporal patients showed only a mild retrograde loss in the recall of autobiographical episodes with a temporal gradient. It was only as the atrophy extended into lateral temporal regions that a severe and flattened retrograde memory loss was observed. Lah and Miller [14] reported the same pattern in their review of retrograde amnesia following temporal lobe lesions. Secondly, the theory predicts a steeper gradient in semantic memory than episodic, but this does not always occur. Greene, Baddeley and Hodges [15] found no significant group by time-period interaction for personal semantic memory in Alzheimer patients, but a significant interaction for autobiographical episodes, implying a steeper pattern for the latter. Kopelman *et al.* [16] found the same pattern in Korsakoff and encephalitis patients, i.e. the opposite pattern from the multiple trace theory prediction.

Svoboda *et al.*'s [17] meta-analysis of 24 functional imaging studies of activations in autobiographical memory is often interpreted as arguing for a hippocampal network in autobiographical memory retrieval. However, the actual data points showed activation of a frontal polar region, a more posterior frontal region, a retrosplenial region, a parietal region and a lateral temporal region. In other words, multiple, widespread cortical regions were activated during autobiographical memory retrieval.

It is possible that there is a retrieval deficit in retrograde amnesia. The correlations between the severity of retrograde amnesia and the severity of anterograde amnesia are often low [18, 19]. Much higher correlations have been found between the severity of retrograde amnesia and executive performance or frontal lobe volumes [12, 18, 20]. Moreover, frontal activations are consistently found on autobiographical memory retrieval in functional imaging studies [17, 21]. Finally, there is some evidence of a recall-recognition discrepancy, or a disproportionate context/ no context difference on remote memory retrieval [2, 22]. Sanders & Warrington [23] argued that a retrieval deficit should give a flat retrograde amnesia curve. However, a temporal gradient does not exclude the possibility of a retrieval deficit.

In a previous study, Kopelman, Wilson and Baddeley [4] found an age effect in autobiographical memory loss. Younger patients showed a flat gradient, while patients over 40 showed a steeper gradient. They argued that:

"(This) suggests that...older subjects (but not the younger) encode or consolidate new memories in a way that somehow makes them more vulnerable to the effects of brain damage than are earlier memories."

Elsewhere, Kopelman [24] wrote:

"The specific manner in which memories are encoded in earlier life protects those early memories—particularly autobiographical memories—from the effects of subsequent brain damage, giving rise to a temporal gradient in retrograde memory loss."

One of the factors involved in that early encoding may be the autobiographical memory 'bump' [25]. Some studies of amnesic groups (although admittedly not all) show relative preservation of autobiographical memories for the period of early adulthood, 18-30 years: an autobiographical memory 'bump' can be observed in findings from patients with Alzheimers disease, frontotemporal dementia (frontal variant), and semantic dementia [2, 26-28].

In summary, we have argued that the 'main' current theories do not explain completely satisfactorily an extensive retrograde amnesia in the presence of a temporal gradient in episodic memory. Secondly, we have argued that frontal and lateral temporal damage can contribute to retrograde amnesia, as well as hippocampal and medial temporal pathology. We have postulated a retrieval deficit with a temporal gradient that results from age effects at encoding, which overlap with those which produce the autobiographical memory 'bump' in healthy people.

■ Confabulation

Confabulation can be defined as false or erroneous memories arising unintentionally in the context of a neurological amnesia. The memories may be false in themselves or 'real' memories jumbled and confused in temporal context and retrieved inappropriately. Elaborating on Berlyne [29], Kopelman [30, 31] distinguished between spontaneous and provoked confabulation. Spontaneous confabulation can be defined as '*a persistent, unprovoked outpouring of erroneous memories*', whilst momentary or provoked confabulation consists of '*fleeting intrusion errors or distortions in response to a challenge to memory, such as a memory test*' [31]. Within the category of provoked confabulation, Coltheart [32] has made a further distinction between memory-recall-provoked confabulation (alterations or intrusions in recall content) and question-provoked confabulation (in response to specific questions).

There are various theories of confabulation. Theories of context memory confusion [33] propose that confabulations result from a tendency to retrieve memories out of context, usually out of temporal sequence. More recently, this has been described as a failure in reality orientation [34, 35]. Secondly, and somewhat related, is Dalla Barba's [36, 37] argument that temporal consciousness is intact but malfunctioning. Thirdly, there are theories emphasising a failure to specify the trace to be recalled, and/or to edit out errors [38-40]. Fourthly, there are motivational accounts

[41-43], which emphasise the relative preservation of pleasant memories. Finally, there are interactionist accounts, which combine a number of different factors [44, 45].

There appears to be agreement, however, that spontaneous confabulation arises in cases where executive dysfunction and frontal lobe damage are present alongside an amnesic deficit [46-49]. Many studies have emphasised lesions in either the ventro-medial frontal or orbito-frontal regions [34, 40, 50-53].

In a recent study, Bajo *et al.* [54] examined the correlates of the occurrence of confabulation and its decline through time. We wanted to investigate which cognitive and emotional factors are associated with the presence and course of confabulation. The study investigated: first, whether temporal context confusions were predictive of confabulation and its time course (the Schnider hypothesis); secondly, whether affective factors (mood state) or lack of insight were predictive of confabulation and its time-course, as suggested by Corwin *et al.*'s [55] findings; and thirdly whether autobiographical memory and executive performance were predictive of confabulation and its time course, a more traditional notion found in a single case by Johnson *et al.* [44].

Twenty four confabulating patients were compared with 11 non-confabulating brain-injured participants from the same Rehabilitation Centre, and 6 healthy controls. Twenty of the confabulating patients had acted on their confabulations. Participant groups were matched for mean age, gender, and years of education. The patients were seen approximately 6 months after onset (baseline), and were followed up at 3 months (9 months post-onset), and at 9 months (15 months post-onset). Of the 21 patients for whom imaging was available, 18 had some kind of pathology in either the ventromedial or orbitofrontal regions. Underlying aetiologies in the confabulating group included hypoxic brain damage ($n=10$); traumatic brain injury ($n=6$); subarachnoid haemorrhage ($n=4$); Wernicke-Korsakoff syndrome ($n=2$); cerebral infection ($n=1$); and tumour ($n=1$). For the non-confabulating group, underlying pathologies were hypoxic brain damage ($n=2$); traumatic brain injury ($n=6$); subarachnoid haemorrhage ($n=1$); cerebral infection ($n=1$); and tumour ($n=1$).

Participants were given tests to estimate premorbid and current IQ. The Wechsler Memory Scale-III (WMS-III) [56] was used as a measure of anterograde memory, and the Autobiographical Memory Interview (AMI) [57] for autobiographical memory. Executive tests included the Trail-making test, the Hayling and Brixton Tests, and the cognitive estimates test (references as in Bajo *et al.* [54]). We also included measures of elated mood, depressed mood and insight (a 7 item Insight interview) [54]. We used Dalla Barba's [36] Confabulation Battery (the version adapted for the UK by Kopelman *et al.* [45]) to look at confabulations in personal semantic memory, episodic memory, orientation in time and place, and general semantic memory. Finally participants were given the original version of Schnider's temporal context confusion (TCC) test [33]. In this test, participants were shown line drawings of 120

objects and animals. Some of these drawings were repeated (targets), and others were shown just once, acting as distractors. Participants were asked to state whether they had seen each item before. Following a 20 minute time gap, previous targets became distractors and 8 of the previous distractors became targets. Participants were again asked to state whether they had seen a given item in this 'run' or not.

In terms of performance on the TCC task at baseline, the majority of the confabulators (17 of 24) did indeed fall above Schnider *et al.*'s [33] suggested cut-off of 0.28, whilst all 6 of the healthy controls fell below it. However, the scores of the non-confabulating brain-injured patients were evenly distributed across both sides of the critical cut-off point, overlapping with both the confabulators and the healthy controls. Gilboa *et al.* [40] found a similar range of scores in another group of non-confabulating brain injured patients. Taken together, these findings indicated that TCC performance cannot reliably discriminate confabulating from non-confabulating memory-disordered patients.

We also looked at the correlates of the severity of confabulation (within the confabulating group), based on the Dalla Barba confabulation interview [36]. There was a near-significant correlation between TCC score and total confabulations ($r=.34$, $p=.05$), though not with the number of episodic confabulations ($r=.01$, $p=.48$). Significant and stronger correlations were observed between the total number of confabulations and AMI episodic memory scores for early adult ($r=-.45$, $p=.02$) and recent life ($r=-.48$, $p=.02$), logical memory immediate ($r=-.41$, $p=.03$), and delayed ($r=-.40$, $p=.03$). Error scores on both cognitive estimates ($r=.43$, $p=.02$) and the Hayling test ($r=.49$, $p=.01$), also correlated significantly with the number of episodic confabulations. A stepwise regression based on AMI episodic 'recent life', TCC, and AMI episodic 'early adulthood' scores predicted 49% of the variance (R^2) on total confabulation scores.

The resolution of confabulations was examined over a further 9 months. As expected, there was a steep decline in the proportion of total memory statements and proportion of episodic memories that were confabulations at 3 and 9 months. Scores on the TCC measure decreased in parallel, as did scores for lack of insight and elated mood. Depressed mood scores show an inverted U-shape, increasing slightly at 3 months, but falling again at 9 months. In terms of correlates of the decline in confabulations across individual patients, none of these measures (TCC, elated mood, depressed mood, or insight) correlated significantly with the decline in confabulation (as indicated by difference scores between baseline and 9 months). What did correlate significantly were changes in autobiographical memory scores (AMI episodic childhood, $r=-.43$, $p=.025$; AMI episodic early adulthood, $r=-.63$, $p=.001$) and changes in number of errors on the cognitive estimates test ($r=.46$, $p=.02$). Change in delayed logical memory scores also correlated with decline in episodic confabulations ($r=.40$, $p=.03$). A stepwise regression based on change in AMI episodic

young adulthood score and change in cognitive estimates error scores predicted 44% of the variance (R^2) of change in total confabulation scores over 9 months [54].

In summary, we have argued that temporal context confusions (TCC) are sensitive, but not specific, to confabulation. TCC and insight both improved in parallel with confabulation, but they were not significantly correlated with change in confabulation scores. It was memory, particularly autobiographical memory, and executive function (as measured by cognitive estimate errors) that were the strongest correlates of both the initial severity of confabulation and change in confabulation scores over 9 months. This supports the view of Johnson *et al.* [44], who had suggested on the basis of a single case, that weakness in autobiographical memory and executive dysfunction provokes and/or promotes confabulation.

■ Psychogenic amnesia

Psychogenic amnesia is another 'anomaly' of autobiographical memory. Kopelman [58] has distinguished between global psychogenic amnesia, where people lose memories for their entire life together with their sense of personal identity, and situation-specific gaps in memory, such as occur in post-traumatic stress disorder.

A psychogenic fugue state can be defined as a sudden loss of memory, involving the loss of all autobiographical memories and the sense of personal identity [59]. This is usually associated with a period of wandering, and typically lasts for a few hours or days up to about 4 weeks. Upon recovery there is an amnesic gap for the period of the fugue. If the amnesia persists, it should be labelled as psychogenic focal retrograde amnesia, although, in such cases, one should always consider whether the patient is simulating.

Focal retrograde amnesia (FRA) is a term coined by Kapur [60], which describes retrograde amnesia in the absence of any anterograde loss. This is sometimes initially accompanied by a transient loss of personal identity but, unlike fugue, the memory disorder persists. FRA often (but not always) follows a mild concussion or other cerebral event. In early reports, it was assumed to reflect underlying brain pathology, but neuroimaging is usually normal. Some of the cases reported in the literature were not in fact focal, because they also showed anterograde memory loss, and many, if not all, such cases may well be psychogenic [61]. Harrison *et al.* [59] have demonstrated that there are at least two routes to a persisting focal retrograde amnesia. Some follow a mild head injury or cerebral event where, in a particular psychological context, the focal retrograde amnesia becomes persistent. Others begin as a fugue-like episode, in which, if not appropriately treated or if the behaviour is reinforced, the amnesia becomes persistent.

The clinical literature suggests that predispositions to psychogenic amnesia include severe precipitating stresses, such as marital problems, financial crisis, offences, war, or bereavement. There is very commonly a preceding

depressed mood, sometimes associated with suicidal ideas. There is also frequently a history of a transient 'neurological' amnesia, for example a head injury, alcohol 'blackout', or epilepsy, which may operate as 'learning experiences' [58].

Harrison *et al.* [59] reviewed 53 cases seen by Prof Kopelman during the course of 20 years. These cases were classified into 4 sub-groups. 16 were classified as psychogenic fugue; 16 were labelled as psychogenic focal retrograde amnesia (FRA), for example following a minor head injury; 16 began as a fugue-like episode but then the amnesia persisted (F-FRA); and the final group of 5 cases described gaps in their memory which related (directly or indirectly) to stressful life events. Using the case records and the neuropsychological test scores, these patients were compared with 21 neurological memory-disordered patients and 14 healthy controls. We determined predisposing factors, finding that clinical depression, a failure to recognise family members, family or relationship problems, financial or employment problems, and a history of PTSD, were all significantly more common in psychogenic than neurological amnesia. Interestingly, a past history of head injury was also significantly more common in the psychogenic group (41% versus 10%), perhaps predisposing individuals to developing psychogenic memory loss at a later time of severe stress.

Scores on anterograde memory tests were obtained during the acute episode and at 3-6 months follow-up. These tests included measures of visual and verbal recall and recognition memory. Patient scores, converted into IQ-type quotients, were typically 10-15 points lower at onset than 3-6 months later. This improvement was statistically significant for verbal recall memory.

Figure 2 shows autobiographical episodic memory and personal semantic memory performance on the AMI, both during the amnesic episode (a,c) and at 3-6 month follow-up (b,d). With regard to personal semantic memory for facts, fugue patients could recall very few personal facts across all time periods during the amnesic episode, showing a 'flat' temporal gradient, relative to healthy controls. The temporal patterns of the two FRA groups were virtually identical: both showed a recency effect with poor memory for facts from their childhood or early adulthood, but relative sparing of more recent memories. This is opposite to the pattern typically observed in Harrison *et al.*'s neurological patients [59], and in studies of transient global amnesia (compare also figure 1). At follow-up, the fugue group had improved to normal, scoring at the same level as controls. The two FRA groups had also improved, although they were still impaired, relative to controls, and still showed a 'reversed' temporal gradient.

In terms of recall of autobiographical incidents or episodes, the fugue group were again severely impaired across all time-periods, compared with controls, and the two FRA groups again showed a 'reversed' temporal gradient. At follow-up, the fugue group had substantially improved: they were no longer significantly different from controls, although their scores were still slightly below

Figure 2. Personal semantic and autobiographical episodic memory performance on the AMI during the amnesic episode and at 3-6 month follow-up: From Harrison *et al.*, *Brain*, 2017.

normal. The two FRA groups had also improved, but still showed a 'reversed' temporal gradient.

In summary, in fugue cases memories returned to normal for personal semantic facts at follow-up, and to near-normal for episodic incidents. There was a lesser and more variable improvement in focal retrograde amnesia, and these patients retained a 'reversed' temporal gradient. One interpretation of this is that it reflects a process of inhibition in memory retrieval during the amnesic episode.

Figure 3 shows a model of social factors and brain systems influencing autobiographical memory retrieval and personal identity [58]. Given the combination of a severe precipitating stress, depressed mood, and/or a past history of transient neurological amnesia, frontal inhibitory mechanisms operate to prevent the retrieval of autobiographical memories. A negative feedback loop is proposed such that, in extreme cases where the sense of personal identity is lost, the patient now appears affectless and perplexed, rather than depressed. During a fugue state, the medial temporal

system appears to be working normally, allowing the person to operate in his/her environment. However, this system cannot be functioning completely normally because, when people come out of a fugue, they have no memory for what they had been doing during the fugue episode (see also [59]).

This model is broadly consistent with fMRI studies of memory suppression [62, 63]. For example, Anderson *et al.* [63] found activation of the ventrolateral and dorsolateral frontal regions during memory suppression, alongside deactivation of medial temporal regions. Kikuchi *et al.* [64] found a similar pattern in two psychogenic cases, one of whom we would describe as being in a fugue state, and the other was a case of 'psychogenic focal retrograde amnesia'. During the presentation of photographs of personal acquaintances that these patients should have recognised, but did not, there was increased activation in the prefrontal cortex and decreased activation in the hippocampus. This pattern disappeared in the fugue patient when memories recovered,

Figure 3. Social factors and brain systems influencing autobiographical memory retrieval and personal identity. The figure shows social factors (in ovals) and brain systems (in rectangles) involved in psychogenic amnesia: inhibition leads to impaired retrieval of incidents and facts, and severe inhibition affects orientation in person. From Kopelman, *Brain*, 2002.

but remained unchanged in the FRA patient whose amnesia persisted.

In cases of psychogenic memory loss, there is always the question of whether the amnesia is deliberate or unconscious. The early Freud [65] wrote that:

"[In] psychical trauma...it was a question of things which the patient wished to forget, and therefore intentionally repressed from his conscious thought and inhibited and suppressed...In my view this intentional repression is also the basis for the conversion..."

However, by the time he wrote his Introductory Lectures [66], Freud's views had changed. He now considered it an unconscious process:

"If they [memories] are inadmissible to consciousness; we speak of them as repressed...Repression consists in its not being allowed...to pass from the system of the unconscious into that of preconscious."

Based on his experience in treating First World War officers suffering from shell shock, Rivers [67], wrote that:

"Many kinds of mental experience may be repressed. Thus, after one of my patients had for long baffled all

attempts to discover the source of his trouble, it finally appeared that he was attempting to banish from his mind feelings of shame due to his having broken down...In another case an officer had carried the repression of grief through the war to the point of suppression, the suppressed emotion finding vent in attacks of weeping, which came on suddenly with no apparent reason...In nearly every case...they were either deliberately thrusting certain unpleasant thoughts from their minds, or were occupying every moment of the day in some activity that these thoughts might not come into the focus of attention."

These descriptions were more compatible with the early Freud than the later Freud; and they are also consistent with Anderson *et al.*'s [62, 63] studies of memory inhibition and memory suppression. In reality, it is very difficult, if not impossible, to know the extent to which people are deliberately avoiding painful or difficult memories, or the extent to which this is a 'truly' unconscious process. Patients make statements such as:

'It's like a box locked away, and I don't really want to open it.'

'I put things in boxes, I choose to put them in the back of my mind. I've always done that. I know the memories are there, but I cannot get access to them.' [59].

In summary, psychogenic amnesias can be interpreted at different levels, cognitive, neurophysiological (imaging), or psychodynamic. They involve the avoidance of painful or unpleasant memories, with varying degrees of conscious awareness. Frontal inhibitory control mechanisms may well be implicated, and there is some functional imaging evidence to support this.

Conclusion

With respect to retrograde amnesia, we have queried whether the 'main' theories explain an extensive retrograde amnesia with a temporal gradient, or the (variable) pattern of findings across episodic and semantic memory. Frontal and lateral temporal damage can contribute to retrograde amnesia in addition to hippocampal pathology. We have argued for a retrieval deficit operating across both personal semantic and episodic autobiographical memories, with a temporal gradient that is secondary to age effects at encod-

ing, including the autobiographical memory 'bump'. On confabulation, we have described an association between spontaneous confabulation and impairments at tests of executive function and autobiographical memory, possibly the consequence of a damaged 'filter' in the ventro-medial and orbito-frontal cortex [54]. In psychogenic amnesia, we have argued for the 'frontal' inhibition of autobiographical memory retrieval with subsequent improvement or recovery [59], and we have cited neuroimaging findings that support this hypothesis. Taken together, these observations point to the importance of frontal and executive systems in the retrieval of 'old' memories. ■

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Conflicts of interest

None

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