

# Transient epileptic amnesia

Adam Zeman<sup>a</sup> and Christopher Butler<sup>b</sup>

<sup>a</sup>Peninsula Medical School, University of Exeter, Exeter, Devon and <sup>b</sup>Department of Clinical Neurology, University of Oxford, Oxford, UK

Correspondence to Adam Zeman, Professor of Cognitive and Behavioural Neurology, Peninsula Medical School, University of Exeter, St Luke's Campus, Magdalen Road, Exeter, Devon EX1 2LU, UK  
Tel: +44 1392 726151/2;  
e-mail: adam.zeman@pms.ac.uk

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## Purpose of review

Case reports over the past 100 years have raised the possibility that epilepsy can manifest itself in episodes of amnesia. Recent research has established that this is indeed the case, and indicates that characteristic varieties of interictal memory disturbance co-occur with this form of epilepsy.

## Recent findings

Transient epileptic amnesia is a distinctive syndrome of temporal lobe epilepsy principally affecting middle-aged people, giving rise to recurrent, brief attacks of amnesia, often occurring on waking. It is associated with novel forms of interictal memory disturbance: accelerated long-term forgetting, remote memory impairment, especially affecting autobiographical memory, and topographical memory impairment. The seizure focus lies in the medial temporal lobes. The seizures respond promptly to treatment, whereas the interictal impairments generally persist. Further work is required to establish whether the interictal memory impairment is due to physiological or structural disturbance.

## Summary

Transient epileptic amnesia is an under-recognized but treatable cause of transient memory impairment. Accelerated long-term forgetting and autobiographical amnesia, which are invisible to standard memory tests, help to explain the discrepancy between normal test performance and prominent memory complaints among patients with epilepsy. Further investigation of these forms of memory impairment promises to shed light on processes of human memory.

## Keywords

accelerated forgetting, autobiographical amnesia, epilepsy, transient amnesia

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## Introduction

Transient epileptic amnesia (TEA) is a relatively recently characterized syndrome of temporal lobe epilepsy [1]. It gives rise to brief, recurrent episodes of amnesia, often occurring on waking, generally in middle-aged or older people. It is associated with two striking but 'nonstandard' forms of persistent interictal memory impairment, accelerated long-term forgetting and remote memory impairment [2]. The syndrome is of clinical importance, as the amnesic episodes are often misdiagnosed initially but respond promptly to anticonvulsant treatment, and the interictal memory deficits, which are invisible to standard neuropsychological tests, appear to be a common cause of memory impairment in epilepsy generally. It is of theoretical importance as the deficits observed in TEA are providing insights into processes of memory consolidation, storage and retrieval. This review sketches the background of this under-recognized disorder, and reviews recent work on TEA itself, and its

associated interictal memory deficits, accelerated long-term forgetting and remote memory impairment.

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**The syndrome of transient epileptic amnesia**  
Hughlings-Jackson [3] gave one of the earliest descriptions of an episode of TEA. His epileptic patient, Dr Z, who was himself a physician, recorded in his diary an occasion on which he assessed, diagnosed and treated a child with pneumonia following the onset of the characteristic aura of his seizures. Dr Z subsequently realized that he had no recollection of the consultation, but his notes revealed that he had conducted it competently and the seizure had apparently gone unnoticed. The most natural interpretation is that his seizure had disabled his anterograde memory, while sparing other cognitive functions. During the following century several authors described comparable single cases and small series of cases of epilepsy manifesting itself as transient amnesia. These are tabulated in a recent review [2].

In 1991 Hodges [4] reported that in a large cohort of patients who satisfied criteria for transient global amnesia (TGA), 8 of his 114 patients went on to develop temporal lobe epilepsy: these patients were, in retrospect, atypical, with brief, recurrent attacks of amnesia. In the 1990s Narinder Kapur [5,6] coined the term 'transient epileptic amnesia' to describe this subtype of temporal lobe epilepsy presenting with recurrent episodes of amnesia, highlighting the similarities to and contrast with TGA. Zeman et al. [7], in 1998, proposed diagnostic criteria for the disorder (given below), and reported 10 personal cases of TEA together with 21 from the literature. More recently Butler et al. [1] have described a series of 50 cases, and reviewed the world literature [2], which at the time of the review comprised 94 cases in all. The topic has also been reviewed, more briefly, by Bilo et al. [8] and Kosmidis and Papanicolaou [9].

The diagnostic criteria for TEA are as follows [7]:

- (1) A history of recurrent witnessed episodes of transient amnesia
- (2) Cognitive functions other than memory judged to be intact during typical episodes by a reliable witness
- (3) Evidence for a diagnosis of epilepsy based on one or more of the following:
  - (a) epileptiform abnormalities on electroencephalography
  - (b) the concurrent onset of other clinical features of epilepsy (e.g. lip-smacking, olfactory hallucinations)
  - (c) a clear-cut response to anticonvulsant therapy.

The accumulating evidence indicates that TEA is a distinctive syndrome of temporal lobe epilepsy.

**Clinical features of transient epileptic amnesia**  
 Patients with TEA are typically middle-aged, with a mean age at onset of 57 years [2]. Approximately two-thirds are men. The median duration of attacks is 30–60 min, though episodes occasionally last for several hours. Amnesia on awakening is a helpful diagnostic clue, occurring on at least some occasions in around 70% of cases. The amnesia usually involves impairment of both the ability to lay down memories during the episode (anterograde amnesia) and difficulty in retrieving memories from the past (retrograde amnesia). The anterograde amnesia is often partial: 44% of patients report afterwards that they can 'remember not having been able to remember'. Fifty per cent of patients question their companions repetitively during attacks. The retrograde amnesia varies in extent from days to years.

Transient amnesia is the most conspicuous manifestation of the seizures in the great majority of patients with TEA, and the sole manifestation in 34%. In the series described

by Butler et al. [1], 46/50 patients reported at least one attack of which amnesia was the sole feature. However, other manifestations sometimes occur alongside the transient amnesia. In descending order of frequency, these include: olfactory hallucinations, which may have a particularly prominent association with TEA [1]; automatism, most commonly oral automatism, such as chewing or lip-smacking; a brief period of unresponsiveness. Some patients also report seizures in which amnesia is not a prominent feature, usually complex partial seizures. Tonic–clonic seizures occur rarely. Electroencephalography (EEG) reveals epileptiform abnormalities in about one-third of cases, although sleep deprivation prior to recording may increase the yield. Clinical neuroimaging with CT or MRI is usually unremarkable. Anticonvulsant therapy is generally highly effective, abolishing amnesic episodes in the great majority of patients [2].

Eighty-one per cent of patients with TEA, however, complain of additional, interictal, memory impairments which persist despite treatment [2]. These include a tendency for recently acquired memories to fade abnormally rapidly over days or weeks, reported by 44% of Butler et al.'s patients, a patchy but consistent difficulty in recalling the details of salient remote events, such as weddings and holidays, reported by 70% of patients, and problems in recognizing familiar landmarks and remembering familiar routes, reported by 36% of patients. These are discussed further below.

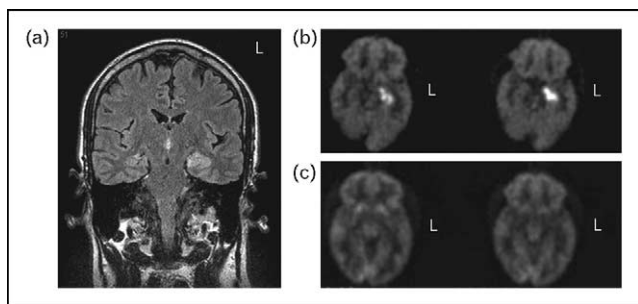
#### Pathophysiology of transient epileptic amnesia

Several features of TEA suggest that the medial temporal lobes (MTLs) are the seizure source. These include the prominence of amnesia in attacks of TEA, the high frequency of olfactory hallucinations and oroalimentary automatism, and the localization of epileptiform activity, when this can be detected by EEG. Additional evidence of a seizure source in the MTLs has come to light recently from neuroimaging.

When structural abnormalities are detected in patients with TEA, they consistently impinge on the temporal lobes [2]. A single case was studied at the time of a flurry of attacks: MRI scanning revealed high signal in the left hippocampus with hypermetabolism in the left hippocampus on peri-ictal PET which had resolved after successful treatment of his seizures (Fig. 1) [10]. As a group, the 50 patients studied by Butler et al. [11] had mild but significant bilateral hippocampal atrophy.

Evidence obtained from EEG monitoring indicates that the amnesic spells of TEA can occur either as an ictal or a postictal phenomenon. Ten cases have been monitored during attacks of TEA [2]. Ictal EEG abnormalities were seen in association with the amnesic attacks in all of

Figure 1 Radiological localization of the seizure focus in transient epileptic amnesia



(a) MRI scan showing high signal on T2-weighted imaging in the left medial temporal lobe of a patient with a flurry of attacks of TEA. (b) PET scan showing hypermetabolism in the left hippocampus at the time of the attacks, (c) with resolution after successful treatment 1 month later. Reproduced from [10].

these: the amnesia coincided with the ictal activity itself in six cases, and followed postictally in four. The EEG abnormalities observed during monitoring were bilateral in 8/10 cases. Interictal epileptiform abnormalities, when present, are also more often than not bilateral – 56% bilateral, 32% left-sided, 12% right-sided [2].

The ultimate cause of TEA remains uncertain and requires further research. Candidates include cerebrovascular disease, neurodegeneration and immune-mediated limbic encephalitis [12], but so far there is no compelling support for any of these possibilities.

Differential diagnosis of transient epileptic amnesia  
 Transient amnesia can occur after head injury, as a result of migraine, following ingestion of a range of substances including alcohol and benzodiazepines, in the context of transient global amnesia [13], transient ischaemic amnesia (TIA) [14], and as a form of ‘functional’ or ‘psychogenic’ amnesia [15]. The occurrence of head injury, migraine or drug use will generally be evident from the history. TGA, TIA and functional amnesia are the conditions most often requiring differentiation from TEA. The distinctive features of the four conditions are summarized in (Table 1).

Standard neuropsychology in transient epileptic amnesia

In most cases, general intellectual functioning is normal in patients with TEA. Indeed the group of 50 patients studied by Butler et al. [1] had an above average mean full-scale intelligence quotient (IQ) of 118. This may reflect the difficulty of recognizing TEA in the absence of an articulate description of its symptoms. There was, however, a mild impairment of anterograde memory on tests of delayed recall and recognition in this cohort. Performance on standard tests of delayed recall was significantly correlated with the volumes of MTL struc-

Table 1 Differentiating features of transient epileptic amnesia (TEA), transient global amnesia (TGA), transient ischaemic amnesia (TIA) and functional (psychogenic) amnesia

	TEA	TGA	TIA	Functional
Typical age	50± years	50± years	50± years	Any age
Duration	Typically <1 h	Typically 4–6 h	Variable	Typically days or months
Ictal memory	Variable mix of anterograde and retrograde amnesia – may later remember attack	Dense anterograde amnesia; variable retrograde amnesia	Not well characterized	Variable, but often dense retrograde amnesia (p/ loss of personal identity) with preserved anterograde memory
Other features (sometimes present)	Olfactory hallucinations, automatisms, brief loss of awareness	Headache, nausea	Focal neurological symptoms and signs, usually arising from posterior circulation	Other functional symptoms and signs may be present; mood disturbance
Precipitants	Waking	Physical (esp. immersion in cold water) and psychological stresses	–	Stressful life events, minor head injury, mood disturbance
Recurrence	Around 1/month	6–10% recurrence rate/year	Recognized but not well characterized	Unusual but occurs in e.g. recurrent psychogenic fugue
Past medical history	No established risk factors	migraine	Cerebrovascular risk factors	Psychiatric illness, ‘organic’ transient amnesia, substance abuse
Interictal/postictal memory	p/ Mild impairment on standard tests; accelerated long-term forgetting, remote memory loss, topographical amnesia	Subtle subclinical impairment may persist for days – months, but no permanent deficit	Risk of permanent memory impairment from completed stroke	Variable

tures [11]. The extent of the deficit – generally less than one standard deviation from the mean – is such that standard neuropsychological tests in individual patients with TEA will often give normal results.

These results were obtained in treated patients who were no longer having seizures: memory impairment, therefore, can occur independently of seizures. Occasionally patients with TEA report that their memory improves on treatment, suggesting that the seizures themselves can also play a role: a recent case report documents objective improvement on standard neuropsychological tests in such a patient [16]. Gallassi [17] has described a related group of patients with the ‘epileptic amnesic syndrome’ who present with persistent memory disturbance in association with relatively subtle temporal lobe seizures. These ‘epileptic amnesic attacks’ are sometimes followed by a period of accentuated amnesia. Gallassi has noted improvement of delayed recall in some patients on treatment of the epilepsy. Hogg et al. [18] reported a group of three patients in whom memory impairment gave rise to an initial suspicion of Alzheimer’s disease. A past history of seizures, concurrent seizures – amnesic in one case – or epileptiform changes on the EEG led to initiation or alteration of anticonvulsant therapy with a marked resulting improvement in memory. Ito et al. [19] and others have described somewhat similar cases [20,21].

The relatively normal performance of many patients with TEA on standard tests of immediate and delayed memory contrasts with their prominent complaints of interictal memory disturbance. The explanation for this lies, at least in part, in the limited scope of standard tests, which, for understandable reasons, fail to test delayed recall at

intervals of more than half an hour so, and rarely probe autobiographical recollection. Recent work, discussed below, has begun to explore these neglected aspects of memory in patients with epilepsy. These ‘nonstandard’ memory deficits correlate more closely with memory complaints in this patient group than performance on standard tests [11].

**Accelerated long-term forgetting**

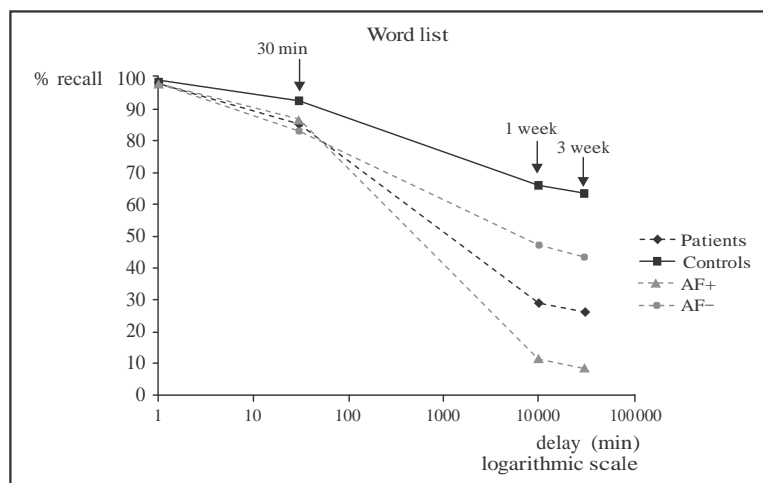
Roughly 50% of patients with TEA complain of rapid fading of memories which they appear to have acquired adequately: this phenomenon has been described as ‘accelerated long-term forgetting’ (ALF). We shall describe the clinical features, and what is known of the cognitive psychology and neurobiology of ALF in turn.

**Clinical features of accelerated long-term forgetting**

In a typical example, a University lecturer was able to discuss the merits of a film he had seen with his daughter on the following day, but 1 week later had no recollection of the movie [10 ]. Patients with TEA as a group show evidence of ALF on objective testing (Fig. 2) [1 ,22]. Those who specifically complain of the problem show especially severe long-term forgetting [1 ].

Accelerated long-term forgetting has also been described and quantified in patients with other forms of temporal lobe epilepsy [2 ]. Whereas the cause of epilepsy in these patients is mixed, they have several features in common. Their temporal lobe epilepsy is not of the typical early-onset form associated with hippocampal sclerosis. They tend to remain able to lead active, independent lives, sometimes remaining in employment, despite their long-term amnesia. They are often

Figure 2 Accelerated long-term forgetting in a group of 24 patients with TEA, 12 of whom complained specifically of accelerated long-term forgetting (AFR), whereas 12 did not (AFS)



Reproduced from [1 ].

seizure-free. In addition to ALF, they show evidence of remote memory impairment, discussed further below. In view of the association between temporal lobe epilepsy and ALF, it is worthwhile to chase the possibility of epilepsy in patients complaining of the accelerated loss of memories [23 ,24].

Accelerated long-term forgetting has not been observed in all studies of people with epilepsy [25–28]. Most studies of ALF, including both those with positive and negative results, are open to methodological criticism. The principle problems are that in some studies ceiling effects may have masked early forgetting, whereas in others varying levels of initial learning in patients and controls complicate the interpretation of forgetting curves.

#### Cognitive psychology of accelerated long-term forgetting

Taken at face value, the loss, over a day to several weeks, of memories which appear to have been acquired normally, suggests a problem with a late stage of memory ‘consolidation’. This view is supported by some individual cases with apparently entirely normal memory acquisition [10 ]. However, in many patients with ALF there is subtle evidence of memory impairment on standard tests or at standard intervals [1 ]. Establishing whether ALF reflects a disturbance of an early stage of memory ‘encoding’ or a later stage of memory ‘consolidation’ requires a deeper understanding of the biological basis of the deficit.

#### Neurobiology of accelerated long-term forgetting

In general, cognitive impairment occurring in epilepsy can be due to epileptic activity in the brain, the underlying pathology, psychosocial sequelae or drug effects [29]. Patients with TEA complain of ALF prior to treatment and in the absence of significant psychopathology [1 ,2 ]. ALF often remains problematic after successful treatment of the amnesic seizures [1 ,2 ], although there are two studies showing improvement in ALF in patients with temporal lobe epilepsy following the abolition of seizures [30,31]. In view of these facts, the most plausible explanations for ALF in TEA are the occurrence of subclinical epileptic activity, perhaps especially during sleep, disrupting the consolidation of recently acquired memories [32 ] or the presence of structural pathology in the medial temporal lobes, a brain region clearly implicated in memory acquisition and consolidation [33,34]. Further research is required to adjudicate between these possibilities.

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### Remote memory impairment

Over 50% of patients with TEA complain of an apparently patchy but striking loss of recall for salient personal

events affecting much of their past lives [2 ]. As with ALF, we consider the clinical, cognitive and neurobiological features of remote memory impairment in turn.

#### Clinical features of remote memory impairment

Typically patients with TEA in whom remote memory is impaired recall that the events in question occurred and they participated in them, but are unable to ‘re-experience’ them in the manner in which we can normally re-evolve autobiographical memories. A recent study confirms that there is a life-long depletion of autobiographical memories, particularly affecting the recollection of episodic detail [35]. Memory for public events was also affected, but to a lesser degree and only for recent decades. A subgroup of patients also complains of difficulty in recognizing familiar places and navigating along familiar routes [1 ].

As is the case with ALF, this pattern of impairment is not unique to TEA, and comparable retrograde memory deficits have been reported in patients with other types of temporal lobe epilepsy [2 ,36–40]. In a review of the literature Butler and Zeman [2 ] identified seven patients with mesial temporal lobe epilepsy, with onset late in life in six, in whom remote memories from the previous 30–40 years were impaired in the face of completely or near-normal standard neuropsychology in five of the seven cases. In most cases public and personal remote memory were both affected. This suggests that in patients with isolated retrograde amnesia, as in patients with isolated complaints suggestive of ALF, it is worthwhile considering the possibility of underlying epilepsy. Some group studies reveal similar findings in temporal lobe epilepsy [39], but, with the exception of studies of TEA, coexisting anterograde memory deficits tend to complicate the interpretation of remote memory impairment.

#### Cognitive psychology of remote memory impairment

In principle, memory impairment for remote autobiographical events could reflect a failure of initial acquisition or consolidation, a degradation of established memories, or a disorder of retrieval. The remote memories affected in TEA were accessible previously, according to patients and relatives, suggesting that an early impairment of acquisition or consolidation is not likely to be the explanation. A detailed single case study demonstrated that the autobiographical amnesia in that case was consistent across time, and could not be corrected by cueing with photographs or written descriptions, arguing against a disturbance of retrieval [41]. This suggests that degradation of established memories is the likeliest underlying cognitive deficit. However, we have recently encountered a patient in whom long lost memories were recovered following a series of episodes of *déjà vu*, indicating that in some cases the problem may indeed lie at the level

of retrieval [42]. ALF, when present, would also be expected to generate a retrograde amnesia over time.

Neurobiology of remote memory impairment

As in the case of ALF, treatment and psychosocial factors are unlikely to be the explanation for remote memory loss in TEA. The two main candidates are physiological and structural: recurrent seizures originating in the MTLs, or subclinical epileptiform activity, propagating through the autobiographical memory network [43] may reset the synaptic weights on which episodic autobiographical memories depend; alternatively, subtle structural damage in the hippocampi may disrupt autobiographical memories. This would be predicted by the Multiple Trace theory of autobiographical memory, which proposes that the hippocampi are required permanently to evoke rich autobiographical memories, but not by the standard model, which holds that memory traces are transferred over time, via ‘systems consolidation’, into neocortical locations in which they are independent of the hippocampi [34].

Conclusion

Transient epileptic amnesia is a distinctive variety of temporal lobe epilepsy causing brief, recurrent, treatment-responsive attacks of transient amnesia, often on waking, generally in middle-aged and elderly people. It is usually accompanied by interictal memory deficits: accelerated long-term forgetting, autobiographical amnesia and topographical amnesia. Several key questions require further research: what is the cause of the epilepsy? Why do attacks occur on waking? Which stages of memory processing are implicated by the interictal memory deficits? Are they a manifestation of subclinical epileptic activity or of structural change in the medial temporal lobes?

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References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

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- of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 707).

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