Chapter 8
Epidemiology of TGA (2): Possible Precipitating Factors

Abstract This chapter examines factors identified in clinical and epidemiological studies as possible precipitating factors for episodes of TGA. Of these, emotional stress and physical effort are the most commonly identified. It is possible that these precipitating factors may give some insights into the pathogenesis of TGA. Predisposing factors for TGA are considered in the previous chapter.

Keywords TGA · Precipitating factors

A number of factors have been described in close temporal association with (i.e. immediately before) the onset of an attack of TGA and hence may be designated as precipitating factors, as opposed to those with a more distant temporal association which may be designated predisposing factors (Chap. 7). Miller Fisher identified such precipitating factors in 26/85 TGA episodes (30.6%) [1], whilst Rösler et al. identified precipitants in 58% of their 72 TGA patients based on administration of a standardised questionnaire [2]. In their review of cases reported in the literature, Quinette et al. noted precipitating factors in 462 of 881 cases (= 52.4%) and in their own series in 131 of 147 episodes (= 89.1%) [3]. Cejas et al. identified triggering factors in 41/79 (51.9%) of their TGA patients who agreed to complete a questionnaire [4], and in a retrospective survey of 389 patients, Hoyer et al. identified a precipitating factor in 266 (68.4%) [5]. Morris et al. found a similar percentage of cases with identifiable triggers in single-episode (28.8%) and recurrent (35.0%) TGA [6].

8.1 Emotional Stress

In his report on a series of 85 episodes of TGA, Miller Fisher found that highly emotional experiences were found to be the most common recognised precipitating event (8/26) [1]. Hodges and Warlow noted an emotionally stressful event in the 24 h before an episode of TGA in 14% of their prospective sample, examples including receiving bad news, witnessing an accident or being involved in an argument

A.J. Larner, *Transient Global Amnesia*,
https://doi.org/10.1007/978-3-030-98939-2_8

[7]. Emotional arousal was also noted as a precipitating factor for TGA by Merriam and colleagues [8, 9] who emphasised the differentiation of TGA from the forms of transient psychological amnesia (Sect. 3.3) which may also follow stressful experiences. Quinette et al. noted that emotionally charged events may be a precipitant of TGA, reported in 28% of cases in their literature survey and in 29% of their personally observed cases [3]. Emotional stress may be a predisposing factor for as well as precipitating event to TGA (Sect. 7.10).

The specific emotional stress may take many various forms, including the experience of a burglary [10], a disturbing dream [11] or an emotionally charged psychotherapy session [12]. A similar explanation might account for TGA following general anaesthesia in an anxious patient undergoing otolaryngological surgery [13].

Quinette et al. reported that TGA in women was mainly associated with an emotional precipitating event (cf. men, physical precipitating event) [3]. In a retrospective study of a cohort of 389 TGA patients, Hoyer et al. examined gender-related differences in stressful precipitating events and confirmed that emotional triggers were more often experienced by women (37.2% vs. 22.8%) [5]. Noh and Kang reported "extreme stress" occurring in 64.6% of their cohort of TGA patients, most of whom (114/128 = 89%) were women [14].

8.2 Physical Effort

Many forms of physical exercise have been described as precipitating factors for TGA, including cycling (e.g. [15, 16]; see also Case Studies 2.1 and 7.1), swimming (e.g. [17], and [18], case 2), skiing ([19]; also Case Study 7.1), gardening/digging (e.g. [4, 20]), sawing wood, after extreme exercise (e.g. [21]) and after medical procedures involving an exercise testing procedure such as the bicycle ergometer or treadmill [22, 23]. Other recorded effortful activities include defecation in constipated patients, coughing, vomiting and repeated yawning [4].

Hodges reported exercise as a precipitant in 18% of patients in the Oxford TGA study ([24], p.19). Quinette et al. noted physical effort as a precipitant of TGA in 31% of cases in their literature survey and in 25% of their personally observed cases [3]. Hoyer et al. found physical stressors were more common in men than women (41.1% vs. 30.7%) [5]. Hence, physical triggers of TGA are more often experienced by men and emotional triggers more often by women.

8.3 Water Contact or Temperature Change

Fisher and Adams "first case" (see Sect. 1.1) was associated with a swim in cold water [25]. In a report on a series of 85 episodes of TGA, Fisher found that bathing in cold water (specifically the Atlantic Ocean) was found to be a common recognised precipitating event (3/26) [1]. Other examples have also been reported (e.g. Case Studies 2.1 and 6.1). Quinette et al. reported water contact or temperature change (which could include hot baths) in 14% of patients in their literature review and in 11% of their own

series [3]. Martin coined the term "amnesia by the seaside" for TGA cases associated with cold water immersion [26]. Tubridy et al. also used the term [27], even when there was no cold water immersion, merely a walk by the seaside ([28], p.11–6).

An episode of transient amnesia in a volunteer undergoing experimental repeated cold water (20 °C) immersion has been reported as TGA [29], but the details are not convincing for this diagnosis, specifically the development of "altered affect … whimpering, anxious delirium-like state" for 20 min. It was also atypical in the subject's age (23).

Temperature change might also possibly contribute to or explain TGA cases associated with skiing ([19]; Case Study 7.1), high altitude [30–32], transoceanic flight [33], paragliding [34], infusion of cryopreserved cells [35] and whole-body cryotherapy (brief exposure to very cold and dry air) [36]. A study from relatively high altitude (Davos, Switzerland) found TGA cases peaked in the winter, suggesting that low temperature might be either a predisposing factor or a trigger for TGA, but no relation was found with atmospheric pressure, wind or humidity [37].

8.4 Sexual Activity

Amnesia is one of the recognised acute neurological consequences of sexual activity, as is headache, but whether these are separate or pathophysiologically related conditions is not currently known [38].

Sexual activity was noted as a precipitating factor for TGA in 2/17 patients reported by Fisher and Adams [25], and in a later report on a series of 85 episodes of TGA, Fisher found it to be the second most common recognised precipitating event for TGA (7/26) [1]. Hodges and Warlow reported sexual intercourse to be the precipitant of TGA in 3% of their cases [7]. Quinette et al. reported sexual intercourse as a precipitant in 12% of patients in their literature review and in 9% of their own series [3].

In addition, many individual possible reports of "coital" or "post-coital" amnesia have also been presented (e.g. [39–51].; see also Case Study 4.1 and Case Study 8.1). These include examples of recurrent episodes of amnesia after intercourse [52–54].

Case Study 8.1: Sexual Activity as a Precipitating Factor of TGA
A previously healthy 61-year-old man had an episode of memory loss. Somewhat abashed, he reported that about 7 weeks earlier he and his wife had been making love at 5 o'clock in the morning, "not something we usually do". He then got up and was found in the kitchen some minutes later by his wife. She reported that he was repeatedly asking "where am I?". Questioned by her, he had forgotten the names of his medications for high blood pressure and the fact that his son had recently passed his driving test. However, these functions returned after a period of about 4 h and did not recur. His general practitioner made a provisional diagnosis of transient ischaemic attack (TIA). The patient was commenced on aspirin, non-urgent structural brain imaging was arranged and referral made to the neurology clinic (adapted from [45]).

Monzani et al. reported on 10 patients (all male; age range 41–64 years) with transient amnesia related to sexual activity, which comprised 18% of all acute global amnesia patients observed during the study period; of these ten, one had a subarachnoid haemorrhage whilst all the others were diagnosed as TGA [55].

Episodes of TGA reported in association with the use of phosphodiesterase type 5 (PDE-5) inhibitors, sildenafil (Viagra) and tadalafil, and with the intracavernosal injection of alprostadil (Caverject) (Sect. 3.4.2 and Table 3.8), may also be precipitated by sexual activity rather than being an acute adverse drug effect.

Concurrence of sex-related TGA and primary headache associated with sexual activity (PHSA), whose features differ from migraine, has been reported on occasion [56–58]. This may suggest the possibility either of shared pathophysiological mechanisms, perhaps related to activation of pathways within the trigeminocervical complex [58] or a concurrence of two disorders with a shared trigger [57].

8.5 Pain

In a report on a series of 85 episodes of TGA, pain was found to be one of the most common recognised precipitating events (6/26) [1]. Examples may include abdominal pain [25], dental extraction [59], trigeminal ganglion stimulation ([1], in 2/26), pain from a pilonidal sinus ([24], p.18) and myocardial infarction, although TGA after painless MI has also been reported (Sect. 3.1.6). TGA associated with other painful medical procedures is also well described (Sect. 8.8). Quinette et al. noted acute pain as a precipitant of TGA in 2% of cases in their literature survey and in 3% of their personally observed cases [3].

Migraine headache is a not infrequent accompaniment of TGA episodes, possibly as a precipitant (Sect. 8.6), as well as being a predisposing factor (Sect. 7.9) and a symptomatic cause of amnesia which enters the differential diagnosis of TGA (Sect. 3.4.1). Whether the pain associated with migraine headache per se may be considered a precipitant of TGA, rather than the migraine pathophysiology, does not seem to be commented upon in the literature.

The absence of case reports of TGA related to events acknowledged to be associated with severe pain such as parturition or cluster headache might be taken to argue against pain per se as a precipitant. Nephrolithiasis or ureteral colic is a rarely reported association with TGA [1, 60]. Fisher and Adams ([25], p.40) reported "One woman was in the throes of rather severe ureteral colic (this patient thought two of her previous attacks had also been precipitated by pain)". Another possible association of TGA and kidney stone was confounded by use of multiple doses of opioid and non-opioid analgesia [61].

8.6 Migraine

Migraine has already been discussed in the context of the differential diagnosis of TGA (Sect. 3.4.1) and as a possible predisposing factor for TGA (Sect. 7.9). It may also be a precipitating factor.

TGA occurring during a migraine attack has been reported by many authors (e.g. [62–68]. However, this is probably a very rare occurrence. A large retrospective analysis from a centre in France identified six cases of TGA occurring (hence, according to the paper's title, "triggered") by a migraine attack amongst a cohort of 8821 new patients seen over an 11-year period [69]. Other mechanisms might also be envisaged, for example forceful vomiting in the context of a migraine attack might be associated with the Valsalva manoeuvre (Sect. 8.9).

Many of the familial examples of TGA (Sect. 7.8) had TGA episodes which reportedly occurred at the same time as a migraine (see Table 7.3).

8.7 Brain Infections

Brain infections are included amongst the symptomatic causes of amnesia (Sect. 3.5.2; Table 3.2) and may enter the differential diagnosis of TGA. Cases labelled diagnostically as "TGA" have on occasion been reported in association with infective disorders of the brain, including herpes simplex encephalitis [70, 71], neurosyphilis [72] and Epstein–Barr virus encephalitis [73], although Daniel states that these latter authors "clearly described a case of transient epileptic amnesia" ([74], p.205).

8.8 Medical Procedures and Therapies

Onset of TGA concurrent with the performance of various medical procedures has been described. Of these, angiography (cerebral, coronary) appears to be the most frequently described (see Sect. 3.1.5; Table 3.6).

Angiography was recognised as a possible precipitating event for transient amnesia even before the TGA nomenclature was coined ([75]; see Sect. 1.2). A retrospective analysis of over 20,000 angiographic procedures undertaken at one hospital over a period of 7.5 years identified 9 cases of TGA (= 0.04%), which followed either cerebral angiography (5 in 4360 = 0.11%) or cardiac angiography (4 in 8817 = 0.05%) but no cases following peripheral angiography were identified (0 in 7659), indicating the infrequency of the association of TGA with angiography [76]. Even cardiologists with extensive (>25 years) experience of cardiac angiography, encompassing many thousands of procedures, may not encounter a case of TGA (Dr WL Morrison, personal communication, Liverpool Heart and Chest Hospital, 24/12/16).

Various other medical procedures have sometimes been associated with the onset of TGA (Table 8.1). For example, upper gastrointestinal endoscopic procedures have on occasion been followed by TGA (e.g. [85, 87, 89, 90]), as has transoesophageal echocardiography [82, 83]. Possible explanations might include the emotional stress of instrumentation, associated pain, autonomic activation from passing the

Table 8.1 Reports of medical procedures associated with onset of TGA (see text for caveats)

Procedure	Reference(s)
Acupuncture	Hodges (1991) (n = 1) ([24], p.18)
Anaesthesia	Ghoneim (1998) [77] Bortolon et al. (2005) [78] Galipienzo et al. (2012) [13]
Aneurysm coiling	Graff-Radford et al. (2013) [79]
Angiography(cerebral, coronary)	See Table 3.6
Carotid artery stenting	Lee (2020) [80]
Cryotherapy	Carrard et al. (2017) [36]
Cystoscopy	Miller et al. (1987) [22]
Deep brain stimulation (misplaced electrode)	Baezner et al. (2013) [81]
Dental extraction	Godlewski (1968) [59]
Echocardiogram (transoesophageal)	Profice et al. (2008) [82] Cassar and Balkhausen (2020) [83]
Electroencephalography (EEG)	Cole et al. (1987) [84] Ung and Larner (2014) [50]
Endoscopy(upper gastrointestinal)	Hiraga and Matsunaga (2006) (n = 3) [85] Neuzillet et al. (2009) [86] Sayilir et al. (2009) [87] Ahn et al. (2011) (n = 4) [88] Cesar and Perdigao (2012) [89] Jeong et al. (2018) (n = 2) [90]
Exercise testing(cycle ergometer, treadmill)	Miller et al. (1987) [22] Richardson et al. (1998) [23]
Intracarotid amobarbital procedure	Benke et al. (2005) [91]
Nasogastric tube insertion	Miller et al. (1987) [22]
Nasopharyngeal swab	Ravaglia et al. (2021) [92]
"Oral provocation test" (rofecoxib)	Hirschfeld et al. (2007) [93]
Photodynamic therapy	Reinholz et al. (2015) [94]
Psychotherapy	Espiridion et al. (2019) [12]
Pulmonary function testing	Miller et al. (1987) [22] Robbins et al. (2010) [95] Williamson and Larner (2016) [96]
Radio frequency catheter ablation for premature cardiac ventricular beats	Mokabberi et al. (2010) [97]
Stellate ganglion block	Park et al. (2015) [98]
Stem cell infusion (autologous peripheral blood)	Otrock et al. (2008) [35]
Trigeminal ganglion stimulation	Fisher (1982) (n = 2) [1]
Urinary catheterisation	Ahn et al. (2011) (n = 1) [88]
Urography (excretory)	Miller et al. (1987) [22]
Venesection	Hodges (1991) (n = 2) ([24], p.18)

scope and medication use (scopolamine in [85], although TGA is also recorded following endoscopy without medication [87]). No account of TGA after colonoscopy has been identified.

A review by Jeong et al. published in 2018 found 89 patients with medical procedure-related TGA described in 49 articles. The most common procedure was angiography (cerebral > coronary) followed by general anaesthesia, although with only nine cases the latter could simply be chance. Neurological procedures were more common than cardiac, anaesthetic, gastrointestinal and pulmonary procedures. The authors concluded that Valsalva-associated activities, emotional stress with anxiety and acute pain were predisposing (sic) conditions in these cases [90]. Hoyer et al. recorded TGA following a medical procedure in 2.1% of their cohort of 389 patients [5].

All these accounts of TGA associated with medical procedures are rare, considering the frequency with which the various procedures are undertaken, so could be chance concurrence rather than causal association. Moreover, medical procedures are often accompanied by patient emotional stress (e.g. psychotherapy [12]), particularly anxiety (Sect. 8.1) and pain (Sect. 8.5), which might also be contributory factors in the cases observed.

8.9 Valsalva Manoeuvre

Bedside spirometry (forced vital capacity), one of the medical procedures reported on occasion to precipitate an episode of TGA [22, 95, 96] (Sect. 8.8), requires breath holding before forced expiration, effectively the performance of a Valsalva manoeuvre. This manoeuvre has sometimes been implicated in a number of other situations associated with TGA onset, including physical exercise, sexual activity, cold water immersion and response to pain. It has also been considered relevant to the retrograde internal jugular vein blood flow due to jugular vein valve incompetence (Sect. 4.3.3.2) which is pertinent to one of the hypotheses of TGA pathogenesis (Sect. 9.2.2).

However, attempts to reproduce the typical clinical and neuroradiological findings of TGA by voluntary Valsalva manoeuvre have failed. Patients with a previous episode of TGA were subjected to a controlled Valsalva manoeuvre, at least 3 months post-event, and suffered no recurrence of either typical symptoms or MR-DWI findings of TGA [99]. Jeong et al. noted that more than half of their patients (n = 8) with incidental MR-DWI hippocampal hyperintensities had performed Valsalva manoeuvre-associated activities but this was also true of their TGA group (n = 16) [100].

Valsalva manoeuvre may thus be an associated, rather a precipitating, factor in some episodes of TGA.

8.10 Other Possible Precipitating Factors

TGA cases associated with medication use have been reported on occasion (Table 3.8). These drugs might possibly be considered as precipitating factors, but the paucity of reports and the variety of drugs suggest no one specific class of drugs as being particularly culpable. Emerging pharmacovigilance data suggest COVID-19 vaccines may be an exception.

There have been occasional reports of TGA occurring at high altitude [30–32] suggesting a possible role for cerebral hypoxia, although physical effort and temperature change may be contributory (and/or confounding) factors in these cases.

8.11 Summary and Recommendations

Many possible precipitating factors for TGA have been examined. Some consistent observations have been made, but no factor seems to be necessary and/or sufficient to induce TGA. This has prompted various aetiological theories for TGA which are reviewed and elaborated upon in the next chapter.

References

1. Fisher CM. Transient global amnesia. Precipitating activities and other observations. Arch Neurol. 1982;39:605–8.
2. Rösler A, Mras GJ, Frese A, Albert I, Schnorpfeil F. Precipitating factors of transient global amnesia. J Neurol. 1999;246:53–4.
3. Quinette P, Guillery-Girard B, Dayan J, de la Sayette V, Marquis S, Viader F, Desgranges B, Eustache F. What does transient global amnesia really mean? Review of the literature and thorough study of 142 cases. Brain. 2006;129:1640–58.
4. Cejas C, Cisneros LF, Lagos R, Zuk C, Ameriso SF. Internal jugular vein valve incompetence is highly prevalent in transient global amnesia. Stroke. 2010;41:67–71.
5. Hoyer C, Ebert A, Sandicki V, Platten M, Szabo K. Sex-related differences in stressful events precipitating transient global amnesia—a retrospective observational study. J Neurol Sci. 2021;425:117464.
6. Morris KA, Rabinstein AA, Young NP. Factors associated with risk of recurrent transient global amnesia. JAMA Neurol. 2020;77:1551–8.
7. Hodges JR, Warlow CP. Syndromes of transient amnesia: towards a classification. A study of 153 cases. J Neurol Neurosurg Psychiatry. 1990;53:834–43.
8. Merriam AE. Emotional arousal-induced transient global amnesia. Case report, differentiation from hysterical amnesia, and an etiologic hypothesis. Neuropsychiatry Neuropsychol. Behav Neurol. 1988;1:73–8.
9. Merriam AE, Wyszynski B, Betzler T. Emotional arousal-induced transient global amnesia. A clue to the neural transcription of emotion? Psychosomatics. 1992;33:109–13.
10. Pillmann F, Broich K. Transitory global amnesia – psychogenic origin of organic disease? Psychopathologic basis and pathogenetic considerations [in German]. Fortschr Neurol Psychiatr. 1998;66:160–3.

11. Marinella MA. Transient global amnesia and a father's worst nightmare. N Engl J Med. 2004;350:843–4.
12. Espiridion ED, Gupta J, Bshara A, Danssaert Z. Transient global amnesia in a 60-year-old female with post-traumatic stress disorder. Cureus. 2019;11(9):e5792.
13. Galipienzo J, Lablanca MS, Zannin I, Rosado R, Zarza B, Olarra J. Transient global amnesia after general anaesthesia. Rev Esp Anestesiol Reanim. 2012;59:335–8.
14. Noh SM, Kang HG. Clinical manifestation and imaging characteristics of transient global amnesia: patent foramen ovale as an underlying factor. J Integr Neurosci. 2021;20:719–25.
15. Milburn-McNulty P, Larner AJ. Transient global amnesia and brain tumour: chance concurrence or aetiological association? Case report and systematic literature review. Case Rep Neurol. 2015;7:18–25.
16. Shuping JR, Rollinson RD, Toole JF. Transient global amnesia. Ann Neurol. 1980;7:281–5.
17. Jeong HS, Moon JS, Baek IC, Lee AY, Kim JM. Transient global amnesia with post-hyperventilation temporal sharp waves—a case report. Seizure. 2010;19:609–11.
18. Sakashita Y, Sugimoto T, Taki S, Matsuda H. Abnormal cerebral blood flow following transient global amnesia. J Neurol Neurosurg Psychiatry. 1993;56:1327.
19. Ay H, Furie KL, Yamada K, Koroshetz WJ. Diffusion-weighted MRI characterizes the ischemic lesion in transient global amnesia. Neurology. 1998;51:901–3.
20. Corston RN, Godwin-Austen RB. Transient global amnesia in four brothers. J Neurol Neurosurg Psychiatry. 1982;45:375–7.
21. Magazi D. Amnesia after a half marathon—a case study. Clin J Sport Med. 2012;22:448–9.
22. Miller JW, Petersen RC, Metter EJ, Millikan CH, Yanagihara T. Transient global amnesia: clinical characteristics and prognosis. Neurology. 1987;37:733–7.
23. Richardson RS, Leek BT, Wagner PD, Kritchevsky M. Transient global amnesia: a complication of incremental exercise testing. Med Sci Sports Exerc. 1998;30(10Suppl):S403-5.
24. Hodges JR. Transient amnesia. Clinical and neuropsychological aspects. London: WB Saunders; 1991.
25. Fisher CM, Adams RD. Transient global amnesia. Acta Neurol Scand. 1964;40(Suppl9):1–81.
26. Martin EA. Transient global amnesia. A report of eleven cases, including five of amnesia at the seaside. Ir J Med Sci. 1970;3:331–5.
27. Tubridy N, Hutchinson M, Murphy RP. Transient global amnesia: "amnesia by the seaside" revisited. J Neurol. 1999;246:500–1.
28. Tubridy N. Just one more question. Stories from a life in neurology. London: Penguin Ireland; 2019.
29. Castellani JW, Young AJ, Sawka MN, Backus VL, Canete JJ. Amnesia during cold water immersion: a case report. Wilderness Environ Med. 1998;9:153–5.
30. Bucuk M, Tomic Z, Tuskan-Mohar L, Bonifacic D, Bralic M, Jurjevic A. Recurrent transient global amnesia at high altitude. High Alt Med Biol. 2008;9:239–40.
31. Litch JA, Bishop RA. Transient global amnesia at high altitude. N Engl J Med. 1999;340:1444.
32. Litch JA, Bishop RA. High-altitude global amnesia. Wilderness Environ Med. 2000;11:25–8.
33. Rashid J, Starer PJ. Transient global amnesia following a transoceanic flight. Psychiatry Clin Neurosci. 2006;60:516–20.
34. Milheiro I, Rocha S, Machado A. Falling (or ascending) into oblivion: transient global amnesia with paragliding. J Neuropsychiatry Clin Neurosci. 2011;23:E40.
35. Otrock ZK, Beydoun A, Barada WM, Masroujeh R, Hourani R, Bazarbachi A. Transient global amnesia associated with the infusion of DMSO-cryopreserved autologous peripheral blood stem cells. Haematologica. 2008;93:e36–7.
36. Carrard J, Lambert AC, Genné D. Transient global amnesia following a whole-body crytotherapy session. BMJ Case Rep. 2018;2017:bcr2017221431.
37. Erba L, Czaplinski A. Transient global amnesia: an altitude sickness? Eur J Neurol. 2017;24(Suppl1):146 (EP1050)
38. Larner AJ. Transient acute neurologic sequelae of sexual activity: headache and amnesia. J Sex Med. 2008;5:284–8.

39. Agosti C, Borroni B, Akkawi N, Padovani A. Three sisters covering the transient global amnesia spectrum. Int Psychogeriatr. 2007;19:987–9.
40. Alonso-Navarro H, Jimenez-Jimenez FJ. Transient global amnesia during sexual intercourse [in Spanish]. Rev Neurol. 2006;42:382–3.
41. Bucuk M, Muzur A, Willheim K, Jurjevic A, Tomic Z, Tuskan ML. Make love to forget: two cases of transient global amnesia triggered by sexual intercourse. Coll Anthropol. 2004;28:899–905.
42. Dandapat S, Bhargava P, Ala TA. Familial transient global amnesia. Mayo Clin Proc. 2015;90:696–7.
43. Dang CV, Gardner LB. Transient global amnesia after sex. Lancet. 1998;352:1557–8.
44. Gallagher J, Murphy MS, Carroll J. Transient global amnesia after sexual intercourse. Ir J Med Sci. 2005;174:86–7.
45. Larner AJ. Amnesia as a sex-related adverse event. Br J Hosp Med. 2011;72:292–3.
46. Maloy K, Davis JE. "Forgettable" sex: a case of transient global amnesia presenting to the emergency department. J Emerg Med. 2011;41:257–60.
47. Marques-Vilallonga A, Aranda-Rodriguez S, Trallero-Araguas E, Jimenez-Moreno FX. Transient global amnesia associated to sildenafil and sexual activity [in Spanish]. Rev Neurol. 2014;59:93.
48. Mayeux R. Sexual intercourse and transient global amnesia. N Engl J Med. 1979;300:864.
49. Okura M, Nakayama H, Nagamine I, Ikuta T. Sexual intercourse as a precipitating factor of transient global amnesia. Jpn J Psychiatry Neurol. 1993;47:13–6.
50. Ung KYC, Larner AJ. Transient amnesia: epileptic or global? A differential diagnosis with significant implications for management. Q J Med. 2014;107:915–7.
51. Velasco R, Al-Hussayni S, Bermejo PE. Sexual intercourse as a trigger of transient global amnesia [in Spanish]. Rev Neurol. 2008;47:301–3.
52. Bermejo PE, García-Cobos E. Recurrent post-coital transient global amnesia. Rev Neurol. 2010;51:316–7.
53. Gonzalez-Martinez V, Comte F, de Verbizier D, Carlander B. Transient global amnesia: concordant hippocampal abnormalities on positron emission tomography and magnetic resonance imaging. Arch Neurol. 2010;67:510–1.
54. Lane RJ. Recurrent coital amnesia. J Neurol Neurosurg Psychiatry. 1997;63:260.
55. Monzani V, Rovellini A, Schinco G, Silani V. Transient global amnesia or subarachnoid haemorrhage? Clinical and laboratory findings in a particular type of acute global amnesia. Eur J Emerg Med. 2000;7:291–3.
56. Antunes F, Rosário Marques I, Grunho MD. Sex, headache and amnesia: filling in the blanks. Eur J Neurol. 2015;22(Suppl1):833. (abstract D168)
57. Knapen SE, Onderwater GL, Roon KI. Double whammy: a concurrence of two disorders with a shared trigger. Acta Neurol Belg. 2020;120:993–4.
58. Ziso B, Larner AJ. Double whammy: sex-related headache and amnesia. Acta Neurol Belg. 2020;120:699.
59. Godlewski S. Amnesic episodes (transient global amnesia). (clinical study based on 33 unpublished cases) [in French]. Sem Hop. 1968;44:553–77.
60. Kettaneh A, Gobron C, Fain O, Mohib S, Thomas M. Calculi and memory. Eur J Neurol. 2001;8:195–6.
61. Durrani M, Milas J, Parson G, Pescatore R. Temporary memory steal: transient global amnesia secondary to nephrolithiasis. Clin Pract Cases Emerg Med. 2018;2:334–7.
62. Caffarra P, Scaglioni A, Malvezzi L, Manzoni GM. Transient global amnesia and migraine. Ital J Neurol Sci. 1988;9:287–9.
63. Caplan L, Chedru F, Lhermitte F, Mayman C. Transient global amnesia and migraine. Neurology. 1981;31:1167–70.
64. Crowell GF, Stump DA, Biller J, McHenry LC Jr, Toole JF. The transient global amnesia-migraine connection. Arch Neurol. 1984;41:75–9.

65. Fernandez A, Rincon F, Mazer SP, Elkind MS. Magnetic resonance imaging changes in a patient with migraine attack and transient global amnesia after cardiac catheterization. CNS Spectr. 2005;10:980–3.
66. Olivarius BD, Jensen TS. Transient global amnesia in migraine. Headache. 1979;19:335–8.
67. Pradalier A, Lutz G, Vincent D. Transient global amnesia, migraine, thalamic infarct, dihydroergotamine, and sumatriptan. Headache. 2000;40:324–7.
68. Santoro G, Casadei B, Venco A. The transient global amnesia-migraine connection. Case Rep Funct Neurol. 1988;3:353–60.
69. Donnet A. Transient global amnesia triggered by migraine in a French tertiary-care center: an 11-year retrospective analysis. Headache. 2015;55:853–9.
70. Kimura S, Kumano T, Miyao S, Teramoto J. Herpes simplex encephalitis with transient global amnesia as an early sign. Intern Med. 1995;34:131–3.
71. McCorry DJ, Crowley P. Transient global amnesia secondary to herpes simplex viral encephalitis. Q J Med. 2005;98:154–5.
72. Fujimoto H, Imaizumi T, Nishimura Y, et al. Neurosyphilis showing transient global amnesia-like attacks and magnetic resonance imaging abnormalities mainly in the limbic system. Intern Med. 2001;40:439–42.
73. Pommer B, Pilz P, Harrer G. Transient global amnesia as a manifestation of Epstein-Barr virus encephalitis. J Neurol. 1983;229:125–7.
74. Daniel BT. Transient global amnesia. Print version and ebook: Amazon; 2012.
75. Hauge T. Catheter vertebral angiography. Acta Radiologica Suppl. 1954;109:1–219.
76. Duan H, Li L, Zhang Y, Zhang J, Chen M, Bao S. Transient global amnesia following neural and cardiac angiography may be related to ischemia. Biomed Res Int. 2016;2016:2821765.
77. Ghoneim MM. Transient global amnesia: a cause for postanesthetic memory disorder. Anesth Analg. 1998;87:980–1.
78. Bortolon RJ, Weglinski MR, Sprung J. Transient global amnesia after general anesthesia. Anesth Analg. 2005;101:916–9.
79. Graff-Radford J, Clapp AJ, Lanzino G, Rabinstein AA. Transient amnesia after coiling of a posterior circulation aneurysm. Neurocrit Care. 2013;18:245–7.
80. Lee BH. Transient global amnesia following carotid artery stenting: a case report. Radiol Case Rep. 2020;15:1159–63.
81. Baezner H, Blahak C, Capelle HH, Schrader C, Lutjens G, Krauss JK. Transient global amnesia associated with accidental high-frequency stimulation of the right hippocampus in deep brain stimulation for segmental dystonia. Stereotact Funct Neurosurg. 2013;91:335–7.
82. Profice P, Rizzello V, Pennestri F, et al. Transient global amnesia during transoesophageal echocardiogram. Neurol Sci. 2008;29:477–9.
83. Cassar MP, Balkhausen K. Transient global amnesia following transoesophageal echocardiography. BMJ Case Rep. 2020;13:e234751.
84. Cole AJ, Gloor P, Kaplan R. Transient global amnesia: the electroencephalogram at onset. Ann Neurol. 1987;22:771–2.
85. Hiraga A, Matsunaga T. Transient global amnesia and gastroscopy. J Neurol Neurosurg Psychiatry. 2006;77:995–6.
86. Neuzillet C, Merrouche M, Jouet P, Sabate JM, Coffin B. Transient global amnesia induced by esophageal functional exploration [in French]. Gastroenterol Clin Biol. 2009;33:1068–70.
87. Sayilir A, Kurt M, Ibis M, Kekilli M, Onal IK, Sasmaz N. Transient global amnesia following upper gastrointestinal endoscopy without premedication. Gastroenterol Nurs. 2009;32:362.
88. Ahn S, Kim W, Lee YS, et al. Transient global amnesia: seven years of experience with diffusion-weighted imaging in an emergency department. Eur Neurol. 2011;65:123–8.
89. Cesar S, Perdigao S. Transient global amnesia after gastroscopy. J Neurol. 2012;259(Suppl1):S140–1. (abstract P558)
90. Jeong M, Kim WS, Kim AR, Park JJ, Choi DH, Kim HY. Medical procedure-related transient global amnesia. Eur Neurol. 2018;80:42–9.

91. Benke T, Chemelli A, Lottersberger C, Waldenberger P, Karner E, Trinka E. Transient global amnesia triggered by the intracarotid amobarbital procedure. Epilepsy Behav. 2005;6:274–8.
92. Ravaglia S, Zito A, Ahmad L, Canavero I. How to forget a "traumatic" experience: a case report of transient global amnesia after nasopharyngeal swab for coronavirus disease 19. BMC Neurol. 2021;21:266.
93. Hirschfeld G, Sperfeld AD, Kassubek J, Scharffetter-Kochanek K, Sunderkotter C. Transient global amnesia (TGA) during an oral provocation test [in German]. Hautarzt. 2007;58:149–52.
94. Reinholz M, Heppt MV, Hoffmann FS, et al. Transient memory impairment and transient global amnesia induced by photodynamic therapy. Br J Dermatol. 2015;173:1258–62.
95. Robbins MS, Breidbart DH, Robbins HY. Transient global amnesia complicating pulmonary function testing. Respir Med CME. 2010;3:230–1.
96. Williamson JC, Larner AJ. Confused after spirometry: a unifying diagnosis. BMJ Case Rep. 2016;2016:pii:bcr2016216645.
97. Mokabberi R, Assal C, Afsaneh HM, Storm R, Dandamudi G. Transient global amnesia after ablation of premature ventricular beats arising from the right coronary cusp. Indian Pacing Electrophysiol J. 2010;10:372–5.
98. Park S, Park S, Jang Y. Transient global amnesia after stellate ganglion block. J Anesth. 2015;29:643.
99. Gomez-Choco M, Mariaca AF, Gaebel C, Valdueza JM. A controlled Valsalva maneuver causes neither diffusion-positive hippocampal lesions nor clinical symptoms after transient global amnesia. Eur Neurol. 2019;82:113–5.
100. Jeong M, Jin J, Kim JH, Moon Y, Choi JW, Kim HY. Incidental hippocampal hyperintensity on diffusion-weighted MRI: individual susceptibility to transient global amnesia. Neurologist. 2017;22:103–6.