Letters to the Editor

The ‘Petites Madeleines’ phenomenon in two amnesic patients. Sudden recovery of forgotten memories

Narinder Kapur

Department of Clinical Neuropsychology, Wessex Neurological Centre, Southampton General Hospital, Southampton, UK

Correspondence to: Dr Narinder Kapur, Department of Clinical Neuropsychology, Wessex Neurological Centre, Southampton General Hospital, Southampton SO16 6YD, UK

I was interested to read the report by Luchelli et al. (1995). Their paper is a welcome contribution to the debate on the nature of focal retrograde amnesia. I would like, if I may, to offer a few comments, and also to mention a similar case which may help to shed light on the mechanisms underlying the memory disorder that they reported.

It would seem that both patients reported in Luchelli et al. (1995) had suffered a loss of personal identity. Case G.R. ‘experienced a sense of uncertainty about his identity ... did not know his occupation ... he could not say whether he used to smoke or not ... when shown pictures of his family members, relatives and acquaintances, he failed to recall anything about them’ (1995, p. 168). It is not specifically stated in the report whether or not case G.R. knew his own name, but these statements indicate a significant loss of personal identity.

Case M.M. had a more clear-cut loss of personal identity. ‘He realised that he did not know who he was and failed to recognize relatives when they arrived at the hospital’ (1995, p. 172).

In my experience, loss of personal identity which is so severe that the person does not know his own name is usually psychogenic in origin. While loss of personal identity may very rarely occur in cases of brain pathology (Glees and Griffith, 1952; High et al., 1990), this is usually in the context of the acute stages of a very severe insult to the brain. The brain injury to case M.M. appeared to be mild. It is difficult to comment on some of the CT scan and PET scan changes that were reported in the absence of relevant scan figures. Case G.R. undoubtedly had a more serious insult to the brain, though it did not appear to be very severe. I would therefore interpret the loss of personal identity as shown by these two cases as being psychological in origin. The co-existence of psychological and cerebral aetiologies in a case of marked memory disorder is, of course, uncommon but not at all rare. The case described by Stuss (Stuss, 1993; Stuss and Guzman, 1988), and also the cases described by Binder (1994), probably fall into such a category. The occurrence of transient global amnesia (TGA) after minor trauma needs to be borne in mind (Haas, 1990). Likewise, the selective loss of autobiographical memory in classical TGA and trauma-related TGA also needs to be remembered (Evans et al., 1993; Stracciari et al., 1994). However, neither of the present cases would appear to fall into such categories.

Two other features of the Lucchelli et al. (1995) cases lead me to favour a psychogenic basis. The first is the sudden return of past memories. My own experience is that, where shrinkage of retrograde amnesia occurs, cases of neurologically based retrograde memory loss show a gradual rather than sudden return of past memories, while the latter form of recovery pattern is usually found in cases of psychogenic retrograde amnesia. A classic case of psychogenic retrograde amnesia, with sudden return of past memories, is that described by Schacter et al. (1982). The second feature, which applied to case M.M., is his loss of memory for how to use previously familiar devices and tools. In my experience, loss of such knowledge without the presence of definitive cerebral pathology is usually supportive of a psychogenic rather than a neurological explanation for a memory disorder—support for this view is also found in the paper by Binder (1994).

In the case of psychiatric factors that may account for this type of amnesia, I do not think that it is sufficient to use the absence of any apparent pre-existent conflict, secondary gain, etc. as a means of excluding the presence of contributory psychiatric factors in such cases. While the same argument could apply in the case of possible neural mechanisms that underly such conditions, it is important in such cases initially to pursue the more parsimonious and more probable explanation.

I have recently seen a case of loss of personal identity in the context of a dense retrograde amnesia, where there was a sudden return of past memories. There was no evidence of conscious malingering, nor could any cerebral basis to the condition be found.

The patient was a 37-year-old female (date of birth, January 1, 1958) who was hit on the head by a piece of shelving while working in a secluded part of a supermarket store, this
event occurring in July 1994. A skull X-ray and brain scans were normal. She was alone at the time of the accident, and it is unclear whether or not she lost consciousness. The patient indicated that in the weeks and months after her accident she had complete loss of memory for her past life. She did not recognize her husband nor her child. She indicated that when she was in hospital, she did not know her surname, nor where she lived. She did not know that her mother had died 2 years earlier and she had to go through a grieving process again. She could not recognize her own clothes at home. She did not know where to put things in her kitchen cupboards. She had to relearn how to drive a car. Her handwriting changed such that she printed in upper-case letters.

When she was assessed by me in January 1995, she could recall waking up and feeling cold in the area where the accident took place. Her memory for the first day of the accident is rather fuzzy, but her memory for subsequent days is fairly clear. Thus, it appears that she had a post-traumatic amnesia of 1 day. In the case of her pre-injury memories, at the time of this assessment she could recall attending a dance in March 1994, but no events between that episode and the accident 4 months later. She initially lost her sense of smell—this loss lasted for 4 months, and then it gradually returned. On neuropsychological testing, she showed mostly mild impairment in a few cognitive tasks, primarily those relating to anterograde memory.

Her husband’s account is as follows. When he first visited her in hospital, she did not recognize him. She did not know her age (giving it as 23 years, 14 years less than her true age), nor did she know her address. He thought that she probably did not know her own name, but he is unclear if he specifically asked her. She tried to identify him using the name of her previous husband. She did not recognize other members of the family. Her husband thought that, during her hospital stay, her everyday short-term memory was intact. She also did not know general facts such as those relating to World War II, the current Prime Minister and the current US President. Routes and buildings that had previously been familiar to her were now unfamiliar. Looking through family photographs did not bring back any memories to her.

There was a relatively sudden return of her past memories on the morning of November 15, 1994. The previous evening she had been watching a film about World War II. Her father, who was Polish, had served in the war. She remembered a song that was being played on the programme, and she could not get the song out of her mind. That night she was rather restless. The next morning, she suddenly regained true recognition of her daughter, and memories relating to her husband also came back. Over the following months, most of her lost memories returned.

This case has a number of similarities to the cases described by Lucchelli et al. (1995). I think that a complex explanation in the form of ‘neuronal patterned matrices’ may not be necessary for such cases and that a more parsimonious psychogenic explanation may be appropriate. I appreciate that this begs the question as to what one means by the term ‘psychogenic’, and what are the specific psychological mechanisms that could be present in the Lucchelli et al. (1995) cases. While in my experience, most of these mechanisms entail conscious simulation by the patient, other types of cases (such as the one I have presented above) probably do occur. Kopelman et al. (1994) have presented a useful framework that may help to unravel the psychological mechanisms that underlie such non-neurological forms of memory loss.

Finally, it is important to distinguish cases of apparent isolated retrograde amnesia after minor trauma/minimal evidence of definitive cerebral pathology, such as the Lucchelli et al. (1995) cases and other similar cases that have been reported with loss of personal identity or atypical memory symptoms (Andrews et al., 1982; De Renzi et al., 1995) cases which, in my view, probably have a psychogenic origin, with cases that follow major brain injury/brain illness, and which I have designated ‘focal retrograde amnesia’ (Kapur, 1993). The latter cases usually also have some degree of residual anterograde memory impairment, though this is disproportionately mild compared with the retrograde memory loss, and in the early stages of recovery the anterograde memory deficits have usually been quite marked.

While Lucchelli et al. (1995) are commendably cautious in not completely excluding a psychogenic explanation for their patients’ memory disorder, I would regard it as the preferred explanation, compared with one based on putative neural mechanisms.

References
In his letter referring to our paper, Lucchelli et al. (1995), published in Brain (‘The ‘Petites Madeleines’ phenomenon in two amnesic patients. Sudden recovery of forgotten memories’), Dr Narinder Kapur raises the doubt that our two cases of retrograde amnesia (RA) (G.R. amnesic after a left thalamic stroke and M.M. after a very mild head trauma) were inappropriately interpreted as ‘organic’. Instead, Dr Kapur proposes that both are examples of ‘psychogenic’ (or ‘psychological’) RA, and he describes in his letter a new post-traumatic case of RA with a psychological explanation.

We are grateful to Dr Kapur for giving us the opportunity to put forward our updated views on the vexing question of the organic/psychogenic RA-mechanism. The debate on the nature of the underlying mechanism of RA is particularly vivid when the discussion arises from case reports of the ‘pure’ variety of RA (i.e. RA without any other cognitive impairment, mostly in patients without demonstrable brain lesions).

We will offer our views under two headings: a preliminary general statement and our viewpoints on the mechanism underlying RA. Finally, there will be a direct reply to Dr Kapur’s arguments with respect to our cases, G.R. and M.M.

(i) We surmise that RA is the stereotyped clinical outcome of a number of different face-value triggering conditions (aetiologies). They range from vascular and mechanical insults (e.g. in many instances, mild head traumas; E. De Renzi, F. Lucchelli, S. Muggia and H. Spinnler, unpublished results) to psychological conditions. The variety of aetiologies is almost identical to that occurring in transient global amnesia (TGA) (Markowitsch, 1990). The aetiologies trigger a sudden brain dysfunction, whose intrinsic nature remains obscure. Its consequence is RA, namely a memory dissociation roughly akin to the reversed pattern of TGA. We claim that per se the presumed aetiology qualifies neither the mechanism nor the clinical RA syndrome. By analogy, a myocardial infarct triggered by an emotional stress remains an arteriosclerotic accident of the coronary arteries, just as RA is the behavioural outcome of a neuronal dysfunction, irrespective of aetiology.

(ii) The focus of our reasoning is precisely this intermediate level: the mechanisms linking different aetiologies to the clinical RA syndrome. As is the case for TGA, it has to be said that a model linking either a psychological sequence of events or a brain lesion to RA has yet to be developed. The only exception, irrespective of the overall credit it may earn nowadays, is the Freudian hypothesis of ‘Verdrängung’ (repression). This would prevent the access to consciousness of pieces of autobiographical memories, that, after an exhaustive enquiry, can be convincingly suspected to be poorly manageable in the patients unconscious psychological economy. In this view, successful repression may be conceived as the achievement of an ‘unconscious advantage’. In a small number of RA patients a reasonable link between the psychological context and the forgotten pieces of autobiographical memory may be identified (Abeles and Schilder, 1935; Stengel, 1966). With this one exception, the absence of an interpretative model implies the absence of rational diagnostic criteria and, unfortunately, of stringent logical arguments to convince the readers of either the psychological or the organic interpretation. Herein, our aim is to cast doubt on the traditional psychological/organic dychotomy. Actually in our opinion the mechanism underlying RA is the same in all circumstances, and we assign it to the organic domain. It may be consequent to the circumscribed destruction of neuronal networks in brain regions involved in memory with a clear-cut CT and/or MRI evidence [for a review, see Kapur (1993), ‘focal retrograde amnesia’]. This could apply to our case G.R. Admittedly, there is great uncertainty about which neuronal systems are crucial to the retrieval of remote traces [see, for instance, the conflicting evidence from the studies of Hodges and McCarthy (1993), as well as Markowitsch et al. (1993), on one side, and Lucchelli et al. (1995), on the other]. Alternatively, the same mechanism may take the form of a purely dysfunctional deficit, without any apparent neuronal...
destruction (De Renzi et al., 1995), that encroaches upon the same ill-defined neuronal networks speculated above. This could apply to our case MM and, in general, to all ‘pure’ RA cases without evidence of brain damage.

As far as the intimate substrate of the RA-mechanism is concerned, in our paper reference was made, within a connectionistic framework, to the ‘patterned neuronal matrices’, envisaged, in the wake of Penfield and Perot’s (1963) studies, for some memory phenomena of temporal lobe epilepsy by Gloor (1990). We maintain that the essentials of the mechanism underlying RA correspond to the above mentioned neuronal dysfunction. It must also be reminded that the brain structures specifically devoted to the surfacing to consciousness of remote traces are still ill-specified. For instance, are they separable from those subserving retrieval from the remote archives, eventually adding to the surfaced memories their ‘explicit’ trait? It might be that some of the RA cases are linked to the hypothetical dysfunction of this latter level (see, for instance, Freudian repression).

Turning now to Dr Kapur’s letter, we would hesitate to trust for the heuristic role of ‘personal experience’. From the growing debate on the nature of the RA-underlying mechanism (XII European Workshop on Cognitive Neuropsychology, Brixen, January 1995; Memory Disorders Research Society Meeting, Cambridge, August 1995) it seems to us that a circular argument often surfaces. For instance, saying that ‘in my experience, loss of personal identity . . . is usually psychogenic in origin’ (quoted from Dr Kapur’s letter) links a behavioural aspect to a diagnostic statement (i.e. the psychogenic mechanism) which, in turn, was made on a purely a priori basis (i.e. again, personal experience). Actually, a detailed lay-out of the events connecting loss of self identity and the psychogenesis of RA is never provided. In other words, there is a mismatching of ex post facto observations and ex pre facto statements. The same reasoning applies, in our opinion, to the credit given to the extra-autobiographical extension of amnesia. For want of a reliable model, any interpretation of such a clinical observation only on the basis of personal experience is unwarranted. The suddenness of recovery, may find an explanation in the neurophysiological ‘distortion’ of Gloor’s (1990) matrices, whereas a psychological interpretation is flawed by in the same indeterminacy mentioned above. We wonder how attaching the label of ‘psychological’ to RA cases without demonstrable brain lesion could be ‘a more parsimonious and more probable explanation’ than forwarding a hypothetical explanation of the distorted neuronal patterned matrices.

We would like to draw readers’ attention to the value of reporting further cases with ‘pure’ RA irrespective of their presumed psychogenic or organic origin. On the clinical side, having recognized a new syndrome (‘pure’ RA) following minor head traumas is a nosographic achievement (E. De Renzi, F. Lucchelli, S. Muggia and H. Spinell, unpublished observations). The recent cases of Kapur (his letter) and Barbarotto et al. (1996) are possibly two further instances of the dramatic sequelae of very mild head traumas. From a cognitive stance, there are at least two good reasons for studying as thoroughly as possible all occurring RA patients. First, the careful neuropsychological analysis of features of RA patients, together with findings from semantic dementia and post-herpetic patients, are likely to contribute to the shaping of the architecture of the remote memory archives, given the different sensitivity of different remote traces to the amnesic mechanism. Secondly, the characterization of RA in the terms of the uncoupled retrieval of the same memory traces according to explicit or implicit settings may soon go beyond the present anecdotal state (De Renzi et al., 1995; Barbarotto et al., 1996).

By and large, we suspect that the organic/psychogenic distinction is going to lose its traditional sharpness, recognizing the core of the pathogenesis of RA, irrespective of the triggering aetiologies, in a unitary neurophysiological dysfunction of some relevant sets of neurons in the temporal lobes (Kapur et al., 1992). This could also provide further insights into a conceptually very similar and equally obscure memory disorder, namely TGA.

References