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SHORT COMMUNICATION

Psychogenic amnesia and self-identity: a multimodal functional investigation

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Background: Patients with psychogenic amnesia generally suffer from episodic memory deficits associated with an impairment of self-identity. While the first is generally attributed to limbic dysfunction, the latter might be related to posterior parietal cortex.
Methods and Results: In a patient with acute repetitive psychogenic amnesia, three different functional investigations (fMRI, electrical-neuroimaging, PET) during both resting-state and a behavioural paradigm testing 'time-travel' showed left posterior parietal activation, unlike in 12 control subjects.
Conclusion: Impairment of self-identity and episodic memory in psychogenic amnesia may be associated with functional alterations of left posterior parietal cortex.

Introduction

Psychogenic amnesia (PA) is defined as an impaired access to episodic memories for a certain time period (or consolidation of these memories) in the context of psychogenic stress [1–3]. Theories about PA highlight the phenomenological and neuropsychological similarity between PA and organic amnesia and relate PA to dysfunction in the limbic memory system [2–4]. These theories also mention emotional stress as leading to PA, likewise related to activity in the limbic system. However, unlike patients with organic amnesia, PA is often accompanied by sudden 'loss of personal identity' [5,6] and might therefore be related to dysfunction in more posterior, self-related, brain regions. Here, we present a patient with acute repetitive retrograde PA accompanied by a 'loss of identity'. The patient was investigated with three different functional tests and three different paradigms. All these tests found altered brain activity in the left posterior parietal cortex (PPC), suggesting that a common deficit leads to self-identity impairment and amnesia in this patient.

Patient

The patient was a 30-year-old single Hispanic woman, who had lived in Europe for 3 years. Shortly after moving in with her new partner, the patient presented once every several weeks with acute and repetitive, paroxysmal events in which for several days, she felt that she would 'lose her own identity' and be displaced in time back to her moment of arrival in Europe. During the acute phase, she had no evidence of episodic memory (personal and non-personal) for the prior 3 years and also showed a marked change in personal identity. The episode then resolved spontaneously.

The patient's early history was significant for sexual abuse by her grandfather when she was 8 years old. She held a high-level job and was involved in many social activities. Some months after moving in with her partner, she broke up, stating that she 'did not feel satisfied'. She moved to Europe, where she met a new person with whom she moved in 1 year later. Following this, she began to suffer from amnesic events, in which she did not recognize local acquaintances and did not speak the local language (which she in fact spoke fluently). Additionally, during the acute amnesic period, the patient also showed a marked childish personality, dependent and alexithymic, much different from her usual considerate, mature and serious one. Later, she mentioned that during amnesic epochs, 'I lost the sense of being myself' and that she would like to 'travel back' to the time 'when I was myself'.

Neuropsychological examination during her amnesic ictus showed an amnesic lacune with remarkable deficits over the prior 3 years for the retrieval of episodic memories, as was also shown by the Autobiographical Memory Interview [7] (Table S1). Various verbal and

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visual memory testings were superior to the norm (Table S1). Her neurological status was unremarkable. Prolonged video-EEG examination during amnesic and non-amnesic periods showed no abnormality. MRI examination was unremarkable, as were various blood tests and toxin screen. In an in-depth psychiatric evaluation, the patient was diagnosed as having episodic PA in association with an intrapsychic discordance stemming both from recent interpersonal difficulties as well as from her childhood abuse. No other psychiatric conditions were found. Following a brief psychological consultation, the patient decided to return to her homeland and re-construct her relationships with her old partner. Since then (~2 years), she has had no further events.

Multimodal functional investigation

To investigate her PA, the patient underwent three studies (with three paradigms and methods) during a psychogenic amnesic event in compare to 12 healthy subjects. All participants gave written informed consent before inclusion in the study, which was approved by the Ethical Committees of Geneva University Hospital and the University Hospital of Lausanne (Switzerland). The data were rated blindly by three investigators.

1. Comparison of events from amnesic and non-amnesic periods using fMRI: The patient and 12 healthy age-matched control subjects were presented with 64 events from four different time periods, one of which corresponded to the patient's amnesic period (Fig. 1a; Data S1). Patient and controls were asked to mention whether the presented events had already happened or were supposed to happen in the future. While the patient managed to respond correctly to all the presented events, her reaction times were significantly higher for events that had happened in the amnesic period (amnesic period: 1052.3 ± 56.4 ms; others (average): 833.4 ± 53.4 ms), unlike control subjects (corresponding periods: 598.3 ± 41.2 ms and 576.4 ± 40.1 ms, respectively; Fig. 1b). fMRI contrasting the amnesic period with the previous non-amnesic period showed hyperactivation in the left PPC (Fig. 2a; $P < 0.05$, FDR-corrected).

2. Comparison of acute amnesic and non-amnesic periods using topographical electrophysiological analysis in the frequency domain [8]: analysis of electrophysiological signals recorded via 32 surface electrodes during resting-state in an acute amnesic phase as compared with non-amnesic phase using power-frequency analysis and topographical statistical comparison tests (TANOVA; Data S1 online) revealed a significant change in beta power (13–18 Hz) in the left PPC (Fig. 2b). No differences were found to the same analysis conducted on two periods with the same time interval, in 12 control subjects.

3. Comparison of resting-state cerebral FDG-PET imaging to a normal database of 12 healthy controls [9] revealed hypermetabolism in PPC with left predominance, and to a lesser extent in the left inferior frontal cortex (Fig. 2c). Hypometabolism was found in the left inferior temporal cortex.

Discussion

Patients with PA classically suffer from childhood abuse or trauma that may affect brain development [2,3]. This can be reflected in abnormalities of functional neuroimaging during PA. In PA, amnesia is often associated with loss of self-identity, characterized by

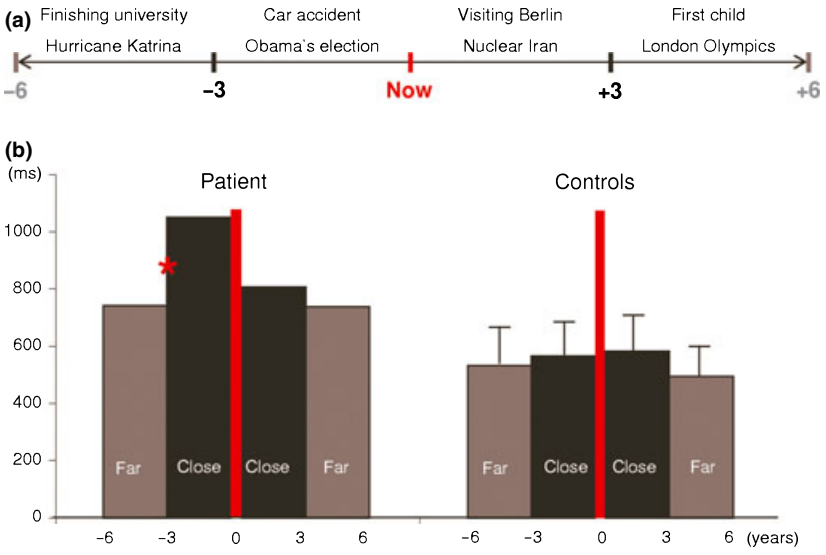

Figure 1 Behavioural study (a) Stimuli and procedure. Participants were asked to judge whether different events in a range of ± 6 years from the present time already happened or are yet to happen. (b) Behavioural results. Reaction times are plotted separately for far-past (−6 to −3 years, grey), close-past (−3 to 0 years, black), close-future (0–3 years, black) and far-future (3–6 years, grey) for patient (left) and controls (right). Patient's results were found significantly higher for close-past than for other periods. No differences were found in the control group.

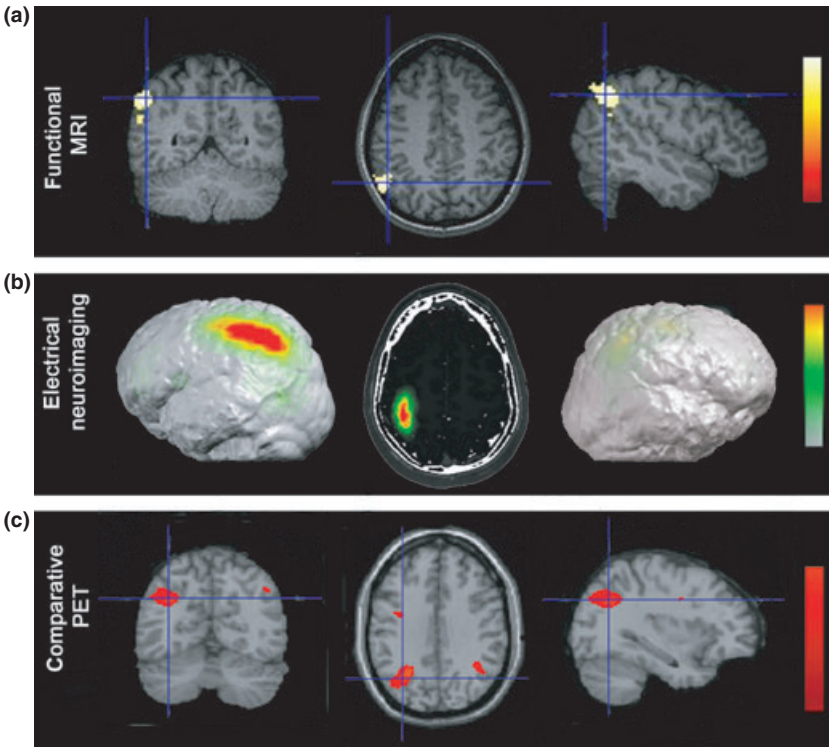

Figure 2 Multimodal functional neuroimaging. (a) fMRI: comparison of events from amnesic an non-amnesic periods showed hyperactivation in the left posterior parietal cortex (PPC). The opposite contrast or contrast of other periods did not show any difference. No such effects were found in the control group. (b) Electrical neuroimaging: comparison of acute amnesic and non-amnesic resting-state periods using topographical electrophysiological analysis in the frequency domain revealed a significant change in beta power (13–18 Hz) in left PPC during acute psychogenic amnesia. (c) Comparative PET: Comparison of resting-state cerebral FDG-PET during acute psychogenic amnesia to a normalized database of healthy subjects revealed hyperactivation in PPC predominant in the left.

changes in personality and behaviour [2–4]. While memory disturbance in PA might be similar to those of retrograde amnesia of organic origin, loss of self-identity is unique for PA [4].

Psychogenic amnesia has been studied in a few functional neuroimaging studies (Table S2). Most of these patients showed right fronto-temporal hypoperfusion as measured by metabolic neuroimaging or metabolic functional studies during a face recognition task (Table S2). The fronto-temporal theory of PA relates self-identity deficits manifested by PA patients to autobiographical memory loss [2–4,6]. However, memory dysfunction cannot account for the remarkable self-identity loss presented by these patients. In addition, our behavioural results show that there was a limited memory deficit in the patient, as she could correctly judge the timing of all events, including those of the amnesic period, even though she could declaratively recall none of them. This pointed to a deficit in a self-related multifaceted autobiographical memory network [10]. Moreover, self-identity involves not only mechanisms of memory but also to other 'self-processing' such as mental imagery, agency or perspective taking [11]. Alteration of PPC activation as found here might reflect both identity and memory-related deficits, as the PPC is implicated in various processing related to 'scaling' and self and its environment such as praxis of actions (agency), visual-perspective taking and self-other distinctions [11]; likewise, this region is known to be implicated in autobiographical memory [12,13]. In our case, the patient's desire to 'travel back' in time suggests an active inhibition of more recent unwanted memories which might be reflected by PPC hyperactivation. In conclusion, although this study investigates a single patient, and the fMRI study used a 'time travel' paradigm for events and not for personal identity, it suggests that hyperactivation in PPC might account for both identity and memory deficits in PA.

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Disclosure of conflicts of interest

The authors declare no financial or other conflict of interests.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Data S1. Methods.

Table S1. Neuropsychological evaluation.

Table S2. Functional studies of psychogenic amnesia.

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References

1. Botzung A, Denkova E, Manning L. Psychogenic memory deficits associated with functional cerebral changes: an FMRI study. *Neurocase* 2007; **13:** 378–384.
2. Kopelman MD. Disorders of memory. *Brain* 2002; **125:** 2152–2190.
3. Markowitsch HJ. Psychogenic amnesia. *Neuroimage* 2003; **20**(Suppl. 1)**:** S132–S138.
4. Reinhold N, Markowitsch HJ. Retrograde episodic memory and emotion: a perspective from patients with dissociative amnesia. *Neuropsychologia* 2009; **47:** 2197–2206.
5. Kopelman MD, Christensen H, Puffett A, Stanhope N. The great escape: a neuropsychological study of psychogenic amnesia. *Neuropsychologia* 1994; **32:** 675–691.
6. Markowitsch HJ. Functional neuroimaging correlates of functional amnesia. *Memory* 1999; **7:** 561–583.
7. Kopelman MD. The Autobiographical Memory Interview (AMI) in organic and psychogenic amnesia. *Memory* 1994; **2:** 211–235.
8. Arzy S, Allali G, Brunet D, Michel CM, Kaplan PW, Seeck M. Antiepileptic drugs modify power of high EEG frequencies and their neural generators. *Eur J Neurol* 2010; **17:** 1308–1312.
9. Brand M, Eggers C, Reinhold N, *et al*. Functional brain imaging in 14 patients with dissociative amnesia reveals right inferolateral prefrontal hypometabolism. *Psychiatry Res* 2009; **174:** 32–39.
10. Arzy S, Collette S, Ionta S, Fornari E, Blanke O. Subjective mental time: the functional architecture of projecting the self to past and future. *Eur J Neurosci* 2009; **30:** 2009–2017.
11. Vogeley K, Fink GR. Neural correlates of the first-person-perspective. *Trends Cogn Sci* 2003; **7:** 38–42.
12. Levine B. Autobiographical memory and the self in time: brain lesion effects, functional neuroanatomy, and lifespan development. *Brain Cogn* 2004; **55:** 54–68.
13. Cabeza R, Ciaramelli E, Olson IR, Moscovitch M. The parietal cortex and episodic memory: an attentional account. *Nat Rev Neurosci* 2008; **9:** 613–625.