SHORT REPORT

Transient memory loss for people

Narinder Kapur, Haider Katifi, Heba El-Zawawi, Michael Sedgwick, Simon Barker

Abstract
A patient had transient memory loss for close family members. She could not even recognise their names as familiar. Her everyday memory was relatively preserved and she retained a clear recollection of the episode. Standard and sleep deprived EEG showed a mild abnormality of the left temporal lobe. Neuropsychological testing found evidence for a mild verbal memory impairment. The findings provide further evidence for the fractionation of transient forms of amnesia, support the dissociation of semantic/retrograde amnesia from episodic/anterograde amnesia, and offer evidence in favour of a left temporal lobe site for retrieval of past memories relating to the identification of people.

(J Neurol Neurosurg Psychiatry 1994;57:862–864)

Sudden, transient retrograde memory loss has been briefly acknowledged in a few clinical reports. In all of these cases, there was a striking contrast with transient global amnesia—namely, that patients subsequently retained relatively clear recollection of the actual period of memory loss, whereas patients with transient global amnesia are invariably left with a complete blank for the period of impaired memory. These cases, however, had a lack of witnessed information a critical factor in the accurate documentation of such forms of memory loss. We report a patient who showed a form of transient memory loss similar to these previous cases, and where the patient had post-ictal preservation of memory for the episode. In our case, the memory loss was witnessed by two people and it also had a more dramatic manifestation—namely, loss of memory for familiar people. We also had the benefit of detailed neuropsychological and imaging studies to complement clinical and neurophysiological examinations.

Case history
Our patient, a 39 year old right handed nursing assistant presented to the casualty department in February 1993 complaining of an episode of amnesia. Two days later she had tingling of the left hand, with blue discolouration together with a cold and heavy feeling. She herself could give a clear account of the amnestic episode. The following account is taken from her recollection, and also from the accounts given by the two witnesses, to the episode—namely, her daughter and her daughter’s boyfriend. From talking to these witnesses, most of her account is accurate apart from impaired memory for a few minor details of the episode such as particular things that the patient said.

She had taken some regular exercise in the morning, and realised that she had forgotten where she had put her handbag—the day previously she had left it in the glove compartment of her car. She went upstairs, and when she encountered her daughter and her daughter’s boyfriend she suddenly realised that she could not identify them, and said “What’s your name?” When told the name (“Clare”), she responded “Clare?” indicating that she did not recognise the name as familiar. It was difficult to elicit from the patient precisely how total was her loss of identification for her daughter—one on the one hand, she indicated that she looked on her daughter and her daughter’s boyfriend as strangers, but she also reported having a “gut feeling” that her daughter was her child, possibly from the fact that she knew she had a daughter, that this was her house, and she believed her daughter when she indicated who she was. Our patient had no loss of personal identity. She realised that she was in her own house, but she did not know her address. When told the names of her younger children (aged 9 and 10 years), she said “Who are they?” She did not recognise these names as being the names of her children, saying that they were “horrible names”. She could not picture her children; nor did she even seem aware that she had these other children. She said that she would never have chosen one of the names (James) for her child, whereas she did in fact choose it herself as it was the middle name of her father. This name was apparently chosen so that her father’s blood would be seen to continue in subsequent families, her son being the first grandson to be born among her immediate family. She did not know the name of her partner (with whom she had been living for seven or eight years); nor did she know what he looked like. She did not know the name of the road where she lived;
nor did she seem to recognise this as familiar when it was spoken to her. She also had some
general word finding difficulties around this
time—for example, she had difficulty retrieving
the word “handbag”. She clearly remember-
ed this difficulty, having to mime the word
she was trying to retrieve, and finding it all
very funny. She then left her house to go to
her sister’s house—she was able to catch the
correct bus, and was able to find her way to
her sister’s house. When she arrived there,
she indicated that she knew the person was
her sister but could relate nothing about her.
We asked our patient if she would have been
able to distinguish her sister from a complete
stranger, and she said “yes”, although it was
difficult for her to make a retrospective judge-
ment on this, especially as there were other
cues—such as the house, furniture, etc—that
would have led her to conclude that she was
in her sister’s house. She was certain that she
could not recollect anything about her, how-
ever, and could not indicate her name. About
an hour later (and three and a half hours after
the start of the attack), the attack suddenly
cleared up. She now became aware of where
her handbag was, and regained familiarity of
her family and friends. Witnesses to the
attack did not consider her to be perplexed or
to have a pronounced anterograde memory
loss during the episode. Her verbal anterograde
memory was considered to be a little
impaired—on two occasions she asked for
information that had already been told to her,
but this was not a continuous, outstanding
feature as occurs in transient global amnesia.
It was not possible for her or her family to say
whether, as well as her loss of memory for
familiar people, there was at the time of the
attack a more general retrograde amnesia for
past events, although she did not display any
behaviour to indicate such a global retrograde
amnesia.

Our patient also recounted an episode
eight years previously that seemed like a tran-
sient ischaemic attack. She had been carrying
out her regular aerobics a few hours previ-
ously, and was in a bingo hall in the evening.
She suddenly found herself incorrectly tran-
scribing the numbers that were being called
out. She also noted having difficulty in under-
standing speech, and in expressing herself.
She went home, went to bed, and by the next
morning the episode had cleared up com-
pletely. Her memory for this episode has
remained clear.

Our patient considered her everyday mem-
ory to be slightly impaired since the most
recent episode, especially for things people
tell her. She had no autobiographical memory
symptoms, and could clearly describe her job
and events that had happened in the past few
years.

Physical examination did not reveal any-
thing of note. In the past, she had had occa-
sional migraine headaches without visual
disturbances. Otherwise, her medical history
was unremarkable. MRI was normal, except
for an incidental finding of a small arachnoid
cyst in the right middle cranial fossa at the tip
of the right temporal pole. It was considered
to be long standing, and having at the most a
mild effect on adjacent temporal lobe gyri.

Neuropsychological investigations
On an adult reading test her score was equi-
alent to an IQ of 103. She was given a short
form of the Wechsler adult intelligence
scale–revised (information, arithmetic, simi-
larities, picture arrangement, block design,
and digit symbol). She had a verbal IQ of 96,
an performance IQ of 89, and a full scale IQ of
93. Her verbal scores were generally within
normal limits, apart from a low aver-
age age-scaled score of 7 on the digit symbol
subtest. On the Wechsler memory
scale–revised, her overall quotient scores were
also in the low average range—she had a ver-
bal memory quotient of 90, a visual memory
quotient of 92, a general memory quotient of
89, a delayed memory quotient of 82, and an
attention/concentration quotient of 80. The
only subtest where she performed less than
two standard deviations below the mean for
her age group was on the initial trials of the
verbal paired associate learning subtest—
13/24 compared with a mean of 20/624 for
her age group. On the recognition memory
test,5 her performance for words was in the
high average range (49/50), and her perfor-
ence for faces yielded an average score
(42/50). She performed normally on a modi-
fied card sorting task, on a picture naming
test, and on a verbal fluency test.

Neuropsychological investigations
A routine 29 channel EEG and a recording
after sleep deprivation both showed intermit-
tent left temporal lobe irregularities. Episodes
of higher than amplitude theta activity
appeared in the record obtained in February
1993 and sometimes formed sharp waves
with phase reversal in the region of the left
anterior temporal lobe. These were still pre-
sent but less pronounced in the sleep depriva-
tion recording done four months later. No
epileptiform activity could be detected in
either recording.

Although the fractionation of chronic
forms of memory loss has now been well doc-
umented,6 there has been much less progress
towards the fractionation of transient forms
of amnesia. This is partly because their brief
duration lends them more to clinical than to
experimental investigation. Nevertheless, the
more common types of transient amnesia,
such as transient global amnesia, and to a
lesser extent transient epileptic amnesia, have
now been well documented.7 Other forms of
transient amnesia remain poorly described.
Our case points to the selectivity that may
occur in transient forms of amnesia. Although
our patient did show some word finding diffi-
culties during the attack, her memory loss for
people cannot simply be due to a transient
episode of expressive dysphasia, as she could
not even recognise family names as familiar
when the names were spoken to her. She also
showed temporary loss of memory for a specific event from the day before (forgetting where she had put her handbag), although it remains possible that this was coincidental and unrelated to the episode. Her attack could not be classified as transient prosopagnosia because she also had total loss of familiarity for the names of her children and of her partner and reported having a "gut feeling" that her daughter was in fact her daughter. Neither did it resemble transient global amnesia or a more general memory loss, as she retained a clear recollection of the event. It seems that her memory loss for people was due to a specific form of "identification amnesia". We suggest that this condition may in part be an analogue of the more chronic forms of memory loss for people that have recently been reported. 8,10 Our patient also seemed to have an inability to evoke visual images to familiar verbal cues, and in this respect our case has similarities to the case of Damasio et al.3 The findings provide further evidence to support the dissociation of retrograde amnesia from anterograde amnesia,11 as our patient had a clear loss of past knowledge with minimal impairment in acquiring new information. These two sets of lost and spared memories differ in a number of respects—it may therefore be more appropriate to simply note the memory loss as one of identification amnesia in the context of normal acquisition of everyday information and events. It is possible that the term "transient semantic amnesia" may prove to be a better description of the condition we have described, and that further cases may substantiate a distinction between transient semantic amnesia and transient retrograde amnesia.

Although a psychogenic basis must always be considered in cases of focal retrograde amnesia, there was no evidence in our patient to suggest that psychogenic factors such as a dissociative reaction played a part in her memory loss. Detailed interviews with the patient and with members of her family failed to yield support for variables such as stress or secondary gain. Her observation during the attack that the names of her children were "horrible names" probably reflected the totality of her memory loss for the names at the time of the attack. It is possible that she herself would never have chosen these names for her children, and that they were only chosen so that her father's lineage could be seen to continue in a grandson. There was no evidence that she had other than a normal relation with her father (after whom one of the children was named). The fact that her amnesic episode was accompanied by clear naming difficulties (for the word "handbag") further suggested that the episode was neurological in nature. The combination of a left temporal lobe EEG abnormality and a verbal paired associate learning deficit provides support for a neurological rather than psychogenic basis to our patient's memory loss.

In the light of our EEG findings showing a left temporal focus, and evidence both from previous similar case studies and from studies of chronic focal retrograde amnesia,11 it seems that left temporal lobe structures play a critical part in the storage and retrieval of past memories. Studies of the combined loss of familiarity of familiar faces and familiar names have tended to implicate more widespread, usually bilateral pathology2— in our patient, she had a "gut feeling" that her daughter was in fact her daughter, and when she met her sister towards the end of the episode, she did not behave towards her as if she were a total stranger. We therefore have to conclude that she had loss of semantic memory that enabled her to provide identification information about familiar people, but cannot say for certain whether it was similar to a prosopagnosia in the sense of absolute loss of familiarity. To this extent, assuming the right middle cranial fossa was active, the association between our patient's memory loss and a left temporal lobe focus would be consistent with most of the evidence that has been published in this area. Because even densely amnesic patients with additional left temporal pole damage show some familiarity recognition for peoples' names,13,14 it is possible that the locus of our patient's damage is elsewhere in the left temporal lobe. From the clinical point of view, our patient's episodes could be regarded as representing atypical transient ischaemic attacks15 or forms of seizures.4 Finally, the role of structures such as the amygdala16 needs to be borne in mind.

We are grateful to Dr G Sterling, under whose care this patient was initially managed, and to Dr S Thompson for assistance with neuropsychological testing.

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*J Neurol Neurosurg Psychiatry* 1994 57: 862-864
doi: 10.1136/jnnp.57.7.862

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