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Transient global amnesia

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Summary

Transient global amnesia (TGA) has been described since the 19th century. It is characterized by an episode of anterograde and retrograde amnesia lasting less than 24 hours. During this episode patients are conscious and able to interact normally, but are typically distressed and repeat questions without remembering explanations. Despite progress in medical imaging, no cause for the condition has been proven. Prognosis is excellent and management centres on recognition and reassurance. We describe the epidemiology, pathophysiology, clinical features and management.

Key words: transient global amnesia, venous flow, migraine, older people, epilepsy.

Introduction

For over a century there have been records of transient global amnesia (TGA) in the literature, with transient amnesic states suggestive of TGA first described in 1882 by Ribot[1] and in 1909 by Benon.[2] In 1956, further cases suggestive of TGA were described by Bender[3] and by Guyotat and Courjon,[4] and in 1964 Fisher and Adams[5] published a large case series and introduced the term 'transient global amnesia'. Although there are well-described clinical features of the syndrome, the cause of TGA remains unclear, despite advances in imaging.

Search strategy

Medline and EMBASE databases from 1956 to February 2012 were systematically searched with the following terms: transient global amnesia; transient global amnesic states and stroke; transient global amnesia and neuroimaging. 612 articles were retrieved and the abstracts reviewed. If the abstract was not available the title was considered. If the article was not in English it was not reviewed as the great majority were published in the English language and we considered bias from excluding other languages was likely to be minimal. We identified 70 papers to read and their references were reviewed for secondary references. We included randomized control trials, case series, case reports and review articles.

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Epidemiology

The incidence is reported as 3–8 per 100,000 people.[6] A third of these episodes occur in the age group of 50–70 years. The annual recurrence rate is between 6 and 10%.[6] There has been conflicting evidence on gender distribution, with Hodges and Warlow claiming that TGA was more common in men,[6] whilst other studies have shown the opposite.[7] There is emerging evidence that TGA can be hereditary.[8–10]

Clinical features

TGA is characterized by an episode of anterograde and retrograde amnesia lasting less than 24 hours. During this episode patients are conscious and able to interact normally. They are typically distressed and noted to repeat questions without remembering explanations. Although often described, a link between preceding emotional or physical stress and TGA has not been proven. Although these clinical features resolve within 24 hours, mild subclinical neurophysiological deficits with vegetative symptoms can last for days to months.[11] However, a recent meta-analysis of 25 studies showed no difference in long-term cognitive performance between patients and the healthy population.[12]

Common triggers are shown in Figure 1 and include emotional stress (30%), vigorous activity (30%), change in temperature (15%) and sexual intercourse (12%).[13] Quinette *et al.*[14] found an increased frequency of patients with emotional or psychological instability, while Pantoni *et al.*[15]

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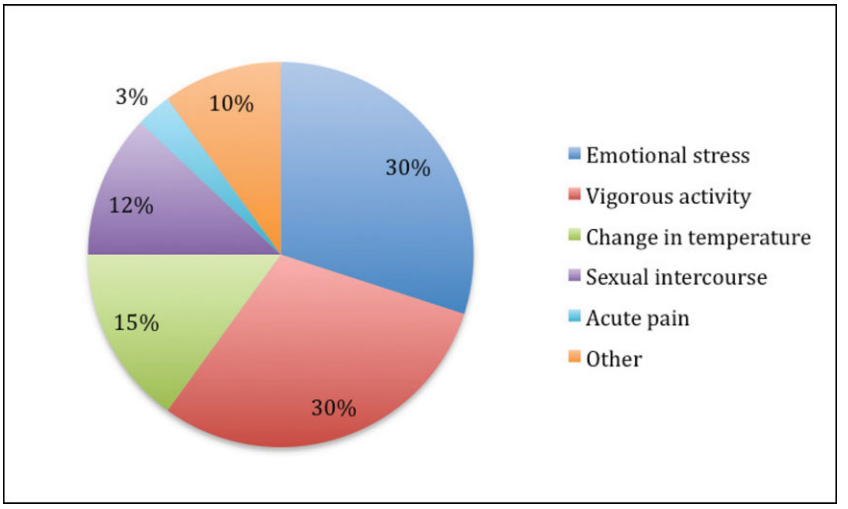


Figure 1. Triggers for transient global amnesia (TGA)

described an increased incidence of personal and family history of psychiatric or phobic disorders.

Diagnosis is clinical and based on the criteria of Caplan and Hodges,[6,16] shown in Table 1.

During the acute episode a temporally graded retrograde amnesia can occur, although this is not part of the current diagnostic criteria. Nausea, headache and dizziness may be present. Other features supporting the diagnosis of TGA are a history of excessive physical activity or strong emotions preceding the onset of symptoms and repetition of the same questions. Patients are co-operative, follow instructions and are able to name objects. Features against the diagnosis are a history of a recent change in medications, evidence of recent hypoglycaemia, memory of the acute episode, retrograde amnesia only, or more than three episodes in one year. Further diagnostic assessment is necessary if there was no witness, the presentation was not typical, or there are vascular risk factors present.

Pathophysiology

The underlying pathophysiology of TGA is unclear. The current hypothesis is that it is associated with lesions within the mediobasal temporal region, hippocampus and the para-hippocampus.[17] Magnetic resonance imaging (MRI) has demonstrated lesions in the hippocampal formation and memory circuits in the mesiotemporal region[18,19] and analysis of the functional anatomy of these lesions has shown selective distribution within the CA1 subfield of the hippocampal cornu ammonis.[20,21] Some images have demonstrated that cellular mechanisms may play a role in developing the lesions, suggesting that a cascade initiated by the selective vulnerability of CA1 neurons to metabolic stress causes a temporary disturbance to the memory pathways. The exact cause of this disturbance in memory is not known, but has been attributed to impaired venous flow, ischaemia, migraine, psychological factors or epilepsy.

Table 1. *Diagnostic criteria for transient global amnesia by Caplan & Hodges*[6,16]

Positive features	Negative features
• Impairment of cognition limited to amnesia	• No loss of consciousness or personal identity
• Witnessed anterograde amnesia	• No focal neurological deficit
• Symptoms resolve within 24 hours	• No epilepsy
	• No recent head trauma or seizures

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Venous flow

Using 3D time-of-flight magnetic resonance angiography (MRA), Chung *et al*.[22] showed that compression of the left brachiocephalic vein led to retrograde intracranial venous flow in half of the TGA patients studied and none of the age- and gender-matched controls. In addition, right-sided internal jugular vein incompetence was found by Agosti *et al*.[23] However, neither is well defined or reproducible.[22,23] Lewis has suggested that venous congestion leading to cerebral venous thrombosis and venous ischaemia could cause TGA.[24] Duplex ultrasonography in 114 TGA cases found a high frequency of insufficient jugular vein valves during Valsalva manoeuvre compared with controls. Together, these results suggest that TGA patients may have an underlying impairment of cerebral venous outflow and vulnerability to venous congestion, leading to TGA.

Ischaemia

The speed of onset and predominant age of patients affected by TGA would support transient ischaemia as a cause.[25] However, this aetiology is unlikely as there is always prompt resolution of the symptoms and, unlike following a transient ischaemic attack (TIA), patients are less likely to suffer a future stroke.[26] There are also no relevant abnormalities found on MRA during an acute episode.[27,28] Interestingly, Winbeck *et al*. did find that carotid atherosclerosis identified by ultrasound was more common among patients with TGA, especially those with diffusion-weighted imaging (DWI) changes, compared with controls. In this subset of patients they concluded that ischaemia may cause the symptoms, although the association was not strong and, compared with TIA patients, other vascular risk factors were not common.[29] The fact that microvascular ischaemic changes on MRI are not increased in TGA patients would also be against an arterial cause.

Migraine

Because migraine is characterized by paroxysmal neuronal dysfunction and a comparable time course, it has been suggested as a cause of TGA. Some investigators have found a strong association,[30] whilst others disagree,[15] but interpretation is difficult because of the many other disorders associated with migraine, e.g. epilepsy, stroke, patent foramen ovale, high antiphospholipid titers, depression, arterial dissection, fibromyalgia and dementia.[31–39]

The pathogenesis of migraine with aura is generally accepted to be caused by cortical spreading depression (CSD) of electrical activity characterized by transient neuronal depolarization and hyperperfusion, and subsequent hyperpolarization, hypoperfusion and neuronal dysfunction. This spreads at a rate of 3–5 mm per minute across the cortex and in animal models and humans has been seen to involve the hippocampus, at times leading to hypoxia in the CA1 field.[7,21,40] Furthermore, induction of CSD in animals produces amnesic behaviour that resolves spontaneously. Given this background and the high prevalence of migraine in some TGA patients who also had headache as an accompanying symptom or precipitating factor, CSD was proposed by Olesen and Jorgensen in 1986 as a possible mechanism underlying TGA.[41] They also noted high hippocampal glutamate concentration, the fact that emotional events could trigger release of glutamate and proposed that TGA tends to follow such events. Initiating a stress response can induce hippocampal dysfunction.[42,43]

DWI abnormalities are not described in migraine, unless associated with stroke, but there have been reports of abnormalities in the apparent diffusion coefficient maps in patients who have had prolonged aura.[11,42,44] This would, perhaps, support CSD as a cause of TGA, but migraine aura is more common in young people, whereas TGA is not. This could be explained by compensatory mechanisms in younger patients preventing CSD in the hippocampal region. However, migraine episodes are generally not associated temporally with TGA so an association with migraine remains possible, but unlikely.

Epilepsy

In 1889, Hughlings-Jackson first suggested that epileptic activity could cause transient amnesia.[45] Quinette *et al*. performed electroencephalograpy (EEG) studies in 106 patients during or after acute TGA.[14] They were unremarkable in 80%, and the remainder showed minor non-epileptiform abnormalities only, which suggests that epilepsy is not a cause of TGA. There is also a low possibility

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of developing epilepsy after TGA. More recently, however, there have been reports of transient epileptic amnesia (TEA) when classic TGA is associated with clear electrographic seizures.[46] These episodes last for 1–3 hours, rather than the usual 24-hour duration of TGA. After typical complex partial seizures with unresponsiveness, automatisms or secondary generalized seizures, the subject remains alert but amnesic. Non-convulsive status epilepticus has been found in a few cases of apparent TGA. EEG studies demonstrate wicket spikes, which are a benign variant, and underlying epileptiform activity is suggested to be the cause.

TEA needs to be considered when there is a history of epilepsy, recurrent attacks of TGA, or atypical duration. Since up to 70% of this group may have a normal inter-ictal EEG, continued surveillance is sometimes necessary. Prolonged or ambulatory EEG studies can identify the nature and frequency of TEA episodes.

Neuroimaging

Single photon emission computed tomography (SPECT), positron emission tomography (PET) and DWI are the three main neuroimaging modalities used to investigate TGA, but are still unable to find a cause and explain the pathophysiology of TGA.[11,17,28,47,48]

Findings from PET and SPECT studies suggest that hypoperfusion and hypometabolism of the hippocampus may be characteristic of TGA,[17] but there have also been numerous cases of dysfunction in other locations such as the thalamic, frontal, cerebellar and striatal regions.[27,28] Haemodynamic changes in SPECT studies confirmed transient dysfunction of the medial temporal lobe during the acute phase of TGA in 42 out of 43 patients.[47,49] PET studies performed after an episode of TGA in 15 patients found abnormalities in the hippocampus of four patients and a high incidence of high and giant cavities.[48,50,51] Resolution of these changes usually occurs, but persistent hypoperfusion was seen as late as one year after an episode of recurrent TGA. Altered perfusion and metabolism are also seen in migraine and epilepsy, so these findings do not help to explain the underlying mechanism of TGA.

DWI is a sensitive technique used to detect early cytotoxic oedema in ischaemia and in the past 20 years it has been reported to show abnormalities suggestive of ischaemic injury in TGA.[44] However, the evidence is conflicting, with some investigators finding no DWI changes during TGA, leading to the conclusion that if changes representing ischaemic injury are seen, a diagnosis of TGA should not be made. Of 31 patients fulfilling Hodges and Warlow's criteria for TGA,[6] abnormalities on DWI during and/or after the episode were found in only 56%.[52] During a 7-year study by Ahn and colleagues,[27] MRI scans were performed on 203 TGA cases. Only 16 of these had changes in the hippocampus, with no obvious clinical difference from those without such changes. However, the scans demonstrating changes were performed at a mean time to scan of 9 hours, whereas the overall mean time to scan was 6 hours. This suggests that precise timing might help to detect more abnormalities, and current recommendations for the imaging of TGA with DWI include use of a 3-T magnet, acquisition between 24 and 72 hours of onset and 3 mm slice thickness.

Bartsch and colleagues[21] studied the evolution of MRI changes in 29 patients. Between 24 and 72 hours post-event, 34 DWI lesions were identified in the CA1 sector of the hippocampus, and these had normalized by 10 days. This is a similar time course to ischaemic lesions, which has led to the suggestion that TGA may have a vascular cause.

MR spectroscopy showed a lactate peak in hippocampal DWI lesions,[53] which suggests local dysfunction of CA1 neurons. Certainly the most consistent finding from neuroimaging studies is the location of abnormalities in the hippocampus, but their origin remains debatable. Whilst DWI is highly sensitive for ischaemia, it is not specific, and other causes for cytotoxic oedema could include hypoglycaemia, cortical spreading depression or epileptic seizures.

Management

The diagnosis of TGA is based on the typical clinical presentation, clear collateral history and absence of any features suggestive of underlying pathology. Careful assessment should avoid the patient being labelled as 'acutely confused' and assumed to have delirium. Screening for haematological abnormalities and hypoglycaemia should be done promptly and a stroke or transient ischaemic attack must be ruled out, so neuroimaging is usually appropriate. If available, high resolution MRI

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with DWI may detect acute hippocampal lesions. EEG may be useful to demonstrate any interictal activity if epilepsy is suspected. Cardiological investigations with electrocardiography, Holter monitoring and echocardiogram may sometimes be required. Adequate investigation without any significant pathology being demonstrated should reassure patients and the excellent prognosis needs to be emphasized. There is no need for any restriction on driving or reporting to the Driving and Vehicle Licensing Agency (DVLA) after diagnosis of a single episode of TGA.

Conclusion

The belief that TGA is related to hippocampal dysfunction is no longer a hypothesis. With advanced imaging and laboratory techniques, clearly there is a metabolic stress localizing to CA1 sector. The precise cause and exact triggers of attacks, however, remains unclear. However, stressors precipitating TGA might be purely coincidental. TGA could be caused by energy exhaustion in a metabolically fragile hippocampus, typical in a demand-supply scenario. Optimal imaging parameters were only recently set and are not uniformly available. This explains why the frequency of DWI changes in TGA patients has not been clearly defined. Uncertainty with regard to treatment may be a result of these studies. It remains unclear whether patients with DWI lesions should be managed as stroke or TIA. While imaging findings of presumed stroke make this less clear, epidemiology on the other hand suggests otherwise. There have been no autopsy studies. Autopsy could help by identifying the cause of the dysfunction, if it was caused by ischaemia or by other causes of energy failure. In recurrent cases or those with very short durations, prolonged or ambulatory EEG should be considered to identify the subgroup of patients with epileptic syndromes. Usually the prognosis of TGA is excellent, and the management depends on recognition and reassurance.

Conflicts of interest

None

References

1 Ribot T. Transient global amnesia. Missing memory. *Mayo Clin Health Lett* 2012; **30**: 6.
2 Benon R. Les ictus amnesiques dans les demences 'organiques'. *Ann Méd Psychol* 1909; **67**: 207–19.
3 Bender MB. Syndrome of isolated episode of confusion with amnesia. *J Hillside Hosp* 1956; **5**: 212–15.
4 Guyotat M, Courjon J. Les ictus amnesiques. *J Med Lyon* 1956; **37**: 697–701.
5 Fisher CM, Adams RD. Transient global amnesia. *Acta Neurol Scand Suppl* 1964; **40** suppl 9: 1–83.
6 Hodges JR, Warlow CP. Syndromes of transient amnesia: towards a classification. A study of 153 cases. *J Neurol Neurosurg Psychiatry* 1990; **53**: 834–43.
7 Melo TP, Ferro JM, Ferro H. Transient global amnesia. A case control study. *Brain* 1992; **115**: 261–70.
8 Vyhnalek M, Bojar M, Jerabek J, Hort J. Long lasting recurrent familiar transient global amnesia after beta blocker treatment withdrawal: case report. *Neuro Endocrinol Lett* 2008; **29**: 44–46.
9 Segers-van Rijn J, de Bruijn SF. Transient global amnesia: a genetic disorder? *Eur Neurol* 2010; **63**: 186–87.
10 Dupuis F, Evrard F, Jacquerye P, Picard G, Goossens C, Ghysens O *et al.* Is transient global amnesia hereditary: 4 families and a review. *Neurology* 2012; **78**: 4–213.
11 Sander K, Sander D. New insights into transient global amnesia: recent imaging and clinical findings. *Lancet Neurol* 2005; **4**: 437–44.
12 Jager T, Bazner H, Kliegel M, Szabo K, Hennerici MG. The transience and nature of cognitive impairments in transient global amnesia: a meta-analysis. *J Clin Exp Neuropsychol* 2009; **31**: 8–19.
13 Velasco R, Al Hussayni S, Bermejo PE. [Sexual intercourse as a trigger of transient global amnesia]. *Rev Neurol* 2008; **47**: 301–3.
14 Quinette P, Guillery-Girard B, Dayan J, de La Sayette V, Marquis S, Viader F *et al.* What does transient global amnesia really mean? Review of the literature and thorough study of 142 cases. *Brain* 2006; **129**: 1640–58.
15 Pantoni L, Lamassa M, Inzitari D. Transient global amnesia: a review emphasizing pathogenic aspects. *Acta Neurol Scand* 2000; **102**: 275–83.
16 Caplan LR. *Handbook of Clinical Neurology*. Amsterdam: Elsevier Science, 1985.
17 Forster A, Griebe M, Gass A, Kern R, Hennerici MG, Szabo K. Diffusion-weighted imaging for the differential diagnosis of disorders affecting the hippocampus. *Cerebrovasc Dis* 2012; **33**: 104–15.

https://doi.org/10.1017/S0959259813000063 Published online by Cambridge University Press

18 Alberici E, Pichiecchio A, Caverzasi E, Farina LM, Persico A, Cavallini A *et al.* Transient global amnesia: hippocampal magnetic resonance imaging abnormalities. *Funct Neurol* 2008; **23**:149–52.

19 Badve MS, Henderson RD, Jhamb A, Read SJ. Hippocampal lesions in transient global amnesia. *J Clin Neurosci* 2011; **18**: 385, 449.

20 Bartsch T, Alfke K, Stingele R, Rohr A, Freitag-Wolf S, Jansen O *et al.* Selective affection of hippocampal CA-1 neurons in patients with transient global amnesia without long-term sequelae. *Brain* 2006; **129**: 2874–84.

21 Bartsch T, Alfke K, Deuschl G, Jansen O. Evolution of hippocampal CA-1 diffusion lesions in transient global amnesia. *Ann Neurol* 2007; **62**: 475–80.

22 Chung CP, Hsu HY, Chao AC, Sheng WY, Soong BW, Hu HH. Transient global amnesia: cerebral venous outflow impairment-insight from the abnormal flow patterns of the internal jugular vein. *Ultrasound Med Biol* 2007; **33**: 1727–35.

23 Agosti C, Borroni B, Akkawi NM, Padovani A. Cerebrovascular risk factors and triggers in transient global amnesia patients with and without jugular valve incompetence: results from a sample of 243 patients. *Eur Neurol* 2010; **63**: 291–94.

24 Lewis SL. Aetiology of transient global amnesia. *Lancet* 1998; **352**: 397–99.

25 Agrawal R, Chandak TR, Zaretskaya M. Transient global amnesia: an uncommon differential diagnosis of transient ischemic attack. *Hospital Physician* 2007; **43**: 50–54.

26 Zorzon M, Antonutti L, Mase G, Biasutti E, Vitrani B, Cazzato G. Transient global amnesia and transient ischemic attack. Natural history, vascular risk factors, and associated conditions. *Stroke* 1995; **26**: 1536–42.

27 Ahn S, Kim W, Lee YS, Kim WY, Lee JH, Oh BJ *et al.* Transient global amnesia: seven years of experience with diffusion-weighted imaging in an emergency department. *Eur Neurol* 2011; **65**: 123–28.

28 Auyeung M, Tsoi TH, Cheung CM, Fong DY, Li R, Chan JK *et al.* Association of diffusion weighted imaging abnormalities and recurrence in transient global amnesia. *J Clin Neurosci* 2011; **18**: 531–34.

29 Winbeck K, Etgen T, von Einsiedel HG, Rottinger M, Sander D. DWI in transient global amnesia and TIA: proposal for an ischaemic origin of TGA. *J Neurol Neurosurg Psychiatry* 2005; **76**: 438–41.

30 Schmidtke K, Ehmsen L. Transient global amnesia and migraine. A case control study. *Eur Neurol* 1998; **40**: 9–14.

31 Davies PT, Panayiotopoulos CP. Migraine triggered seizures and epilepsy triggered headache and migraine attacks: a need for re-assessment. *J Headache Pain* 2011; **12**: 287–88.

32 Caminero AB, Sanchez Del Rio GM. [Migraine as a cerebrovascular risk factor]. *Neurologia* 2012; **27**: 103–11.

33 Schurks M, Kurth T. Is migraine a predictor for identifying patients at risk of stroke? *Expert Rev Neurother* 2011; **11**: 615–18.

34 Trabattoni D, Fabbiocchi F, Montorsi P, Galli S, Teruzzi G, Grancini L *et al.* Sustained long-term benefit of patent foramen ovale closure on migraine. *Catheter Cardiovasc Interv* 2011; **77**: 570–74.

35 Cavestro C, Micca G, Molinari F, Bazzan M, Di Pietrantonj C, Aloi R *et al.* Migraineurs show a high prevalence of antiphospholipid antibodies. *J Thromb Haemost* 2011; **9**: 1350–54.

36 Moschiano F, D'Amico D, Canavero I, Pan I, Micieli G, Bussone G. Migraine and depression: common pathogenetic and therapeutic ground? *Neurol Sci* 2011; **32** suppl 1: S85–88.

37 Demaerschalk BM. Migraine is associated with an increased risk of cervicocephalic arterial dissection. *Cephalalgia* 2011; **31**: 884–85.

38 Evans RW, de Tommaso M. Migraine and fibromyalgia. *Headache* 2011; **51**: 295–99.

39 Sas K, Pardutz A, Toldi J, Vecsei L. Dementia, stroke and migraine – some common pathological mechanisms. *J Neurol Sci* 2010; **299**: 55–65.

40 Bartsch T, Dohring J, Rohr A, Jansen O, Deuschl G. CA1 neurons in the human hippocampus are critical for autobiographical memory, mental time travel, and autonoetic consciousness. *Proc Natl Acad Sci USA* 2011; **108**: 17562–67.

41 Olesen J, Jorgensen MB. Leao's spreading depression in the hippocampus explains transient global amnesia. A hypothesis. *Acta Neurol Scand* 1986; **73**: 219–20.

42 Cho YN, Kim WJ. The clinical factors related with hippocampal lesion of diffusion MRI in transient global amnesia. *Neurology* 2012; **78** (P03.150).

43 Hunter G. Transient global amnesia. *Neurol Clin* 2011; **29**:1045–54.

44 Felix MM, Castro LH, Maia ACJ, da Rocha AJ. Evidence of acute ischemic tissue change in transient global amnesia in magnetic resonance imaging: case report and literature review. *J Neuroimaging* 2005; **15**: 203–5.

45 Hughlings-Jackson J. On a particular variety of epilepsy (intellectualaura), one case with symptoms of organic brain disease. *Brain* 1889; **11**: 179–207.

46 Pearce JM, Bogousslavsky J. 'Les ictus amnesiques' and transient global amnesia. *Eur Neurol* 2009; **62**: 188–92.

47 Takeuchi R, Matsuda H, Yoshioka K, Yonekura Y. Cerebral blood flow SPET in transient global

https://doi.org/10.1017/S0959259813000063 Published online by Cambridge University Press

amnesia with automated ROI analysis by 3DSRT. *Eur J Nucl Med Mol Imaging* 2004; **31**: 578–89.
48 Guillery B, Desgranges B, de La Sayette V, Landeau B, Eustache F, Baron JC. Transient global amnesia: concomitant episodic memory and positron emission tomography assessment in two additional patients. *Neurosci Lett* 2002; **325**: 62–66.
49 Kirshner HS. Transient global amnesia: a brief review and update. *Curr Neurol Neurosci Rep* 2011; **11**: 578–82.
50 Eustache F, Desgranges B, Laville P, Guillery B, Lalevee C, Schaeffer S *et al*. Episodic memory in transient global amnesia: encoding, storage, or retrieval deficit? *J Neurol Neurosurg Psychiatry* 1999; **66**: 148–54.
51 Eustache F, Desgranges B, Petit-Taboue MC, de La Sayette V, Piot V, Sable C *et al*. Transient global amnesia: implicit/explicit memory dissociation and PET assessment of brain perfusion and oxygen metabolism in the acute stage. *J Neurol Neurosurg Psychiatry* 1997; **63**: 357–67.
52 Choi BS, Kim JH, Jung C, Kim SY. High-resolution diffusion-weighted imaging increases lesion detectability in patients with transient global amnesia. *Am J Neuroradiol* 2012; **33**: 1771–74.
53 Bartsch T, Alfke K, Wolff S, Rohr A, Jansen O, Deuschl G. Focal MR spectroscopy of hippocampal CA-1 lesions in transient global amnesia. *Neurology* 2008; **70**: 1030–35.

https://doi.org/10.1017/S0959259813000063 Published online by Cambridge University Press