

Syndromes of Transient Amnesia

For most of us, transient lapses of memory are a familiar and at worst irritating feature of daily life. They are generally brief, item specific and alleviated by a pertinent cue. At the other extreme, the amnesic syndrome is characterised by a profound and usually permanent loss of the ability both to retrieve previously established memories (retrograde amnesia) and to form new ones (anterograde amnesia). A permanent amnesic syndrome is usually caused by extensive bilateral damage to the medial temporal lobes (as in limbic encephalitis or post-hypoxic damage) or to the diencephalon (as in Korsakoff's syndrome), brain regions which play a key role in declarative, 'conscious' memory for facts and events.¹ Some people, however, experience an episode of dense memory loss which is sudden in onset but self-limiting. These syndromes of transient amnesia are the focus of this article.

Transient Global Amnesia (TGA)

The syndrome of TGA has an incidence of 3 to 10 per 100,000 and is characterised by the abrupt onset, usually in middle to old age, of a profound but transient deficit in the retention of new information together with a variable degree of amnesia for past events.² At least 50% of cases appear to be precipitated by a variety of acute stressors including exercise, immersion in cold water, sexual intercourse, pain, or a strongly emotional event. The anterograde amnesia is betrayed by repetitive questioning, usually related to attempts at self-orientation such as "What day is it?" or "What am I doing here?" The retrograde amnesia may cover a few hours prior to the attack onset or be much more extensive. There is no impairment of consciousness or of other cognitive functions such as attention, language or perception, and there are no focal neurological deficits. Spontaneous and apparently complete recovery typical-

ly occurs within 4 to 10 hours, although the individual is left with a permanent amnesic gap for the duration of the attack. Recurrence is rare, with most recent studies reporting a rate of between 3 and 10% per year. A single, uncomplicated episode requires minimal investigation and no specific treatment.

It is widely accepted that the pathological changes in TGA affect the medial temporal lobes, although precisely what those pathological changes are and why they occur is far from resolved. A number of studies have reported increased prevalence of migraine amongst TGA patients³ and migrainous accompaniments, including headache, nausea and vomiting, are not uncommon during the amnesic period. This mechanism alone, however, would not explain the limited age range and rare recurrence of TGA. More recent studies using diffusion-weighted imaging have revealed punctate hippocampal lesions, supportive of a vascular aetiology, in a significant proportion of TGA cases.^{4,5} Interestingly, given the frequency of Valsalva manoeuvre-like precipitants in TGA, it has also been found that patients have a higher prevalence of jugular vein incompetence than controls,^{6,7} lending support to a hypothesis that increased pressure in the superior vena cava causes ischaemia in crucial memory-related brain structures.⁸

Episodes of transient amnesia occurring in the context of epilepsy or head injury and those accompanied by focal neurological symptoms or signs are usually excluded from the rubric of TGA (see Table 1). These are discussed below.

Transient Epileptic Amnesia (TEA)

TEA is a relatively recently described condition in which transient amnesia is the principal manifestation of temporal lobe seizures.^{9,10} The attacks are often mistaken for



Chris Butler is a trainee neurologist from Edinburgh, UK who has recently completed a three-year project investigating the clinical and neuropsychological features of transient epileptic amnesia. He is currently conducting post-doctoral research, with an emphasis on functional imaging in neurodegenerative disease and epilepsy, at the Memory and Aging Center, UC San Francisco, USA.



Adam Zeman is Professor of Clinical and Behavioural Neurology at the Peninsula Medical School, UK. His research interests include the impairment of memory in epilepsy, disorders of cognition and emotion associated with cerebellar disease and the interdisciplinary study of consciousness.

Table 1: Diagnostic criteria

Transient Global Amnesia (Hodges and Warlow 1993)

1. attacks must be witnessed and information available from a capable observer who was present for most of the attack
2. there must be a clear-cut anterograde amnesia during the attack
3. clouding of consciousness and loss of personal identity must be absent, and the cognitive deficit must be limited to amnesia (that is, no aphasia, apraxia, etc)
4. there should be no accompanying focal neurological symptoms during the attack and no significant neurological signs afterwards
5. epileptic features must be absent
6. attacks must resolve within 24 hours
7. patients with recent head injury or active epilepsy (that is, remaining on medication or one seizure in the past two years) are excluded

Transient Epileptic Amnesia (Zeman et al 1998)

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.

Table 2: Characteristic features

TGA

- sudden onset often precipitated by exercise, immersion in water, emotional stress, etc
- dense anterograde amnesia with repetitive questioning
- lasts around 4 – 10 hours
- rarely recurs
- aetiology unknown

TEA

- recurrent, brief (usually < 1 hour) amnesic episodes
- often occur upon waking
- may be associated with olfactory hallucinations or automatisms
- responds to anticonvulsant medication
- persistent memory deficits

Psychogenic amnesia

- history of 'organic amnesia', psychiatric illness and/or substance abuse
- may be triggered by mild head injury or highly emotional event
- extensive retrograde amnesia often with loss of personal identity
- preserved new learning
- duration usually several days at least

Correspondence to:

Christopher R Butler,
Visiting Postdoctoral Scholar in Neurology,
Memory and Aging Center,
University of California,
San Francisco, Suite 706,
350 Parnassus Avenue,
San Francisco,
California 94143, USA.
Email: cbutler@memory.ucsf.edu

Adam ZJ Zeman,
Professor of Cognitive and Behavioural Neurology,
Peninsula Medical School,
Mardon Centre,
Exeter EX2 4UD, UK.
Email: adamzeman@pms.ac.uk

