Chapter 3
Differential Diagnosis of TGA

Abstract This chapter considers the differential diagnosis of TGA. Key considerations include cerebrovascular disease (TIA, stroke), epilepsy (transient epileptic amnesia, TEA) and psychological causes, as well as a variety of other causes of transient amnesia (migraine, adverse drug effect, hypoglycaemia, head injury) and transient cerebral disorder (delirium, infection). On clinical grounds alone, it is often possible to distinguish TGA from other causes of transient amnesia.

Keywords TGA · TEA · TIA · Psychogenic amnesia

There are a number of symptomatic causes of amnesia [1, 2] which, if transient (Table 3.1), may sometimes be mistaken for TGA. However, the differential diagnosis of TGA covers more than just amnesic syndromes (Table 3.2). Some of these conditions will be considered in this chapter.

As may be expected for an acute and transient syndrome, most patients with transient global amnesia (TGA) who come to medical attention are seen by primary care physicians working in community settings or acute care physicians based in district general hospitals rather than by cognitive neurologists in dedicated tertiary neuroscience centres (unless specific services and care pathways have been established). In one small survey involving eight definite cases seen by one neurologist, three were seen in outpatient clinics, five as ward consultations; the majority (7/8, = 88%) were seen in district general hospitals. Of note, the working or suggested diagnoses (sometimes more than one) of the referring clinicians, which were available in seven cases at the time of referral to the neurologist, were stroke or TIA (5 cases), epilepsy (2) and viral illness (1) [4]. Certainly, the former two diagnoses feature prominently in the differential diagnosis of TGA, which also encompasses psychiatric or psychological disorder ([5], p.49–57).

The contrasts between TGA and some of these other conditions are summarised in Table 3.3, although this should not be taken to imply that the clinical differences are always necessarily clear cut. The differential diagnosis of TGA is considered here in greater detail.

A.J. Larner, *Transient Global Amnesia*,
https://doi.org/10.1007/978-3-030-98939-2_3

Table 3.1: Differential diagnosis of amnesia (adapted from [3], p.242–3)

• Acute/transient:
Transient global amnesia (TGA)
Transient epileptic amnesia (TEA)
Transient psychological amnesia (TPA)
Migraine
Adverse drug effect
Hypoglycaemia
Traumatic brain (closed head) injury
• Chronic/persistent:
Alzheimer's disease
Wernicke–Korsakoff syndrome
Sequela of herpes simplex encephalitis
Limbic encephalitis (paraneoplastic or non-paraneoplastic)
Hypoxic brain injury
Bilateral paramedian thalamic infarction/posterior cerebral artery occlusion ("strategic infarct dementia")
Third ventricle tumour, cyst; fornix damage
Temporal lobectomy (bilateral, or unilateral with previous contralateral injury, usually birth asphyxia)
Focal retrograde amnesia

Table 3.2: Differential diagnosis of TGA

• Causes of transient amnesia:
Transient epileptic amnesia (TEA)
Transient psychological amnesia (TPA)
Migraine
Adverse drug effect
Hypoglycaemia
Traumatic brain (closed head) injury
Alcohol-induced amnesia
Fatigue amnesia
• Causes of acute cerebral disorder:
Transient ischaemic attack (TIA)
Acute confusional state/delirium/toxic-metabolic encephalopathy
Intracerebral haemorrhage/subarachnoid haemorrhage
Acute brain infection (encephalitis)

3.1 Cerebrovascular Disease

In the light of the apparently sudden onset of neurological dysfunction in TGA, it is easy to understand why the possibility of cerebrovascular disease featured amongst the pathogenic considerations in early descriptions of the disorder, such as those of Guyotat and Courjon (1956) [7], Poser and Ziegler (1960) [8] and Halsey (1967) [9]. Fisher and Adams noted in their monograph on TGA that "[t]he possibility that such an episode might have been the first evidence of an approaching stroke … was responsible for our seeing so many of these patients" ([10], p.46). Clinicians unfamiliar with TGA may still consider stroke or transient ischaemic attack (TIA) as

Table 3.3: Comparison of typical features of transient global amnesia (TGA), transient epileptic amnesia (TEA), transient ischaemic attack (TIA) and transient psychological amnesia (TPA) (adapted from [6])

Clinical feature	TGA	TEA	TIA	TPA
Anterograde amnesia during attack	Yes	Yes	Yes	No
Focal neurological deficits	No	No	Yes	No
Aura, automatisms	No	Yes	No	No
Symptom duration	<24 h	Usually <1 h	<24 h	Variable
Recurrence rate	Low	High	Varied	Varied
Triggers	Emotional stress or physical exertion	Can occur on waking	No	Emotional stress
Responds to antiepileptic drugs	No	Yes	No	No
EEG abnormalities during attack	No	Yes	No	No

foremost amongst the possible causes [4], justifiably since stroke was the most important differential diagnosis of in a recent large series of suspected TGA cases (6.6%) [11].

On occasion, TGA has been associated with various cerebrovascular events, including infarction and haemorrhage, both arterial and venous, TIA and other conditions affecting the vasculature. Some of these reports have involved memory eloquent brain substrates (medial temporal lobe, thalamus, fornix, corpus callosum, hippocampus), elements within the circuit described by Papez [12], and hence plausible as causes of memory disorder. However, caveats apply before a causal relation between these cerebrovascular disorders and TGA may be accepted. For example (as previously mentioned, see Sect. 2.2.1), many of these cases were reported prior to the definition of widely accepted diagnostic criteria for TGA, and the presence of possible confounding factors may sometimes be identified. Cases of posterior circulation stroke or TIA and of TGA may be elided in some reports. For example, at least one of Lou's (1968) patients with "repeated TGA" probably had ischaemic events (3 episodes causing a persistent memory deficit which gradually improved, along with a right upper quadrantanopia and right limb paraesthesia) [13]. Likewise, de Tribolet et al. [14] reported six cases of cortical blindness and amnesia, which were likely to be due to stroke, but also two cases of transient "Korsakoff's syndrome", more likely to have been examples of TGA. Posterior cerebral artery occlusion is a recognised cause of amnesia (e.g. [15]), and transient amnesia has been reported on occasion to herald brainstem infarction (e.g. [16, 17]). A TGA-like syndrome ("TGA plus") has been reported in pure hippocampal stroke with additional aphasia [18] and in corpus callosum infarction [19], although the nosological position of "TGA-like syndrome" is questionable (Sect. 2.2.2). These entities might be better labelled "amnesic stroke". Certainly amnesia, both persistent and transient, can be a result of strategic infarcts (e.g. [20, 21]).

With the advent of magnetic resonance (MR) brain imaging, the frequent observation of focal punctate areas of high signal change in the hippocampus on diffusion-weighted imaging MR sequences (MR-DWI) has been interpreted as evidence of cerebral ischaemia, albeit not typical of infarction (see Sect. 5.1.2, especially Sect. 5.1.2.6 for a discussion of the pathogenesis of these imaging changes).

3.1.1 Transient Ischaemic Attack (TIA)

Arguments against TGA being a form of TIA include both clinical and epidemiological considerations. TIAs are usually accompanied by focal neurological signs (e.g. hemiparesis, amaurosis fugax) which are absent from TGA (by definition, according to the 1990 diagnostic criteria of Hodges and Warlow [22]; see Sect. 2.2.2 and Table 2.1). Furthermore, episodes of TGA are usually isolated, whereas recurrence rates are much higher in TIA (Table 3.3), sometimes with progression to established stroke, which is not seen in TGA. Comparison of vascular risk factors in patients with TGA and TIA has generally found a significantly greater prevalence in the latter group, with the risk factor profile in TGA patients resembling that of normal controls (see Sect. 7.11 for extended discussion).

Chen et al. [23] described a patient (M76) with "a spell of TGA" followed by several episodes of amaurosis fugax (ocular TIA) who was found on investigation to have progressive occlusion of the right common carotid artery. The concurrence of events was taken to imply a vascular aetiology for TGA.

3.1.2 Stroke: Cerebral Infarction

Occasional cases with the typical clinical phenotype of TGA and with computed tomography (CT) changes indicative of established ischaemic stroke were reported when this neuroimaging modality first became widely available (e.g. [24–26]).

With the advent of higher resolution magnetic resonance imaging (MRI), further cases were identified. A narrative review [27] (Table 3.4) found descriptions of TGA in association with infarction in various locations, including the medial temporal lobe [28–32], hippocampus [18, 31, 33–40], fornix [41, 42], thalamus [43–45], cingulate gyrus or bundle [46–48], striatum (caudate and putamen) [49–53], corpus callosum [19, 54] and frontal lobe [39, 55]. Thus, these strokes involved memory eloquent brain structures, linked through Papez circuit [12], in many cases, as well as frontal lobe structures involved in the organisation and monitoring of memory processes, but with an absence of significant hemisphere strokes. This localisation suggests that stroke-related TGA might be regarded as a (rare) symptomatic (or secondary) form of TGA. Possible pathogenic reasons for the occasional concurrence of ischaemic stroke and TGA are considered later (Sect. 9.2.1). Suffice it to say here that the phenotype of TGA may occur on rare occasions in association with established stroke on neuroimaging.

Table 3.4: Reports of MR-confirmed acute infarction or stroke associated with the clinical phenotype of TGA

Location	Reference	Demographic and other clinical features	MR imaging findings
Temporal lobe			
	Greer et al. (2001) [28]	F77	Left mesial temporal lobe ischaemic infarct
	López-Pesquera et al. (2005) [29]	F49	Tiny ischaemic stroke in white matter of left temporal lobe
	Graff-Radford et al. (2013) [30]	F56; following coiling of small posterior circulation cerebral aneurysm	Small medial temporal lobe strokes
	Duan et al. (2016) [31]	M72; coronary angiography	Acute infarction in left hippocampus and temporal lobe
	Ramanathan & Wachsman (2021) [32] (n = 2)	F48 history of hypertension, COVID-19 +ve F71 history of hypertension, COVID-19 +ve	Bilateral medial temporal lobe infarcts Small R temporal lobe infarct
Hippocampus			
	Adler et al. (2012) [33]	F65	Subtle ischaemic region in the right hippocampus compatible with acute infarct
	Carota et al. (2012) [18]	R41; "TGA plus" (anomic pauses, "amnesic aphasia"); patent foramen ovale	Acute infarct, dorsal part of left hippocampal body
	Gungor-Tuncer et al. (2012) [34] and (2015) [35] (case 2)	F62; history of migraine	Left pons (7h); left hippocampal and right frontal areas (36h)
	Li and Hu (2013) [36]	M61	Bilateral hippocampal lesions, acute ischaemia
	Gungor-Tuncer et al. (2015) [35] (case 1)	F56; history of migraine	Two punctate acute infarcts in the left hippocampus
	Duan et al. (2016) [31] (n = 2)	M73; cerebral angiography, vertebral artery angioplasty M72; coronary angiography	Acute infarction in left hippocampus Acute infarction in left hippocampus and temporal lobe
	Naldi et al. (2017) [37]	F82	Right posterior hippocampal stroke
	Yun et al. (2017) [38]	M68	Bilateral hippocampal lesions

(continued)

Table 3.4: (continued)

Location	Reference	Demographic and other clinical features	MR imaging findings
	Kang et al. (2021) [39]	M54	Right frontal and hippocampus strokes
	Sakihara et al. (2021) [40]	F35; septic embolus from infective endocarditis	Right hippocampus
Fornix			
	Gupta et al. (2015) [41]	F66; paroxysmal atrial fibrillation	Body and left column of fornix infarction
	Meyer (2016) [42]	N/A	Left fornix infarction
Thalamus			
	Pradalier et al. (2000) [43]	F54; history of migraine without aura	Right anteroinferior thalamic ischaemic lesion
	Giannantoni et al. (2015) [44]	F69	Thalamic ischaemic lesion
	Dogan et al. (2017) [45]	F65	Left thalamus and left paramedian mesencephalon infarcts
Cingulate gyrus			
	Gallardo-Tur et al. (2014) [46]	M62; two TGA episodes	Acute ischaemic stroke of small size (15 mm maximal diameter) at right cingulate gyrus
	Chau and Liu (2019) [47]	F60	Left cingulate gyrus
	Meng et al. (2021) [48]	F89; history of hypertension	L retrosplenial infarct (cingulate bundle and retrosplenial cortex)
Striatum (caudate, putamen)			
	Ravindran et al. (2004) [49]	M56	Acute ischaemia in the body of right caudate nucleus
	Kim et al. (2012) [50]	F63	L putamen acute microinfarct
	Koltermann et al. (2015) [51]	M50	Acute ischaemic lacunar infarction, head of caudate nucleus
	Yoshida (2017) [52]	F67	Lacunar infarction of the left putamen
	Tarazona et al. (2021) [53]	F89; history of migraine	R lenticular nucleus (outermost putamen)
Corpus callosum			
	Saito et al. (2003) [54]	M58	Small lesion of high signal intensity in the left retrosplenium of the corpus callosum

(continued)

Table 3.4: (continued)

Location	Reference	Demographic and other clinical features	MR imaging findings
	Beyrouti et al. (2016) [19]	M62	Infarction of genu and body of corpus callosum
Frontal lobe			
	Kim et al. (2018) [55] ($n = 3$)	No details	1. Left orbitofrontal. 2. Left prefrontal. 3. Right frontal and left parietal.
	Kang et al. (2021) [39]	M54	Right frontal and hippocampus strokes

Table 3.5: Reports of cerebral haemorrhage or haematoma associated with the clinical phenotype of TGA

	Location	Reference(s)
Haemorrhage		
	Left temporal haemorrhage	Landi et al. 1982 [60]
	Subarachnoid haemorrhage	Sandyk 1984 [63] Monzani et al. 2000 [61]
	Left frontal haemorrhage	Jacome and Yanez 1988 [59]
	Haemorrhage into a tumour	Sorenson et al. 1995 [64] Honma and Nagao 1996 [58]
	Cingulate gyrus haemorrhage	Yoon et al. 2006 [65]
Haematoma		
	Intraventricular haematoma	Heon et al. 1972 [57]
	Subdural haematoma	Chatham and Brillman 1985 [56]
	Left thalamic haematoma	Moonis et al. 1988 [62]

3.1.3 Stroke: Cerebral Haemorrhage

Intracranial haemorrhage (intracerebral, subdural or subarachnoid) may potentially be confused with TGA by virtue of its acute onset, but amnesia is seldom a prominent feature, and there is often impairment of consciousness. Intracranial haemorrhage or haematoma has on occasion been reported in association with TGA [56–65] (see Table 3.5), but the exact diagnostic status of such cases is uncertain, possibilities including misdiagnosis of TGA or chance concurrence.

3.1.4 Cerebral Vasculopathies

TGA has been reported on occasion with a variety of other disorders affecting the cerebral vasculature.

An amnesic syndrome following anterior communicating artery rupture and/or surgery is well recognised, but only one account of cerebral aneurysm associated

with transient amnesia resembling TGA has been found. The event was apparently triggered by a coiling procedure in the posterior circulation, with evidence of medial temporal lobe strokes found on diffusion-weighted MR imaging [30].

Intracranial dural arteriovenous fistula (dAVF) may sometimes present with cognitive deficits suggestive of a dementia syndrome (e.g. [66]), sometimes rapidly progressing [67]. However, only two definite reports of TGA with dAVF have been identified [68, 69], one with recurrent events [68]. The patient reported by Heine et al. [70] might be another example (see [71], p.189).

Occasional cases of TGA have been reported in association with the reversible cerebral vasoconstriction syndrome (RCVS), a condition typically characterised by severe headaches, including thunderclap headache, and reversible segmental cerebral artery vasoconstriction which may be complicated by ischaemic or haemorrhagic stroke [72–74]. Like migraine (Sect. 3.4.1 and Sect. 7.9) and primary headache associated with sexual activity (Sect. 8.4), headache in this context may be a consequence of activation of the trigeminocervical complex. The posterior reversible encephalopathy syndrome (PRES) may be related to RCVS, sharing some features and risk factors. PRES is typically characterised by headache, visual field and motor deficits, confusion, impaired consciousness and seizures, again with ischaemic or haemorrhagic lesions. TGA has on occasion been described in association with PRES [75, 76].

Other disorders sometimes associated with vasculopathy that have on occasion been reported in association with TGA include Sneddon syndrome [77], scleroderma [78] and cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) [79].

Thrombotic tendencies might be relevant to TGA pathogenesis, secondary to cerebral venous outflow obstruction, as, for example, in antiphospholipid antibody syndrome in which TGA has occasionally been reported ([80, 81]; possibly [82]). However, venous thrombosis of cerebral [83, 84] or cervical (jugular) veins [85] has rarely been reported in association with TGA (see Sect. 9.2.2 for discussion of venous outflow obstruction as a possible aetiological factor in some cases of TGA).

3.1.5 Cerebral Angiography

One of the earliest possible reports of TGA (see Sect. 1.2) related to catheter angiography of the vertebral artery [86]. Further instances of angiography-related memory disturbance, some of which may be cases of TGA, have been reported (Table 3.6), involving procedures visualising both cerebral (carotid or vertebral) and coronary arterial vasculature (but apparently not peripheral limb vasculature [31]). One case related to renal artery angiography has been reported [124]. The procedural use of benzodiazepines (Sect. 3.4.2) may be a confounding factor in some of these reports.

The mechanism(s) by which angiography might trigger TGA remain(s) uncertain. Suggestions have included arterial spasm and the injection of contrast material (both ionic and non-ionic). Neuroradiological evidence of ischaemia, in the form of

Table 3.6: Reports of angiography and dissection associated with the clinical phenotype of TGA

		References
Angiography	Cerebral	Hauge (1954) (*n* = 3) [86] Deak and Toth (1964) [87] Whishart 1971 [88] de Tribolet et al. (1975) [14] Wales and Nov (1981) (*n* = 2) [89] Cochran et al. (1982) (*n* = 7) [90] Haas (1983) [91] Pexman and Coates (1983) (*n* = 12) [92] Giang and Kido (1989) (*n* = 2) [93] Minuk et al. (1990) [94] Juni et al. (1992) [95] Brady et al. (1993) [96] Schamschula and Soo (1994) (*n* = 2) [97] Jackson et al. (1995) (*n* = 6) [98] Meder et al. (1997) [99] Woolfenden et al. (1997) [100] Kapur et al. (1998) [101] Tanabe et al. (1999) [102] Kim et al. (2006) [103] Foss-Skiftesvik et al. (2015) [104] Duan et al. (2016) (*n* = 5) [31] Tiu et al. (2016) [105] Lee (2020) [106]
	Coronary	Fischer-Williams et al. (1970) [107] Shuttleworth and Wise (1973) (*n* = 2) [108] Lockwood et al. (1983) [109] Koehler et al. (1986) [110] Yildiz et al (2003) [111] Kurokawa et al. (2004) (*n* = 2) [112] Fernandez et al. (2005) [113] Wong et al. 2005 [114] Udyavar et al. (2006) [115] Duan et al. (2016) (*n* = 4) [31]
Dissection	Aorta	Rosenberg (1979) [116] Gaul et al. (2004) [117] Mondon et al. (2007) [118] Irioka et al. (2009) [119] Colotto et al. (2011) [120] Kaveeshvar et al. (2015) [121]
	Vertebral artery	Michel et al. (2004) [122] Yokota et al. (2015) [123]

small acute infarctions in the hippocampus, has been documented in some cases (e.g. [31]). Angiography-related TGA might also conceivably be related to inadvertent, iatrogenic, arterial dissection at the time of the procedure, predisposing to embolisation. Similar explanations might pertain in a case following carotid artery stenting followed by carotid angiography [106]). TGA has also been reported after vertebral artery angioplasty and stenting [103].

TGA has been described with arterial dissections, of either the aorta or the vertebral artery (Table 3.6). However, no reports of TGA in fibromuscular dysplasia, a disorder associated with arterial dissection, have been identified.

Another pathogenic possibility relates to migraine (Sect. 3.4.1 and 7.9): certainly, migrainous phenomena may on occasion be triggered by angiographic procedures (e.g. [125]), and this may have played a role in the angiography-related case of Fernandez et al. [113].

3.1.6 Cardiac Disorders

In addition to episodes related to coronary angiography (see Sect. 3.1.5; Table 3.6), TGA has also been reported on occasion in association with a variety of coronary syndromes including acute myocardial infarction [126–129], cardiac arrhythmia [130] and cyanotic heart disease [131]; mitral valve prolapse has also been mentioned [132]. However, the paucity of reports suggests that these might be simply examples of chance concurrence, unrelated to the cardiac event or disorder, although cerebral hypoperfusion or embolism might occur in these situations and be a precipitating factor for TGA.

A cardiac condition that might be pathogenically relevant to TGA is Takotsubo cardiomyopathy, or the "broken-heart syndrome", concurrence with which has been reported on several occasions (e.g. [133–142]). Petrea et al. speculated that the catecholamine surge associated with myocardial stunning in Takotsubo cardiomyopathy might also be associated with "cortical stunning" and hence that these conditions might have a shared pathogenesis [137]. Of possible interest, in a review of over 1100 reports of Takotsubo cardiomyopathy, emotional and physical stressors preceded the syndrome in 39% and 35% of patients, respectively [143]; these are also significant recognised precipitating factors for TGA (see Sect. 8.1 and 8.2). Hence, the two conditions may have a shared pathogenesis, leading some to characterise TGA as "the cerebral Takotsubo" [144]. Myocardial injury, assessed by means of highly sensitive assays for cardiac troponin, was found in 28 of a series of 113 TGA patients [145]. It might be of interest to investigate whether the unique signature of microRNAs which has been reported to distinguish Takotsubo cardiomyopathy from acute myocardial infarction [146] is also seen in TGA.

Klötzsch et al. [147] reported an increased frequency of patent foramen ovale (PFO) in patients with TGA but this finding has not, to my knowledge, been replicated. PFO may be associated with paradoxical embolism, which might conceivably be of relevance to TGA pathogenesis. TGA occurring immediately after right-left shunt of saline contrast during transoesophageal echocardiography has been reported [148]. Maalikjy Akkawi et al. [149] examined TGA, TIA and control patients for evidence of PFO with contrast transcranial duplex sonography but found no difference between the three groups. Noh and Kang reported that TGA

patients with PFO had fewer vascular risk factors than those without PFO and suggested that paradoxical embolus might be a cause of TGA in these patients [150].

PFO is certainly associated with an increased risk of decompression sickness in divers. TGA has been reported in divers and ascribed to breathing hyperoxic mixtures, but no data on PFO were presented [151]. Cold water immersion (see Sect. 8.3) might also be relevant to diving-related TGA cases. In the light of the putative link between TGA and migraine (Sect. 7.9), the observation that PFO is probably more prevalent in patients with migraine might be significant, although whether the relationship between migraine and PFO is causal or coincident remains unclear [152].

3.2 Epilepsy

The sudden onset of neurological dysfunction in TGA has suggested to some authors the possibility of an epileptic aetiology. Fisher and Adams ([10], p.46) certainly considered it as a cause, and the possibility has recurred from time to time (e.g. [153–157]) and may still be questioned by some clinicians who are not familiar with TGA [4]. Certainly, Miller Fisher [158], a clinician with a deep knowledge of cerebrovascular disease [159–162], continued to argue that TGA was a form of seizure affecting the hippocampal-diencephalic system.

3.2.1 Transient Epileptic Amnesia (TEA)

Probably, the earliest account of attacks of transient amnesia of epileptic origin was by John Hughlings Jackson (1835–1911) in his 1888 report of his physician patient known as "Dr Z" [163]. However, although occasional cases of epileptic amnesia have subsequently been reported (e.g. [164–169]), it was not until the 1990s that the syndrome of transient epileptic amnesia (TEA) was more fully characterised by Kapur [170] and by Zeman et al. [171] and systematic studies and reviews subsequently undertaken (e.g. [172–180]). Diagnostic criteria for TEA have been suggested ([171] and [180], p.143) (Table 3.7; compare with Table 2.1).

Transient epileptic amnesia (TEA) is a distinctive epilepsy syndrome (Table 3.3), characterised by brief amnesic episodes, usually lasting 1 hour or less in duration, and often occurring on waking from sleep (Case Study 3.1). Attacks may be accompanied by other features suggestive of epilepsy such as automatisms or olfactory hallucinations. Hence, it may be worth asking patients who complain of autobiographical amnesia whether or not they also have automatisms or olfactory hallucinations as possible pointers to an epileptic aetiology. There is a high recurrence rate for episodes of TEA, contrary to the observations in TGA (Sect. 2.1.5).

Table 3.7: Diagnostic criteria for TEA (based on [171] and [180], p.143)

Recurrent witnessed episodes of transient amnesia.
Other cognitive functions intact.
Evidence of epilepsy:
(a) Other clinical features of epilepsy.
(b) Response to anticonvulsant medication.
(c) Epileptiform abnormalities on EEG.

Case Study 3.1: Transient Epileptic Amnesia (TEA)
A 43-year-old man and his wife reported episodes over 1 year in which he could not remember things on waking in the mornings, accompanied by a blank facial expression. There was also a history of accelerated forgetting of events which had occurred a couple of weeks earlier, such that he could not recall a recent holiday or conversations. The history was thought to be typical for the diagnosis of transient epileptic amnesia (TEA). The patient was unimpaired on cognitive screening instruments (Addenbrooke's Cognitive Examination-Revised score = 100/100). MR brain imaging, standard and sleep-deprived EEG were all within normal limits. Initiation of antiepileptic drug therapy (carbamazepine) was followed by a remission of episodes over a 2-year period of follow-up.

Many TEA patients also report interictal memory problems, characterised as accelerated long-term forgetting and autobiographical amnesia; the latter may be prominent [181, 182]. An accelerated loss of new information and impaired remote autobiographical memory has been demonstrated in TEA patients, but the aetiology of these deficits remains uncertain, possibilities including ongoing seizure activity, seizure-induced medial temporal lobe damage or subtle ischaemic pathology [182]. Accelerated forgetting has also been described in medial temporal lobe epilepsy [183]. Symptoms of emotional lability, in particular pathological tearfulness or labile crying in response to relatively minor stimuli, has also been reported in the context of TEA [173, 184].

The syndrome of "isolated autobiographical amnesia" [185] may be related to TEA. Likewise, some patients who have been reported with the syndrome of focal (isolated) retrograde amnesia [186] (Sect. 3.3) may have an underlying epileptic disorder, possibly related to other brain insults such as encephalitis or alcohol misuse ([187, 188]; see also discussion in [189]).

Electroencephalography (EEG) in TEA may be associated with clear-cut seizure activity during amnesic episodes. Abnormalities may be found in interictal EEG recordings in about one-third of TEA patients, although sometimes sleep-deprived EEG may be required. Magnetic resonance brain imaging may show hippocampal atrophy [190] or amygdala enlargement [191–193] (Case Study 3.2).

Management of TEA may require antiepileptic drug therapy. TEA generally responds favourably to standard antiepileptic medications such as sodium valproate, carbamazepine, lamotrigine or levetiracetam. Advice on appropriate lifestyle modifications, including reference to statutory restrictions on driving, is also an integral aspect of management.

TEA is an infrequent condition. Over the 20-year period 2002–2021 inclusive, the author has encountered only six definite cases (e.g. Case Studies 3.1 and 3.2; [192, 194]), all male, as compared to the predominance of females in cases of TGA (n = 50) seen over the same period (F:M = 29:21; Figure 7.2). In addition, one further possible case has been seen, in which the episodes were initially diagnosed by another consultant neurologist as parasomnias. These episodes on waking occurred at approximately the same age at onset as a more pervasive memory problem which evolved into Alzheimer's disease (AD) [195]. Epileptic seizures in AD may take a number of forms and become more frequent with disease duration although they may occur at onset of cognitive decline [196], so this concurrence might possibly reflect shared pathogenic processes involving synaptic network pathology in the medial temporal lobes [197–200]. TEA has also been suggested as a cause of wandering behaviours observed in AD patients [201].

TEA is usually idiopathic but may sometimes be secondary or symptomatic. Cases associated with medial temporal lobe mass lesions are described, some of which have also manifested episodes more typical of TGA, e.g. of longer duration, and following physical exertion [191, 192, 194, 202], prompting the suggestion that all cases of TGA associated with focal medial temporal lobe tumours are in fact TEA masquerading as TGA [194]. TEA may also on occasion be associated with neurodegenerative disease, such as AD [195, 201]. TEA has also been described as the presenting feature of autoimmune limbic encephalitis in association with various autoantibodies, including NMDAR [203], CASPR2 [204] and $GABA_B$ [205].

Whereas a family history of TGA may sometimes be uncovered (Sect. 7.8), I am aware of only two reports of a possible family history of TEA, one affecting three siblings [206], the other in a 20-year-old man, his mother and grandmother [207].

TEA enters the differential diagnosis of TGA, which it may resemble, but from which it usually differs in a number of respects, including the timing and frequency of attacks (Table 3.3). The key points of differentiation are that TEA attacks are generally briefer in duration and have a higher recurrence rate than TGA. As a rule of thumb, a cut-off of about 2 hours has been used to differentiate TEA from TGA, but some caution is needed as brief (<1 h) episodes of TGA are recognised [208]. There may also be an impression that the anterograde amnesia is denser in TGA than in TEA, patients with the latter condition having partial recall.

The "absence of epileptic features" is one of the proposed diagnostic criteria for TGA [22], although EEG is seldom performed during an episode of TGA, other than fortuitously, and is normal (e.g. [209]; Sect. 4.2.1 and 4.2.2).

3.2.2 TGA and TEA: Is there an Interrelation?

The distinction between TGA and TEA is not always as clear cut as might be implied by presentations such as Table 3.3. For example, some patients reported in the literature as having "TGA" may, in retrospect, have in fact had TEA, e.g. Greene and Bennett's patient who had amnesia on awakening and EEG abnormality [210], although Daniel ([71], p.63) seems to accept this as a case of TGA. In the Oxford TGA study, an unexpected finding was that 8 of 114 patients with apparent TGA (7%) subsequently developed epilepsy, usually of complex partial type, prompting the view that the original attacks were in fact due to seizures ([5], p.41,46–7,56,121,123,124–5,137). In a series of 64 TGA patients reported by Zorzon et al., three were eventually considered to have an epileptic aetiology [211].

Aside from diagnostic confusion, it is possible that there may be an interrelationship between TGA and TEA. Occasional patients have been reported with episodes resembling both TGA and TEA, the latter following the former, with associated medial lobe structural abnormalities on MR imaging (e.g. [191, 192, 194, 202, 212, 213]; Case Study 3.2). These cases raise the possibility that TEA and TGA are not mutually exclusive conditions but may in some instances be interrelated. In the light of the known vulnerability of hippocampal CA1 neurones to transient ischaemia (e.g. [214]) with subsequent apoptosis, perhaps TGA episodes, particularly if recurrent, might damage the hippocampus ([215, 216]; see Sect. 5.1.2 and 5.2.1) in such a way (ischaemic scarring) that the threshold for epileptic attacks is subsequently reduced (Case Study 3.2). Hence, rather than epilepsy being simply mistaken for TGA ([5], p.56), it might be that some of these cases represent an evolution from episodes of TGA to epilepsy.

Case Study 3.2: A relationship between TGA and TEA

A 66-year-old man reported four episodes of transient amnesia over a 6-month period. Each episode occurred within hours of strenuous physical exercise. In the first, he returned home from a bicycle ride confused about the route he had taken. The second event occurred following a walk up a steep incline. The third event occurred the day after a strenuous bicycle ride when the patient awoke in the morning confused as to where he was and what the plan for the day was. This confusion recurred the same day following a post-prandial nap. All the events were witnessed by the patient's wife who noted repetitive questioning to be a feature in each. All lasted between 30 min and 2 h with complete recovery. No other accompanying focal neurological symptoms were noted during the attacks.

At initial neurological assessment, neurological examination and cognitive screening were normal. The first two events were thought to be typical of exercise-related TGA, whereas the latter two had clinical features more suggestive of TEA, particularly the relationship to waking from sleep.

On further follow-up, more events occurred, exclusively related to waking from sleep. Standard electroencephalogram (EEG) was within normal limits, but sleep-deprived EEG showed excess slow waves over the right temporal region and one prolonged run of slow waves followed by brief high amplitude sharp wave bursts. Magnetic resonance (MR) brain imaging showed subtle but unequivocal enlargement of the right amygdala with normal diffusion-weighted imaging and no disruption of limbic white matter tracts or adjacent temporal fibre bundles. The patient was treated with levetiracetam (500 mg bd).

Clinical and neuroradiological follow-up of this patient now extends to 8 years. There has been complete cessation of all amnesic events since prescription of levetiracetam, with no dosage increase required. The patient has noted blank areas in his memory for distant significant personal events, suggestive of autobiographical amnesia, but cognitive screening has remained normal. MR brain imaging, initially performed annually, showed no change in the amygdala enlargement, but between year 6 and year 8 the appearances reverted to normal.

Although it is possible that all the events were epileptic in origin, another possibility is that initial attacks of TGA left residual hippocampal damage resulting in seizure activity or lowered seizure threshold [192, 194].

3.3 Transient Psychological Amnesia (TPA)

Transient amnesia of psychological origin (TPA) enters the differential diagnosis of TGA (Table 3.3). TPA has variously been designated, for example, as hysterical amnesia, fugue state, psychogenic amnesia, functional amnesia, focal retrograde amnesia and dissociative amnesia [186, 217–220].

Once thought to be common, the number of reported cases appears to have declined since the 1950s, at roughly the same time as TGA was becoming recognised as a clinical entity. It has been speculated that earlier cases of TGA might have been "immersed in the literature on psychogenic amnesia" ([5], p.4; but see also [221]). The largest reported patient series of TPA in recent times included 53 patients seen over a period of nearly 20 years [217].

TPA may be differentiated from TGA on a number of grounds (Table 3.3) [222]. Attacks tend to be longer, lasting from days to months or even years. The patient's loss of personal identity is a clear differentiating factor of TPA from TGA [217]. Functional amnesias are typically retrograde in nature, with relatively sparing of anterograde memory, hence a reversal of the typical (Ribot) gradient seen in other forms of amnesia, persistent and transient, including TGA [217, 223]. Patients may be far from their home, with no clear history of how they got there (fugue state), sometimes resulting in media coverage to try to identify the individual.

The behavioural disturbances sometimes seen in TGA (Sect. 2.1.2) are generally not a feature of TPA, wherein patients are often not obviously distressed by their amnesia. This may be because they are apparently able to learn new information, despite the dense retrograde amnesia. TPA patients also tend to be younger than patients with TGA.

Once available, there is often a previous history of mood disorder such as depression, and often a clear stressful precipitating event such as relationship or financial problems, and minor head injury. Failure to recognise family members, once located, is common. Spontaneous recovery of memory may occur after a variable time period, with the prognosis for fugue states particularly favourable [217].

The portrayal of characters with amnesia in motion pictures almost invariably features loss of personal identity [224, 225], as seen in TPA, no doubt for dramatic effect.

3.4 Other Symptomatic Causes of Transient Amnesia

Transient amnesia may result from a variety of other conditions and causes (Table 3.2).

3.4.1 Migraine

The possible pathogenic relationship between TGA and migraine is considered in more detail later (Sect. 7.9 and Sect. 9.3). Here, it is simply noted that, amongst the many transient phenomena that may be encountered in the context of migraine attacks, amnesia is sometimes prominent (Case Study 3.3).

Case Study 3.3: Migraine amnesia
A 27-year-old lady was referred to the clinic following a strange experience whilst driving her car. During daylight hours, she set off on the familiar route to her boyfriend's house, part of which involved driving along a motorway. She recollected joining the motorway, but then had no recollection until she found herself six junctions and several miles further on, when she should have turned off after only three junctions. At this point, she stopped to telephone her boyfriend to explain what had happened and that she would be late. On arrival, he noted that she looked shaken, complained of a headache and took some analgesics, but on direct questioning there was no history of repetitive questioning or loss of personal identity. The patient had a prior history of migraine as a teenager, and headaches had recurred some 5 months earlier. Subsequent neurological examination and structural brain imaging were normal. The provisional diagnosis of her "unconscious driving phenomenon" was migraine (adapted from [226]).

Moersch (1924) [227] was perhaps the first to emphasise amnestic dysfunction occurring in migraine attacks. Comorbidity of TGA and migraine was noted in some of the earliest reports of TGA (e.g. [8, 228–230]). Frank (1976) compared amnesic episodes in migraine ("Migranedammerattacken") with reports of TGA and was of the view that they "seem to be identical" [231]. TGA occurring during a migraine attack has been reported by many authors (e.g. [43, 113, 232, 233]. Many of the familial examples of TGA have either migraine comorbidity, or the episodes have occurred at the same time as a migraine (see Sect. 7.9 and Table 7.3). Some authors consider TGA to be simply a form of migraine aura ([234], p.125–30,168). TGA following mild head injury has been suggested to reflect "traumatic migraine" [235].

A syndrome of "acute confusional migraine" is recognised in children [236] which has been noted to have some features akin to TGA (e.g. [237–239]). Both may be examples of what I have ventured to term "cognitive migraine" [240] (see also discussion in [241]).

3.4.2 Adverse Drug Effect

A large number of pharmacological agents, used for both therapeutic and recreational purposes, have on occasion been associated with episodes of transient amnesia with features considered to be akin to those of TGA (Table 3.8).

There are problems with many of these reports. Many predate diagnostic criteria for TGA (e.g. [130, 268]), and/or present atypical clinical features, sometimes denoted as "TGA-like" episodes (e.g. [251, 269]). For example, reports of an association of TGA with marijuana ingestion include an episode of long duration [251] and a case involving a 6-year-old boy [252]. The description of some events labelled as TGA is not particularly convincing [256]. In some instances, it is not clear whether TGA or TEA is being described [257]. For example, a patient treated with intrathecal baclofen for generalised dystonia had events which "met criteria for transient global amnesia, but were unusual because of their frequent recurrence" [243], an observation that prompts concern about the possibility of an epileptic cause. In this context, a case reported by Zeman et al. is of note: following therapeutic infusion of baclofen, the patient developed short periods of global amnesia, accelerated long-term forgetting and persistent autobiographical amnesia, all features seen in TEA [270].

Some of the implicated medications, such as benzodiazepines [229, 246, 250, 268], are known to be associated with anterograde amnesia (e.g. [271, 272]). Indeed, this association was the stimulus, at least in part, which prompted Merriam (1988) to suggest that endogenous benzodiazepines might play a role in the pathogenesis of TGA [222]. Danek et al. explored the role of the benzodiazepine antagonist flumazenil in reversing TGA (n-of-1 trial), with inconclusive outcome [273].

Alcohol may also have been a confounding factor in some reports (e.g. [268]). The reported association with clioquinol use [244, 245] was excluded from the

Table 3.8: Reports of TGA occurring as an adverse drug effect (see text for caveats about some of these reports)

Drug	Reference(s)
Alprostadil (intracavernosal injection)	Maffei et al. (2020) [242]
Baclofen (intrathecal)	Grande et al. (2008) [243]
Clioquinol	Mumenthaler et al. (1979) [244] Kaeser (1984) [245]
Diazepam	Gilbert and Benson (1972) [229] Mazzucchi et al. (1980) [246]
Digitalis	Greenlee et al. (1975) [130]
Dimethylsulphoxide (DMSO)	Otrock et al. (2008) [247]
Ergots	Pradalier et al. (2000) [43] Gil-Martinez and Galiano (2004) [248]
Heparin	Teh et al. (2010) [249]
Lorazepam	Mazzucchi et al. (1980) [246] Sandyk (1985) [250]
Marijuana	Stracciari et al. (1999) [251] Shukla and Moore (2004) [252] Mansour et al. (2014) [253]
Midazolam	Otrock et al. (2008) [247]
Propafenone	Jones et al. (1995) [254]
Rofecoxib	Hirschfeld et al. (2007) [255]
Rosuvastatin	Healy et al. (2009) [256]
Sibutramine	Fu et al. (2010) [257]
Sildenafil (Viagra)	Savitz and Caplan (2002) [258] Gandolfo et al. (2003) [259] Shihman et al. (2006) [260] Marques-Vilallonga et al. (2014) [261] Finsterer (2019) [262] Lin et al. (2020) [263]
Sumatriptan (Imigran)	Pradalier et al. (2000) [43] Lee et al. (2021) [264]
Tadalafil	Schiefer and Sparing (2005) [265] Bardes et al. (2008) [266] Machado et al. (2010) [267]
Triazolam	Morris and Estes (1987) [268]
Zolpidem	Tsai et al. (2009) [269]

review by Caplan [274] as more likely to reflect a toxic encephalopathy (see Sect. 3.5.1), a conclusion also reached by Hodges ([5], p.10), but other authors seem to have accepted the association as causal ([275], p.84).

The use of opioid and non-opioid analgesia (hydromorphone, ketorolac) may have been a confounding factor in one report of pain-related TGA, although no signs of opioid intoxication were present [276].

The most frequent reports of drug-associated TGA relate to the use of phosphodiesterase type 5 (PDE-5) inhibitors, sildenafil (Viagra) [258–263] and tadalafil [265–267], used for the treatment of erectile dysfunction. Obviously, there is a

possible confounding factor here, namely sexual activity which is a reported precipitating factor for TGA (see Sect. 8.4). TGA has also been reported in association with the use of alprostadil (caverject) [242], a prostaglandin analogue administered by intracavernosal injection for erectile dysfunction. The British National Formulary (BNF) does not mention TGA as a side effect for PDE-5 inhibitors, although "memory loss" is listed amongst the "rare or very rare" side effects of tadalafil when used for pulmonary arterial hypertension or erectile dysfunction (bnf.nice.org.uk/drug/tadalafil.html#cautions; accessed 29/07/21).

Medications prescribed for migraine and associated with TGA might be incidental to migraine-related TGA attacks (Sect. 7.9). Such reports are extremely rare despite the high population prevalence of migraine. Pradalier et al. reported a case of TGA associated with the use of subcutaneous injection of sumatriptan and nasal dihydroergotamine [43]. Gil-Martinez and Galiano reported two cases associated with the use of ergotamine and dihydroergotamine, respectively [248]. Lee et al. reported TGA and myocardial infarction (NSTEMI) in a patient given oral sumatriptan [264], but pain might also be a confounder here (Sect. 8.5). Werner and Woehrle reported triptan overuse as comorbidity in one case in their series of TGA patients [11].

In summary, at best these are anecdotal accounts of a temporal association between drug use and TGA. Few reports include the typical magnetic resonance imaging changes seen in TGA (e.g. [255, 263]). There are, perhaps unsurprisingly, no rechallenge data attempting to corroborate a causal hypothesis. These accounts may therefore simply represent the chance concurrence of unrelated factors.

3.4.3 Hypoglycaemia

Profound hypoglycaemia is a recognised cause of acute amnesia [1]. It has on occasion been considered as a possible cause of TGA [228].

Relatively, few cases of amnesia related to hypoglycaemia in the context of diabetes mellitus and with longitudinal neuropsychological data have been reported (e.g. [277]). Some have evidence of hippocampal lesions on MR brain imaging [278], but these are confluent high signal changes on T2-weighted imaging which are unlike the punctate changes on MR-DWI seen in TGA. A patient seen by the author showed a focal deficit selective for anterograde memory and learning after acute severe hypoglycaemia, which gradually, though incompletely, reversed over a few months, prompting speculation about hippocampal vulnerability to the effects of neuroglycopaenia ([3], p.248 and [279]). Followed up more than 10 years later, the patient had developed an amnesic dementia, with evidence suggesting particular decline over a period of eight months during which he suffered multiple episodes of hypoglycaemia, followed by relative stability of cognition with improvement in glycaemic control. MR brain imaging showed global atrophy including the medial temporal lobes but little in the way of small vessel ischaemic change [280]. There were similarities with the patient reported by Kirchhoff et al. [281] who had multiple

hypoglycaemic episodes over many years and whose neuropsychological assessment showed anterograde amnesia; volumetric MR brain imaging showed atrophic change including loss of subcortical grey matter volume involving the hippocampus.

3.4.4 Traumatic Brain (Closed Head) Injury

Patients suffering a traumatic brain injury may present with confusion and memory loss after the injury. The duration of post-traumatic amnesia (PTA) is a marker of head injury severity and is also related to prognosis.

TGA is differentiated from the transient PTA which may follow mild traumatic brain injury (mTBI) secondary to closed head injury by the absence of head injury and the preservation of consciousness. The Hodges and Warlow TGA diagnostic criteria [22] (Table 2.1) list "recent head injury" as an exclusion criterion although "recent" is undefined. Hence, with the availability of a reliable eye-witness account from a capable observer who was present for most of the attack (an inclusion criterion for TGA [22]), there is usually no diagnostic or differential diagnostic issue.

However, in the absence of reliable collateral history, there may be difficulty differentiating PTA and TGA if there are no obvious stigmata of injury, since there is clinical overlap between these transient forms of amnesia. For example, a case of amnesia following mild head injury reported as early as 1835 by Koempfen [282], and cited by Ribot in his classic text on *Disorders of Memory* [283], may have given rise to the mistaken belief that the latter described TGA in the nineteenth century [284]. Miller Fisher's 1966 account of "concussion amnesia" also illustrates a dissociation between amnesia and impaired consciousness, with transient mnestic features akin to TGA (dense anterograde amnesia, retrograde amnesia, recovery within hours with a persistent memory gap) [285]. Although deficits of anterograde memory are documented in PTA, the repetitive questioning and behavioural changes in TGA are not seen, and there may be additional deficits in attentional and executive functions [286, 287]. Transient psychological amnesia (Sect. 3.3) must also enter the differential diagnosis of transient amnesia following a mild head injury [217].

Although cases of TGA triggered by mild head injury (e.g. [230, 235, 288–290]) and "post-traumatic" transient global amnesia have been reported [291], this may have been incidental, since mild head injury is not uncommon. All these reports predate Hodges and Warlow's criteria [22]. Some have atypical features, such as childhood onset (between age 6.5 and 14.5 years [290], or late teenage onset [288]). Nevertheless, Evans stated that "Rare sequelae of seemingly mild head injury include ... TGA" ([292], p.594) and "Mild head injury can rarely trigger TGA, which in children may actually be confusional migraine" ([292], p.597; see Sect. 3.4.1). However, in the context of head injury, the Hodges and Warlow criteria render the term "post-traumatic TGA" [291] an oxymoron.

One apparent exception is the case reported by Venneri et al. [293] in which the clinical and neuropsychological profile was said to be indistinguishable from TGA, albeit the patient's age (27 years) was atypical. Furthermore, the characteristic, although not specific, abnormality seen on diffusion-weighted magnetic resonance imaging in TGA, namely hyperintense lesion(s) in the hippocampal CA1 region, has also been observed in PTA [294].

3.4.5 Alcohol-Induced Amnesia; Korsakoff Syndrome

Acute alcohol intoxication may be associated with amnesia for events occurring during the period of inebriation, indeed currently this may possibly be the most common cause of transient amnesia, perhaps especially in young people. Islands of preserved memory may be reported, hence the description of this amnesia as of "fragmentary" type. Typically, there is no description of anterograde amnesia or repetitive questioning, so the differential diagnosis from TGA is seldom challenging. Confounding factors (recreational drug use, hypoglycaemia, head injury) may also contribute. Longer, "en bloc", alcoholic blackouts lasting days have also been described.

Korsakoff syndrome, associated with thiamine deficiency, which is often but not invariably a consequence of alcohol misuse [295, 296], may be associated with a chronic cognitive syndrome in which dense and persistent anterograde amnesia is prominent (first well described before Korsakoff, e.g. by Robert Lawson in 1878; see [297]). TGA has on occasion been labelled as an acute but transient Korsakoff's syndrome (e.g. [14, 87] and [298], p.38), but this is a misnomer.

3.4.6 Fatigue Amnesia

Cases of amnesia associated with extreme tiredness have been reported [299].

3.5 Other Causes of Acute Cerebral Disorder

Other acute cerebral disorders may sometimes be mistaken for TGA, even when amnesia is not a symptom. TIA is the most prominent example, but other disorders also enter the differential diagnosis (Table 3.2).

3.5.1 Acute Confusional State/Delirium/ Toxic-Metabolic Encephalopathy

Acute confusional state, or delirium, enters the differential diagnosis of TGA since, by definition, one of the phenotypic features of delirium is change in cognition which may include memory deficit (also disorientation, language impairment, perceptual disturbance) not better accounted for by dementia [300]. Impairment of consciousness, a *sine qua non* for the diagnosis of delirium, may be subtle. As infection, metabolic derangements, and adverse drug effects are the most commonly identified precipitating factors for an acute confusional state, the diagnostic label of toxic-metabolic encephalopathy is sometimes used. Some of the reported examples of TGA associated with medication use (Sect. 3.4.2 and Table 3.7) may in fact be examples of toxic-metabolic encephalopathy.

3.5.2 Acute Brain Infections, Including COVID-19

Benon, who described what may have been the earliest reported unequivocal case of TGA in 1909 [301] (see Sect. 1.2), also described amnesia in cases of syphilitic general paresis [302]. Only occasional cases of TGA associated with neurosyphilis have subsequently been reported [303], some ("TGA-like") with MR imaging changes in the limbic system [304].

Other brain infections which have on occasion been reported in association with TGA include encephalitis due to herpes simplex virus [305, 306] or Epstein–Barr virus [307], although the latter case was associated with partial and generalised epileptic seizures so would not fulfil TGA diagnostic criteria. Herpes simplex encephalitis (HSE) usually manifests with fever, headache, behavioural change and impairments of consciousness so should not be confused with TGA, although very occasionally presentation with isolated memory deficit has been reported [308]. Although cognitive recovery occurs in many patients with HSE, sometimes a dense amnesic syndrome may persist. Other, structural, lesions may sometimes masquerade as HSE [309].

Cases of TGA associated with the pandemic of COVID-19 (SARS-CoV-2) have been reported, associated with the infection per se [310], with infection-related acute stroke (suggested to be thrombotic events) [32], and triggered (possibly) by fear of contracting the infection [311]. Werner et al. reported an increased incidence of TGA seen in their hospital in Germany over a 3.5-month period at the beginning of 2020 which they suggested may be a consequence of the emotional stress occasioned by factors such as social distancing, uncertainty about the future and fear of becoming infected with COVID-19 [312].

3.6 Misdiagnosis

As may be evident from the foregoing sections, TGA has a potentially broad differential diagnosis. Hence, although clinical diagnosis is often straightforward in archetypal cases, misdiagnosis of TGA is not unexpected. In a study of 166 episodes of suspected TGA, Werner and Woehrle found an alternative diagnosis or severe comorbidity impacting the occurrence of the amnestic episode in 10.8%. The most important differential diagnosis was stroke [11]. In the author's cohort, in 23/73 (32%) patients referred to the clinic with suspected TGA, the diagnosis could not be sustained, either because of insufficient clinical evidence (most often the absence of a reliable witness report of the episode) or because an alternative diagnosis was established (see Figure 7.3).

3.7 Summary and Recommendations

The relatively stereotyped clinical features of TGA may render diagnosis straightforward, especially for those clinicians who are familiar with the condition. Nevertheless, acute amnesic episodes have a potentially extensive differential diagnosis, including disorders of cerebrovascular, epileptic, psychological, metabolic, infective, toxic and structural origin. For this reason, investigations may sometimes be required to assist with the diagnosis and differential diagnosis of TGA, and these are elaborated on the next two chapters.

References

1. Fisher CM. Unexplained sudden amnesia. Arch Neurol. 2002;59:1310–3.
2. Papanicolaou AC. The amnesias: a clinical textbook of memory disorders. Oxford: Oxford University Press; 2006.
3. Larner AJ. Dementia in clinical practice: a neurological perspective. In: Pragmatic studies in the cognitive function clinic. 3rd ed. London: Springer; 2018.
4. Larner AJ. Transient global amnesia in the district general hospital. Int J Clin Pract. 2007;61:255–8.
5. Hodges JR. Transient amnesia. Clinical and neuropsychological aspects. London: WB Saunders; 1991.
6. Williamson J, Larner AJ. Transient global amnesia. Br J Hosp Med. 2015;76:C186-8.
7. Guyotat MM, Courjon J. Les ictus amnésiques. J Med Lyon. 1956;37:697–701.
8. Poser CM, Ziegler DK. Temporary amnesia as a manifestation of cerebrovascular insufficiency. Trans Am Neurol Assoc. 1960;85:221–3.
9. Halsey JH Jr. Cerebral infarction with transient global amnesia. Ala J Med Sci. 1967;4:436–8.
10. Fisher CM, Adams RD. Transient global amnesia. Acta Neurol Scand. 1964;40(Suppl9):1–81.
11. Werner R, Woehrle JC. Prevalence of mimics and severe comorbidity in patients with clinically suspected transient global amnesia. Cerebrovasc Dis. 2021;50:171–7.

12. Papez JW. A proposed mechanism of emotion. Arch Neurol Psychiatry. 1937;38:725–43. [Reprinted in J Neuropsychiatry Clin Neurosci. 1995;7:103-12.]
13. Lou H. Repeated episodes of transient global amnesia. Acta Neurol Scand. 1968;44:612–8.
14. de Tribolet N, Assal G, Oberson R. Korsakoff's syndrome and transitory cortical blindness following vertebral angiography [in German]. Schweiz Med Wochenschr. 1975;105:1506–9.
15. Benson DF, Marsden CD, Meadows JC. The amnesic syndrome of posterior cerebral artery occlusion. Acta Neurol Scand. 1974;50:133–45.
16. Howard RS, Festenstein R, Mellers J, Kartsounis LD, Ron M. Transient amnesia heralding brain stem infarction. J Neurol Neurosurg Psychiatry. 1992;55:977.
17. Taylor RA, Wu GF, Hurst RW, Kasner SE, Cucchiara BL. Transient global amnesia heralding basilar artery thrombosis. Clin Neurol Neurosurg. 2005;108:60–2.
18. Carota A, Lysandropoulos AP, Calabrese P. Pure left hippocampal stroke: a transient global amnesia-plus syndrome. J Neurol. 2012;259:989–92.
19. Beyrouti R, Mansour M, Kacem A, Zaouali J, Mrissa R. Transient global amnesia like syndrome associated with acute infarction of the corpus callosum: a case report. Acta Neurol Belg. 2016;116:375–7.
20. Michel P, Beaud V, Eskandari A, Maeder P, Demonet JF, Eskioglou E. Ischemic amnesia: causes and outcome. Stroke. 2017;48:2270–3.
21. Ott BR, Saver JL. Unilateral amnesic stroke. Six new cases and a review of the literature. Stroke. 1993;24:1033–42.
22. Hodges JR, Warlow CP. Syndromes of transient amnesia: towards a classification. A study of 153 cases. J Neurol Neurosurg Psychiatry. 1990;53:834–43.
23. Chen ST, Tang LM, Lee TH, Ro LS, Lyu RK. Transient global amnesia and amaurosis fugax in a patient with common carotid artery occlusion—a case report. Angiology. 2000;51:257–61.
24. Bogousslavsky J, Regli F. Transient global amnesia and stroke. Eur Neurol. 1988;28:106–10.
25. Gorelick PB, Amico LL, Ganellen R, Benevento LA. Transient global amnesia and thalamic infarction. Neurology. 1988;38:496–9.
26. Raffaele R, Tornali C, Genazzani AA, Vecchio I, Rampello L. Transient global amnesia and cerebral infarct: a case report. Brain Inj. 1995;9:815–8.
27. Larner AJ. Stroke as a cause of TGA? Narrative review and hypothesis. J Neurol Disord Stroke. 2022;9(1):1189.
28. Greer DM, Schaefer PW, Schwamm LH. Unilateral temporal lobe stroke causing ischemic transient global amnesia: role for diffusion-weighted imaging in the initial evaluation. J Neuroimaging. 2001;11:317–9.
29. López-Pesquera B, Fratalia-Bax L, Piera-Balbastre A, Badia-Picazo MC, Chamarro LR. Amnesia global transitoria. No siempre un proceso benigno. Rev Neurol. 2005;41:57. (Poster P7)
30. Graff-Radford J, Clapp AJ, Lanzino G, Rabinstein AA. Transient amnesia after coiling of a posterior circulation aneurysm. Neurocrit Care. 2013;18:245–7.
31. 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.
32. Ramanathan RS, Wachsman A. Coronavirus disease-19 (COVID-19) related acute stroke causing transient global amnesia. J Stroke Cerebrovasc Dis. 2021;30:105738.
33. Adler AC, Warum D, Sapire JM. Transient global amnesia caused by hippocampal infarct: case report and review of literature. Clin Imaging. 2012;36:584–6.
34. Gungor-Tuncer O, Okudan Z, Aksay-Koyuncu B, Altindag E, Tolun R, Krespi Y. Posterior circulation stroke: an underdiagnosed situation in transient global amnesia. J Neurol. 2012;259(Suppl1):S115. (abstract P484)
35. Gungor Tuncer O, Aksay Koyuncu B, Vildan Okudan Z, Altindag E, Tolun R, Krespi Y. Vascular ischemia as a cause of transient global amnesia: a patient series. Noro Psikiyatr Ars. 2015;52:59–63.
36. Li J, Hu WL. Bilateral hippocampal abnormalities in magnetic resonance imaging in transient global amnesia. Am J Emerg Med. 2013;31:755e1–3.

37. Naldi F, Baiardi S, Guarino M, Spinardi L, Cirignotta F, Stracciari A. Posterior hippocampal stroke presenting with transient global amnesia. Neurocase. 2016;23:22–5.
38. Yun U, Hwang I, Ha SW. Transient global amnesia caused by bilateral medial temporal-lobe infarction. Dement Neurocogn Disord. 2017;16:132–3.
39. Kang MK, Kim SY, Kang HG, Shin BS, Lee CH. Transient global amnesia caused by cryptogenic ischemic stroke. Interdiscip Neurosurg. 2021;23:100911.
40. Sakihara E, Ajisaka K, Takeoka H, Suzuyama H, Nabeshima S. A case of transient global amnesia with hippocampal infarction due to infective endocarditis. J Infect Chemother. 2021;27:902–5.
41. Gupta M, Kantor MA, Tung CE, Zhang N, Albers GW. Transient global amnesia associated with a unilateral infarction of the fornix: case report and review of the literature. Front Neurol. 2015;5:291.
42. Meyer MA. Neurologic disease. A modern pathophysiologic approach to diagnosis and treatment. London: Springer; 2016.
43. Pradalier A, Lutz G, Vincent D. Transient global amnesia, migraine, thalamic infarct, dihydroergotamine, and sumatriptan. Headache. 2000;40:324–7.
44. Giannantoni NM, Lacidogna G, Broccolini A, et al. Thalamic amnesia mimicking transient global amnesia. Neurologist. 2015;19:149–52.
45. Dogan VB, Dogan GB, Turkmen U, Yayla VA. A case of thalamo-mesencephalon infarct presenting as transient global amnesia: do we overlook the diagnosis? Neurol Sci. 2017;38:909–11.
46. Gallardo-Tur A, Romero-Godoy J, de la Cruz Cosme C, Arboix A. Transient global amnesia associated with an acute infarction at the cingulate gyrus. Case Rep Neurol Med. 2014;2014:418180.
47. Chau L, Liu A. Transient global amnesia as the sole presentation of an acute stroke in the left cingulate gyrus. Case Rep Neurol Med. 2019;2019:4810629.
48. Meng D, Alsaeed M, Randhawa J, Chen T. Retrosplenial stroke mimicking transient global amnesia. Can J Neurol Sci. 2021:48:884–5.
49. Ravindran V, Jain S, Ming A, Bartlett RJ. Transient global amnesia in a patient with acute unilateral caudate nucleus ischemia. J Clin Neurosci. 2004;11:669–72.
50. Kim HJ, Kim H, Lim SM, Moon WJ, Han SH. An acute tiny left putamenal lesion presenting with transient global amnesia. Neurologist. 2012;18:80–2.
51. Koltermann T, Vitali da Silva A, Pires Lazaro Fay Neves M, Parizotto C, da Silva J, Batista de Rezende Filho O. Case report: caudate stroke simulating transient global amnesia. J Neurol Sci. 2015;357(Suppl1):e387–8.
52. Yoshida K. A case of transient global amnesia with small left putamen infarction. J Stroke Cerebrovasc Dis. 2017;26:e27-8.
53. Tarazona LR, Martinez EL, Llopis CM. Transient global amnesia with extra-hippocampal lesion and a normal cardiovascular study. Can J Neurol Sci. 2021;24:1–2. https://doi.org/10.1017/cjn.2021.116. Online ahead of print.
54. Saito K, Kimura K, Minematsu K, Shiraishi A, Nakajima M. Transient global amnesia associated with an acute infarction in the retrosplenium of the corpus callosum. J Neurol Sci. 2003;210:95–7.
55. Kim J, Sohn MJ, Choi SW. Acute ischemic strokes presenting as transient global amnesia. Eur J Neurol. 2018;25(Suppl2):106. (abstract EPO1032)
56. Chatham PE, Brillman J. Transient global amnesia associated with bilateral subdural hematomas. Neurosurgery. 1985;17:971–3.
57. Heon M, Reiher D, Dilenge D, Lamarche J. Ictus amnésique et hématome intraventriculaire. Neuro-chirurgie (Paris). 1972;18:503–10.
58. Honma Y, Nagao S. Hemorrhagic pituitary adenoma manifesting as transient global amnesia. Neurol Med Chir (Tokyo). 1996;36:234–6.
59. Jacome DE, Yanez GF. Transient global amnesia and left frontal haemorrhage. Postgrad Med J. 1988;64:137–9.

60. Landi G, Giusti MC, Guidotti M. Transient global amnesia due to left temporal haemorrhage. J Neurol Neurosurg Psychiatry. 1982;45:1062–3.
61. 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.
62. Moonis M, Jain S, Prasad K, Mishra NK, Goulatia RK, Maheshwari MC. Left thalamic hypertensive haemorrhage presenting as transient global amnesia. Acta Neurol Scand. 1988;77:331–4.
63. Sandyk R. Transient global amnesia: a presentation of subarachnoid haemorrhage. J Neurol. 1984;231:283–4.
64. Sorenson EJ, Silbert PL, Benarroch EE, Jack CR, Parisi JE. Transient amnesic syndrome after spontaneous haemorrhage into a hypothalamic pilocytic astrocytoma. J Neurol Neurosurg Psychiatry. 1995;58:761–3.
65. Yoon B, Yoo JY, Shim YS, Lee KS, Kim JS. Transient global amnesia associated with acute intracerebral haemorrhage at the cingulate gyrus. Eur Neurol. 2006;56:54–6.
66. Wilson M, Doran M, Enevoldson TP, Larner AJ. Cognitive profiles associated with intracranial dural arteriovenous fistula. Age Ageing. 2010;39:389–92.
67. Randall A, Ellis R, Hywel B, Davies RR, Alusi SH, Larner AJ. Rapid cognitive decline: not always Creutzfeldt-Jakob disease. J R Coll Physicians Edinb. 2015;45:209–12.
68. Johnson P, Ghodke B, Khot S. Dural arteriovenous fistula causing recurrent transient global amnesia. Neurohospitalist. 2018;8:200.
69. Takahashi Y, Yamamoto T, Abe T, et al. Transient global amnesia and dural arteriovenous fistula of the anterior cranial fossa. Kurume Med J. 1996;43:223–9.
70. Heine P, Degos JD, Meyrignac C. Cerebral angioma disclosed by 2 episodes of transient global amnesia [in French]. Presse Med. 1986;15:1049.
71. Daniel BT. Transient global amnesia. Print version and ebook: Amazon; 2012.
72. Boitet R, Gaillard N, Bendiab E, et al. Concomitant reversible cerebral vasoconstriction syndrome and transient global amnesia. J Neurol. 2020;267:390–4.
73. Ishaya K, Shinohara K, Akamatu M, et al. Reversible cerebral vasoconstriction syndrome presenting with transient global amnesia. Intern Med. 2017;56:1569–73.
74. Kamm K, Schöberl F, Grabova D, Straube A, Zwergal A. RCVS and TGA: a common pathophysiology? J Neurol. 2019;266:2872–4.
75. Kim J, Jung YI, Seo J, Lee H, Sunwoo MK. A case of posterior reversible encephalopathy syndrome with similar symptoms as transient global amnesia. Dement Neurocogn Disord. 2018;17:176–8.
76. Nakamizo T, Tsuzuki I, Koide T. Transient global amnesia with reversible white matter lesions: a variant of posterior reversible encephalopathy syndrome? Case Rep Neurol Med. 2015;2015:541328.
77. Rumpl E, Rumpl H. Recurrent transient global amnesia in a case with cerebrovascular lesions and livedo reticularis (Sneddon syndrome). J Neurol. 1979;221:127–31.
78. Nishida A, Kaiya H, Uematsu M, Maeda M, Mori S, Wakabayashi S. Transient global amnesia and Raynaud's phenomenon in scleroderma. Acta Neurol Scand. 1990;81:550–2.
79. Pradotto L, Orsi L, Mencarelli M, et al. Recurrent transient global amnesia as presenting symptoms of CADASIL. Clin Case Rep. 2016;4:1045–8.
80. Garcia-Carrasco M, Galarza C, Gomez-Ponce M, et al. Antiphospholipid syndrome in Latin American patients: clinical and immunologic characteristics and comparison with European patients. Lupus. 2007;16:366–73.
81. Ortego-Centeno N, Callejas-Rubio JL, Fernandez MG, Carmello MG. Transient global amnesia in a patient with high and persistent levels of antiphospholipid antibodies. Clin Rheumatol. 2006;25:407–8.
82. Cervera R, Piette JC, Font J, et al. Antiphospholipid syndrome: clinical and immunologic manifestations and patterns of disease expression in a cohort of 1,000 patients. Arthritis Rheum. 2002;46:1019–27.

83. Attarian S, Michel B, Delaforte C, Chave B, Gastaut JL. A case of transient amnesia caused by cerebral thrombophlebitis: contribution of neuroimaging to physiopathogenesis of transient amnesia [in French]. Rev Neurol (Paris). 1995;151:552–8.
84. Sharma RC, Kainth A, Sharma S. Transient global amnesia as a presenting manifestation of cerebral venous thrombosis. J Neuropsychiatry Clin Neurosci. 2015;27:e209–10.
85. Ito AO, Tamura A, Niwa A, et al. Recurrent transient global amnesia associated with internal jugular vein thrombosis. J Neurol Sci. 2019;402:108–10.
86. Hauge T. Catheter vertebral angiography. Acta Radiologica Suppl. 1954;109:1–219.
87. Deak G, Toth S. Vertebral angiography followed by transient Korsakoff's syndrome [in Hungarian]. Ideggyogy Sz. 1964;17:119–25.
88. Whishart DL. Complications of cerebral angiography as compared to non-vertebral cerebral angiography in 447 studies. AJR Am J Roentgenol. 1971;113:527–37.
89. Wales LR, Nov AA. Transient global amnesia: complication of cerebral angiography. AJN R Am J Neuroradiol. 1981;2:275–7.
90. Cochran JW, Morrell F, Huckman MS, Cochran EJ. Transient global amnesia after cerebral angiography. Report of seven cases. Arch Neurol. 1982;39:593–4.
91. Haas DC. Transient global amnesia after cerebral angiography. Arch Neurol. 1983;40:258–9.
92. Pexman JH, Coates RK. Amnesia after femorocerebral angiography. AJNR Am J Neuroradiol. 1983;4:979–83.
93. Giang DW, Kido DK. Transient global amnesia associated with cerebral angiography performed with use of iopamidol. Radiology. 1989;172:195–6.
94. Minuk J, Melancon D, Tampieri D, Ethier R. Transient global amnesia associated with cerebral angiography performed with use of iopamidol. Radiology. 1990;174:285–6.
95. Juni J, Morera J, Lainez JM, Escudero J, Ferrer C, Sancho J. Transient global amnesia after cerebral angiography with iohexol. Neuroradiology. 1992;34:141–3.
96. Brady AP, Hough DM, Lo R, Gill G. Transient global amnesia after cerebral angiography with iohexol. Can Assoc Radiol J. 1993;44:450–2.
97. Schamschula RG, Soo MY. Transient global amnesia following cerebral angiography with non-ionic contrast medium. Australas Radiol. 1994;38:196–8.
98. Jackson A, Stewart G, Wood A, Gillespie JE. Transient global amnesia and cortical blindness after vertebral angiography: further evidence for the role of arterial spasm. AJNR Am J Neuroradiol. 1995;16(4Suppl):955–9.
99. Meder JF, Mourey-Gerosa I, Blustain J, Lemaignen H, Devaux B, Fredy D. Transient global amnesia after cerebral angiography. A case report. Acta Radiol. 1997;38:273–4.
100. Woolfenden AR, O'Brien MW, Schwartzberg RE, Norbash AM, Tong DC. Diffusion-weighted MRI in transient global amnesia precipitated by cerebral angiography. Stroke. 1997;28:2311–4.
101. Kapur N, Millar J, Abbott P, Carter M. Recovery of function processes in human amnesia: evidence from transient global amnesia. Neuropsychologia. 1998;36:99–107.
102. Tanabe M, Watanabe T, Ishibashi M, Hirano M, Tabuchi S, Takigawa H. Hippocampal ischemia in a patient who experienced transient global amnesia after undergoing cerebral angiography. Case illustration. J Neurosurg. 1999;91:347.
103. Kim HY, Kang HS, Roh HG, Oh J, Lee IK, Han SH. Transient global amnesia following vertebral artery angioplasty and stenting. Eur Neurol. 2006;56:133–5.
104. Foss-Skiftesvik J, Snoer AH, Wagner A, Hauerberg J. Transient global amnesia after cerebral angiography still occurs: case report and literature review. Radiol Case Rep. 2015;9:988.
105. Tiu C, Terecoasa EO, Grecu N, Dorobat B, Marinescu AN, Bajenaru OA. Transient global amnesia after cerebral angiography with iomeprol: a case report. Medicine (Baltimore). 2016;95:e3950.
106. Lee BH. Transient global amnesia following carotid artery stenting: a case report. Radiol Case Rep. 2020;15:1159–63.
107. Fischer-Williams M, Gottschalk PG, Browell JN. Transient cortical blindness. An unusual complication of coronary angiography. Neurology. 1970;29:353–5.

108. Shuttleworth EC, Wise GR. Transient global amnesia due to arterial embolism. Arch Neurol. 1973;29:340–2.
109. Lockwood K, Capraro J, Hanson M, Conomy J. Neurologic complications of cardiac catheterization. Neurology. 1983;33(Suppl2):143.
110. Koehler PJ, Endtz LJ, den Bakker PB. Transient global amnesia after coronary angiography. Clin Cardiol. 1986;9:170–1.
111. Yildiz A, Yencilek E, Apaydin FD, Duce MN, Ozer C, Atalay A. Transient partial amnesia complicating cardiac and peripheral arteriography with nonionic contrast medium. Eur Radiol. 2003;13(Suppl4):L113-5.
112. Kurokawa Y, Ishizaki E, Kihara H, Inaba K. Two cases of transient global amnesia (TGA) immediately after coronary angiography: pathogenesis of the primary TGA [in Japanese]. No To Shinkei. 2004;56:69–74.
113. 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.
114. Wong E, Patel A, Javasinghe R. Transient global amnesia following coronary angiography and angioplasty. Intern Med J. 2005;37:435–6.
115. Udyavar AR, D'souza RC, Gadkar N, Rajani RM. Transient global amnesia following coronary angiography. J Postgrad Med. 2006;52:70–1.
116. Rosenberg GA. Transient global amnesia with a dissecting aortic aneurysm. Arch Neurol. 1979;36:255.
117. Gaul C, Dietrich W, Tomandi B, Neundörfer B, Erbguth FJ. Aortic dissection presenting with transient global amnesia-like symptoms. Neurology. 2004;63:2442–3.
118. Mondon K, Blechet C, Gochard A, et al. Transient global amnesia caused by painless aortic dissection. Emerg Med J. 2007;24:63–4.
119. Irioka T, Yamanami A, Yaqi Y, Mizusawa H. Aortic dissection as a possible cause of pure transient global amnesia: a case report and literature review. Neurol Sci. 2009;30:255–8.
120. Colotto M, Maranghi M, Epifania A, Totaro M, Giura R, Durante C. Unmasking aortic dissection in patients of [sic] transient global amnesia: case report and diagnostic algorithm for the emergency department. BMJ Case Rep. 2011;2011:pii:bcr0720103151.
121. Kaveeshvar H, Kashouty R, Loomba V, Yono N. A rare case of aortic dissection presenting as pure transient global amnesia. Cardiovasc J Afr. 2015;26:e8–9.
122. Michel D, Garnier P, Schneider F, Poujois A, Barral FG, Thomas-Antérion C. Diffusion MRI in pure transient global amnesia associated with bilateral vertebral artery dissection. Cerebrovasc Dis. 2004;17:264–6.
123. Yokota H, Yokoyama K, Iwasaki S. Transient global amnesia with intracranial vertebral artery dissection and hippocampal CA1 lesion. Neurol India. 2015;63:604–5.
124. Renault P, Rouchet S. Transient global amnesia and transient cortical blindness secondary to contrast induced encephalopathy after renal artery angiography. Rev Neurol (Paris). 2019;175:335–6.
125. Hankey GJ. Recurrent migraine aura triggered by coronary angiography. Pract Neurol. 2004;4:308–9.
126. Agosti C, Borroni B, Akkawi N, Bordonali T, Padovani A. Acute myocardial infarction presenting with transient global amnesia spectrum. J Am Geriatr Soc. 2006;54:1004.
127. Caramelli B, Dutra AP, Calderaro D, Yu PC, Gualandro DM, Marques AC. Transient global amnesia as a manifestation of acute myocardial infarction: a case of missed sudden cardiac death? Int J Cardiol. 2009;136:e14–5.
128. Courand PY, Sibellas F, Gonidec S, Mechtouff L, Kirkorian G, Bonnefoy E. Acute myocardial infarction: a precipitating event for transient global amnesia. J Cardiovasc Med (Hagerstown). 2014;15:78–9.
129. Harase S, Araki K, Kobayashi T, Katada F, Fukutake T. A case of transient global amnesia associated with painless myocardial infarction [in Japanese]. Rinsho Shinkeigaku. 2021;61:136–9.

130. Greenlee JE, Crampton RS, Miller JQ. Transient global amnesia associated with cardiac arrhythmia and digitalis intoxication. Stroke. 1975;6:513–6.
131. Ullrich NJ, Urion DK. Transient global amnesia in a young adult with cyanotic heart disease. Pediatr Neurol. 2003;29:334–6.
132. Jackson AC. Neurologic disorders associated with mitral valve prolapse. Can J Neurol Sci. 1986;13:15–20.
133. Bobinger T, Köhrmann M, Raaz-Schrauder D, Schwab S, Kallmünzer B. Lost memories can break your heart: a case report of transient global amnesia followed by takotsubo cardiomyopathy. Clin Res Cardiol. 2013;102:693–6.
134. Eisele P, Baumann S, Noor L, et al. Interaction between the heart and the brain in transient global amnesia. J Neurol. 2019;266:3048–57.
135. Finsterer J, Stöllberger C. Simultaneous transient global amnesia and Takotsubo syndrome after death of a relative: a case report. J Med Case Rep. 2019;13(1):22.
136. Grautoff S, Sitzer M, Weitkamp P, Kähler J. Transient global amnesia and Tako-Tsubo cardiomyopathy—coincidence or corollary? [in German]. Dtsch Med Wochenschr. 2012;137:2256–9.
137. Petrea RE, Nguyen T, Pikula A, Wilkenfeld A, Nedeljkovic Z, Kase CS. Transient global amnesia heralding a broken-heart syndrome. Eur J Neurol. 2008;15(Suppl3):212–3. (abstract P1718)
138. Pyle LM, Laghari FJ, Kinem DJ. Concomitant transient global amnesia and takotsubo cardiomyopathy following a stressful event. Clin Auton Res. 2018;28:597–8.
139. Quick S, Speiser U, Richter N, et al. Transient global amnesia and broken heart syndrome: two faces of one pathology. Clin Auton Res. 2015;25:189–91.
140. Sajeev J, Koshy A, Rajakariar K, Gordon G. Takotsubo cardiomyopathy and transient global amnesia: a shared aetiology. BMJ Case Rep. 2017;2017:bcr2017219472.
141. Stöllberger C, DeCilla N, Finsterer J. Tako-tsubo cardiomyopathy with transient global amnesia and cerebellar embolic stroke triggered by existential fear. Neurol Neurochir Pol. 2018;52:394–6.
142. Tso M, Tam JW, Khoo C. An atypical case of Takotsubo cardiomyopathy and transient global amnesia. CJC Open. 2019;1:35–8.
143. Pelliccia F, Parodi G, Greco C, et al. Comorbidities frequency in Takotsubo syndrome: an international collaborative systematic review including 1109 patients. Am J Med. 2015;128(654):e11–9.
144. Finsterer J, Stöllberger C. Transient global amnesia: the cerebral Takotsubo? J Neurol Sci. 2017;376:196–7.
145. Erdur H, Siegerink B, Ganeshan R, et al. Myocardial injury in transient global amnesia: a case-control study. Eur J Neurol. 2019;26:986–91.
146. Jaguszewski M, Osipova J, Ghadri JR, et al. A signature of circulating microRNAs differentiates Takotsubo cardiomyopathy from acute myocardial infarction. Eur Heart J. 2014;35:999–1006.
147. Klötzsch C, Sliwka U, Berlit P, Noth J. An increased frequency of patent foramen ovale in patients with transient global amnesia. Analysis of 53 consecutive patients. Arch Neurol. 1996;53:504–8.
148. Profice P, Rizzello V, Pennestri F, et al. Transient global amnesia during transoesophageal echocardiogram. Neurol Sci. 2008;29:477–9.
149. Maalikjy Akkawi N, Agosti C, Anzola GP, et al. Transient global amnesia: a clinical and sonographic study. Eur Neurol. 2003;49:67–71.
150. 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.
151. Spigno F, De Lucchi M, Migliazzi L, Cocito L. Transient global amnesia after breathing hyperoxic mixtures in otherwise regular dives. Clin Neurol Neurosurg. 2008;110:259–61.
152. Liu K, Wang BZ, Hao Y, Song S, Pan M. The correlation between migraine and patent foramen ovale. Front Neurol. 2020;11:543485.

153. Deisenhammer E. Transient global amnesia as an epileptic manifestation. J Neurol. 1981;225:289–92.
154. Gilbert GJ. Transient global amnesia: manifestation of medial temporal lobe epilepsy. Clin Electroencephalogr. 1978;9:147–52.
155. Melo TP, Ferro JM, Paiva T. Are brief or recurrent transient global amnesias of epileptic origin? J Neurol Neurosurg Psychiatry. 1994;57:622–5.
156. Meo R, Bilo L, Striano S, Ruosi P, Estraneo A, Nocerino C. Transient global amnesia of epileptic origin accompanied by fever. Seizure. 1995;4:311–7.
157. Tharp BR. Transient global amnesia: manifestation of medial temporal lobe epilepsy. Clin Electroencephalogr. 1979;10:54–6.
158. Fisher CM. Transient global amnesia. Precipitating activities and other observations. Arch Neurol. 1982;39:605–8.
159. Caplan LR. C. Miller Fisher. Stroke in the 20th century. New York: Oxford University Press; 2020.
160. Fisher CM. Ataxic hemiparesis. A pathologic study. Arch Neurol. 1978;35:126–8.
161. Fisher CM. Pure sensory stroke and allied conditions. Stroke. 1982;13:434–47.
162. Werring D, Greenberg SM, Gill S. Foundations of modern stroke medicine. The legacy of C Miller Fisher. Adv Clin Neurosci Rehabil. 2015;15(5):18–9.
163. Jackson JH. On a particular variety of epilepsy (intellectual aura). One case with symptoms of organic brain disease. Brain. 1888;11:179–207.
164. Gallassi R, Morreale A, Lorusso S, Pazzaglia P, Lugaresi E. Epileptic transient amnesia. Ital J Neurol Sci. 1988;(Suppl 9):37–9.
165. Gallassi R, Morreale A, Di Sarro R, Lugaresi E. Epileptic amnesic syndrome. Epilepsia. 1992;33(suppl6):S21–5.
166. Palmini AL, Gloor P, Jones-Gotman M. Pure amnestic seizures in temporal lobe epilepsy. Definition, clinical symptomatology and functional anatomical considerations. Brain. 1992;115:749–69.
167. Pritchard PB III, Holmstrom VL, Roitzsch JC, Giacinto J. Epileptic amnesic attacks: benefit from antiepileptic drugs. Neurology. 1985;35:1188–9.
168. Rowan AJ, Rosenbaum DH. Ictal amnesia and fugue states. Adv Neurol. 1991;55:357–67.
169. Stracciari A, Ciucci G, Bianchedi G, Rebucci GG. Epileptic transient amnesia. Eur Neurol. 1990;30:176–9.
170. Kapur N. Transient epileptic amnesia: a clinical update and a reformulation. J Neurol Neurosurg Psychiatry. 1993;56:1184–90.
171. Zeman AZJ, Boniface SJ, Hodges JR. Transient epileptic amnesia: a description of the clinical and neuropsychological features in 10 cases and a review of the literature. J Neurol Neurosurg Psychiatry. 1998;64:435–43.
172. Asadi-Pooya AA. Transient epileptic amnesia: a concise review. Epilepsy Behav. 2014;31:243–5.
173. Baker J, Savage S, Milton F, et al. The syndrome of transient epileptic amnesia: a combined series of 115 cases and literature review. Brain Commun. 2021;3:fcab038.
174. Bartsch T, Butler C. Transient amnesic syndromes. Nat Rev Neurol. 2013;9:86–97.
175. Butler C. Transient epileptic amnesia. Pract Neurol. 2006;6:368–71.
176. Butler CR, Zeman AZ. Recent insights into the impairment of memory in epilepsy: transient epileptic amnesia, accelerated long-term forgetting and remote memory impairment. Brain. 2008;131:2243–63.
177. Butler CR, Zeman A. The causes and consequences of transient epileptic amnesia. Behav Neurol. 2011;24:299–305.
178. Butler CR, Graham KS, Hodges JR, Kapur N, Wardlaw JM, Zeman AZ. The syndrome of transient epileptic amnesia. Ann Neurol. 2007;61:587–98.
179. Ramanan VK, Morris KA, Graff-Radford J, et al. Transient epileptic amnesia: a treatable cause of spells associated with persistent cognitive symptoms. Front Neurol. 2019;10:939.

180. Zeman A, Butler C, Hodges J, Kapur N. The syndrome of transient epileptic amnesia. In: Zeman A, Kapur N, Jones-Gotman M, editors. Epilepsy and memory. Oxford: Oxford University Press; 2012. p. 139–59.
181. Manes F, Hodges JR, Graham KS, et al. Focal autobiographical amnesia in association with transient epileptic amnesia. Brain. 2001;124:499–509.
182. Manes F, Graham KS, Zeman A, de Luján Calcagno M, Hodges JR. Autobiographical amnesia and accelerated forgetting in transient epileptic amnesia. J Neurol Neurosurg Psychiatry. 2005;76:1387–91.
183. Blake R, Wroe S, Breen E, McCarthy R. Accelerated forgetting in patients with epilepsy: evidence for impairment in memory consolidation. Brain. 2000;123:472–83.
184. McGinty RN, Larner AJ. Transient epileptic amnesia and pathological tearfulness. Cortex. 2022;147:206–8.
185. Felician O, Tramoni E, Barbeau E, et al. Isolated autobiographical amnesia: a neurological basis? [in French]. Rev Neurol (Paris). 2009;165:449–59.
186. Kapur N. Focal retrograde amnesia in neurological disease: a critical review. Cortex. 1993;29:217–34.
187. Larner AJ, Ghadiali EJ, Doran M. Focal retrograde amnesia: clinical, neuropsychological and neuroimaging study. Neurobiol Aging. 2004;25(S2):S128. (abstract P1-116)
188. Stuss DT, Guzman DA. Severe remote memory loss with minimal anterograde amnesia: a clinical note. Brain Cogn. 1988;8:21–30.
189. Lozsadi DA, Chadwick DW, Larner AJ. Late-onset temporal lobe epilepsy with unilateral mesial temporal sclerosis and cognitive decline; a diagnostic dilemma. Seizure. 2008;17:473–6.
190. Butler C, van Erp W, Bhaduri A, Hammers A, Heckemann R, Zeman A. Magnetic resonance volumetry reveals focal brain atrophy in transient epileptic amnesia. Epilepsy Behav. 2013;28:363–9.
191. Kanbayahsi T, Hatanaka Y, Sonoo M. Transient epileptic amnesia with amygdala enlargement. Neurol Sci. 2020;41:1591–3.
192. Larner AJ. Transient epileptic amnesia and amygdala enlargement revisited. Psychogeriatrics. 2021;21:943–4.
193. Takeda M, Kasama S, Watanabe S, Kimura T, Yoshikawa H. Transient epileptic amnesia in a temporal lobe epilepsy patient with amygdala enlargement: a case study. Psychogeriatrics. 2020;20:235–6.
194. 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.
195. Krishnan K, Larner AJ. Concurrent onset of transient epileptic amnesia and Alzheimer's disease. Eur J Neurol. 2009;16(Suppl 3):468. (abstract 2386)
196. Lozsadi DA, Larner AJ. Prevalence and causes of seizures at time of diagnosis of probable Alzheimer's disease. Dement Geriatr Cogn Disord. 2006;22:121–4.
197. Adan G, Mitchell JW, Ziso B, Larner AJ. Diagnosis and management of seizures in neurodegenerative diseases. Curr Treat Options Neurol. 2021;23:1.
198. Larner AJ. Epileptic seizures in AD patients. Neuromolecular Med. 2010;12:71–7.
199. Larner AJ. Something in common: Alzheimer's disease and epilepsy. EP Epilepsy Prof. 2011;21:12–5.
200. Powell G, Ziso B, Larner AJ. The overlap between epilepsy and Alzheimer's disease and the consequences for treatment. Expert Rev Neurother. 2019;19:653–61.
201. Rabinowicz AL, Starkstein SE, Leiguarda RC, Coleman AE. Transient epileptic amnesia in dementia: a treatable unrecognized cause of episodic amnestic wandering. Alzheimer Dis Assoc Disord. 2000;14:231–3.
202. Huang CF, Pai MC. Transient amnesia in a patient with left temporal tumor. Symptomatic transient global amnesia or an epileptic amnesia? Neurologist. 2008;14:196–200.

203. Savage SA, Irani SR, Leite MI, Zeman AZ. NMDA receptor antibody encephalitis presenting as transient epileptic amnesia. J Neuroimmunol. 2019;327:41–3.
204. Cretin B, Bilger M, Philippi N, Blanc F. CASPR2 antibody encephalitis presenting as transient epileptic amnesia. Seizure. 2020;81:175–7.
205. Oagawa S, Uchida Y, Kobayashi S, Takeda K, Terada K, Matsukawa N. $GABA_B$ receptor autoimmune encephalitis presenting as transient epileptic amnesia. Rinsho Shinkeigaku. 2021;61:6–11.
206. Rojas-Marcos I, Fernandez A, Caballero JA, Suarez A, Blanco A. Familial transient epileptic amnesia. Report of three siblings. J Neurol. 2012;259(Suppl1):S178. (abstract P672)
207. Paccagnella E, Gosavi TD, Neligan A, Walker M. Transient epileptic amnesia: an unusual case report. Poster presentation, BNA/ABN Meeting of Minds symposium, Cardiff, UK, 29 September 2016.
208. Romoli M, Tuna MA, Li L, et al. Time trends, frequency, characteristics and prognosis of short-duration transient global amnesia. Eur J Neurol. 2020;27:887–93.
209. Ung KYC, Larner AJ. Transient amnesia: epileptic or global? A differential diagnosis with significant implications for management. Q J Med. 2014;107:915–7.
210. Greene HH, Bennett DR. Transient global amnesia with a previously unreported EEG abnormality. Electroencephalogr Clin Neurophysiol. 1974;36:409–13.
211. 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.
212. Fouchard AA, Biberon J, Mondon K, de Toffol B. Transient epileptic amnesia secondary to hippocampal dysplasia mimicking transient global amnesia. Seizure. 2016;43:23–5.
213. Sugiyama A, Kobayashi M, Matsunaga T, Kanai T, Kuwabara S. Transient global amnesia with a hippocampal lesion followed by transient epileptic amnesia. Seizure. 2015;31:141–3.
214. Zola-Morgan S, Squire LR, Amaral DG. Human amnesia and the medial temporal region: enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus. J Neurosci. 1986;6:2950–67.
215. Auyeung M, Tsoi TH, Cheung CM, et al. Association of diffusion weighted imaging abnormalities and recurrence in transient global amnesia. J Clin Neurosci. 2011;18:531–4.
216. Lampl Y, Sadeh M, Lorberboym M. Transient global amnesia—not always a benign process. Acta Neurol Scand. 2004;110:75–9.
217. Harrison NA, Johnston K, Corno F, et al. Psychogenic amnesia: syndromes, outcome, and patterns of retrograde amnesia. Brain. 2017;140:2498–510.
218. Kritchevsky M, Chang J, Squire LR. Functional amnesia: clinical description and neuropsychological profile of 10 cases. Learn Mem. 2004;11:213–26.
219. Pujol M, Kopelman M. Psychogenic amnesia. Pract Neurol. 2003;3:292–9.
220. Staniloiu A, Markowitsch HJ. Dissociative amnesia. Lancet Psychiatry. 2014;1:226–41.
221. Gil R, Abdul-Samad F, Mathis S, Neau JP. Was there a confusion before 1950 between global transient global [sic] amnesia and psychogenic amnesia? [in French]. Rev Neurol (Paris). 2010;166:699–703.
222. 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.
223. Markowitsch HJ, Staniloiu A. The impairment of recollection in functional amnesic states. Cortex. 2013;49:1494–510.
224. Baxendale S. Memories aren't made of these: amnesia at the movies. BMJ. 2004;329:1480–3.
225. Larner AJ. "Neurological literature": cognitive disorders. Adv Clin Neurosci Rehabil. 2008;8(2):20.
226. Larner AJ. Unconscious driving phenomenon. Adv Clin Neurosci Rehabil. 2011;10(6):26.
227. Moersch F. Psychic manifestations of migraine. Am J Psych. 1924;80:697–716.
228. Evans JH. Transient loss of memory, an organic mental syndrome. Brain. 1966;89:539–48.

229. Gilbert JJ, Benson DF. Transient global amnesia: report of two cases with definite etiologies. J Nerv Ment Dis. 1972;154:461–4.
230. Laplane D, Truelle JL. The mechanism of transient global amnesia. Apropos of some unusual cases [in French]. Nouv Presse Med. 1974;3:721–5.
231. Frank G. Amnestic episodes in migraine. A contribution to the differential diagnosis of transient global amnesia (ictus amnésique) [in German]. Schweiz Arch Neurol Neurochir Psychiatr. 1976;118:253–74.
232. Caplan L, Chedru F, Lhermitte F, Mayman C. Transient global amnesia and migraine. Neurology. 1981;31:1167–70.
233. Olivarius BD, Jensen TS. Transient global amnesia in migraine. Headache. 1979;19:335–8.
234. Lane R, Davies P. Migraine. New York: Taylor & Francis; 2006.
235. Haas DC, Ross GS. Transient global amnesia triggered by mild head trauma. Brain. 1986;109:251–7.
236. Pacheva I, Ivanov I. Acute confusional migraine: is it a distinct form of migraine? Int J Clin Pract. 2013;67:250–6.
237. Miller JW, Petersen RC, Metter EJ, Millikan CH, Yanagihara T. Transient global amnesia: clinical characteristics and prognosis. Neurology. 1987;37:733–7.
238. Schipper S, Riederer F, Sander PS, Gantenbein AR. Acute confusional migraine: our knowledge to date. Expert Rev Neurother. 2012;12:307–14.
239. Sheth RD, Riggs JE, Bodensteiner JB. Acute confusional migraine: variant of transient global amnesia. Pediatr Neurol. 1995;12:129–31.
240. Larner AJ. Acute confusional migraine and transient global amnesia: variants of cognitive migraine? Int J Clin Pract. 2013;67:1066.
241. Szabo K, Hoyer C, Caplan LR, et al. Diffusion-weighted MRI in transient global amnesia and its diagnostic implications. Neurology. 2020;95:e206–12.
242. Maffei D, Grizzi F, Zanoni M, Vota P, Justich M, Taverna G. Recurrent transient global amnesia after intracavernous Caverject injection in erectile dysfunction after robotic prostatectomy. Urol Case Rep. 2020;31:101184.
243. Grande LA, Loeser JD, Samil A. Recurrent transient global amnesia with intrathecal baclofen. Anesth Analg. 2008;106:1284–7.
244. Mumenthaler M, Kaeser HE, Meyer A, Hess T. Transient global amnesia after clioquinol: five personal observations from outside Japan. J Neurol Neurosurg Psychiatry. 1979;42:1084–90.
245. Kaeser HE. Transient global amnesia due to clioquinol. Acta Neurol Scand Suppl. 1984;100:175–83.
246. Mazzucchi A, Moretti G, Caffarra P, Parma M. Neuropsychological functions in the follow-up of transient global amnesia. Brain. 1980;103:161–78.
247. 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.
248. Gil-Martinez T, Galiano R. Transient global amnesia following the use of ergots in the treatment of migraine [in Spanish]. Rev Neurol. 2004;39:929–31.
249. Teh CH, Robertson MN, Warkentin TE, Henriksen PA, Brackenbury ET, Anderson JA. Transient global amnesia as the presenting feature of heparin-induced thrombocytopenia. J Card Surg. 2010;25:300–2.
250. Sandyk R. Transient global amnesia induced by lorazepam. Clin Neuropharmacol. 1985;8:297–8.
251. Stracciari A, Guarino M, Crespi C, Pazzaglia P. Transient amnesia triggered by acute marijuana intoxication. Eur J Neurol. 1999;6:521–3.
252. Shukla PC, Moore UB. Marijuana-induced transient global amnesia. South Med J. 2004;97:782–4.
253. Mansour G, Abuzaid A, Bellamkonda P. I do not even remember what I smoked! A case of marijuana-induced transient global amnesia. Am J Med. 2014;127:e5–6.

254. Jones RJ, Brace SR, Vander Tuin EL. Probable propafenone-induced transient global amnesia. Ann Pharmacother. 1995;29:586–90.
255. 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.
256. Healy D, Morgan R, Chinnaswamy S. Transient global amnesia associated with statin intake. BMJ Case Rep. 2009;2009:pii: bcr06.2008.0033.
257. Fu PK, Hsu HY, Wang PY. Transient global amnesia after taking sibutramine: a case report. Neurologist. 2010;16:129–31.
258. Savitz SA, Caplan LR. Transient global amnesia after sildenafil (Viagra) use. Neurology. 2002;59:778.
259. Gandolfo C, Sugo A, Del Sette M. Sildenafil and transient global amnesia. Neurol Sci. 2003;24:145–6.
260. Shihman B, Auriel E, Bornstien NM. Two cases of transient global amnesia (TGA) following sildenafil use [in Hebrew]. Harefuah. 2006;145(656-7):703.
261. 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.
262. Finsterer J. Transient global amnesia associated with sildenafil use. Report of one case. Rev Med Chil. 2019;147:527–9.
263. Lin KY, Liu MN, Wang PH, Lau CI. Positive magnetic resonance imaging evidence of transient global amnesia following the use of sildenafil. Clin Neuropharmacol. 2020;43:52–3.
264. Lee E, Ghafoor N, Jefri M, Black AD, Calello DP, Santos CD. Acute coronary syndrome and transient global amnesia with sumatriptan. Am J Emerg Med. 2021; S0735-6757(21)00754-3. Online ahead of print
265. Schiefer J, Sparing R. Transient global amnesia after intake of tadalafil, a PDE-5 inhibitor: a possible association? Int J Impot Res. 2005;17:383–4.
266. Bardes I, Pujadas F, Ibarria M, Boada M. Transient global amnesia after tadalafil use [in Spanish]. Med Clin (Barc). 2008;131:78.
267. Machado A, Rodrigues M, Ribeiro M, Cerqueira J, Soares-Fernandes J. Tadalafil-induced transient global amnesia. J Neuropsychiatry Clin Neurosci. 2010;22:e28.
268. Morris HH 3rd, Estes ML. Traveler's [sic] amnesia. Transient global amnesia secondary to triazolam. JAMA. 1987;258:945–6.
269. Tsai MY, Tsai MH, Yang SC, Tseng YL, Chuang YC. Transient global amnesia-like episode due to mistaken intake of zolpidem: drug safety concern in the elderly. J Patient Saf. 2009;5:32–4.
270. Zeman A, Hoefeijzers S, Milton F, Dewar M, Carr M, Streatfield C. The GABAB receptor agonist, baclofen, contributes to three distinct varieties of amnesia in the human brain—a detailed case report. Cortex. 2016;74:9–19.
271. Ansseau M, Poncelet PF, Schmitz D. High dose triazolam and anterograde amnesia. BMJ. 1992;304:1178.
272. Brown J, Lewis V, Brown M, Horn G, Bowes JB. A comparison between transient amnesias induced by two drugs (diazepam or lorazepam) and amnesia of organic origin. Neuropsychologia. 1982;20:55–70.
273. Danek A, Uttner I, Straube A. Is transient global amnesia related to endogenous benzodiazepines? J Neurol. 2002;249:628.
274. Caplan LB. [sic]. Transient global amnesia. In: Frederiks JAM, editor. Handbook of clinical neurology. Volume 1 (45). Clinical neuropsychology. Amsterdam: Elsevier Science Publishers; 1985. p. 205–18.
275. Parkin AJ, Leng NRC. Neuropsychology of the amnesic syndrome. Hove: Lawrence Erlbaum Associates; 1993.
276. 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.

277. Chalmers J, Risk MT, Kean DM, Grant R, Ashworth B, Campbell IW. Severe amnesia after hypoglycemia. Clinical, psychometric, and magnetic resonance imaging correlations. Diabetes Care. 1991;14:922–5.
278. Holemans X, Dupuis M, Missan N, Vanderijst J-F. Reversible amnesia in a type 1 diabetic patient and bilateral hippocampal lesions on magnetic resonance imaging (MRI). Diabet Med. 2001;18:761–3.
279. Larner AJ, Moffat MA, Ghadiali E, Majid S, English P, Williams G. Amnesia following profound hypoglycaemia in a type 1 diabetic patient. Eur J Neurol. 2003;10(Suppl1):92. (abstract P1170)
280. Cox C, Larner AJ. Recurrent hypoglycaemia and cognitive impairment: a 14-year follow-up. Br J Hosp Med. 2016;77:540–1.
281. Kirchhoff BA, Lugar HM, Smith SE, et al. Hypoglycaemia-induced changes in regional brain volume and memory function. Diabet Med. 2013;30:e151-6.
282. Koempfen M. Observation sur un cas de perte de mémoire. Mémoires de l'Academie Nationale de Médecine. 1835;4:489–94.
283. Ribot T. Diseases of memory: an essay in the positive psychology. New York: D Appleton and Company; 1882.
284. Larner AJ. Did Ribot describe transient global amnesia in the nineteenth century? Cortex. 2021;138:38–9.
285. Fisher CM. Concussion amnesia. Neurology. 1966;16:826–30.
286. Friedland D, Swash M. Post-traumatic amnesia and confusional state: hazards of retrospective assessment. J Neurol Neurosurg Psychiatry. 2016;87:1068–74.
287. Marshman LA, Jakabek D, Hennessy M, Quirk F, Guazzo EP. Post-traumatic amnesia. J Clin Neurosci. 2013;20:1475–81.
288. Ardila A. Transient global amnesia resulting from mild trauma. Neuropsychology. 1989;3:23–7.
289. Moral A, Beltran A. Transient global amnesia following slight cranial injury. Neurologia. 1988;3:124–5.
290. Vohanka S, Zouhar A. Transient global amnesia after mild head injury in childhood. Acta Nerv Super (Praha). 1988;30:68–74.
291. Accurti I, Proli F. Transient global amnesia: a personal series of idiopathic and post-traumatic cases. Ital J Neurol Sci. 1988;(Suppl 9):25–8.
292. Evans RW. The postconcussion syndrome. In: Aminoff MJ, editor. Neurology and general medicine. 4th ed. Philadelphia: Churchill Livingstone Elsevier; 2008. p. 593–603.
293. Venneri A, Brazzelli M, Della Sala S. Transient global amnesia triggered by mild head injury. Brain Inj. 1998;12:605–12.
294. Coelho P, Schön M, Alves PN, Fonseca AC, Pinho E, Melo T. An image is not always worth a thousand words: an image mimic of transient global amnesia. Neurol Sci. 2021;42:2515–7.
295. Galvin R, Brathen G, Ivashynka I, et al. EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy. Eur J Neurol. 2010;17:1408–18.
296. Scalzo SJ, Bowden SC, Ambrose ML, Whelan G, Cook MJ. Wernicke-Korsakoff syndrome not related to alcohol use: a systematic review. J Neurol Neurosurg Psychiatry. 2015;86:1362–8.
297. Larner AJ, Gardner-Thorpe C. Robert Lawson (?1846-1896). J Neurol. 2012;259:792–3.
298. Sacks O. The man who mistook his wife for a hat. London: Picador; 1986.
299. Dharia S, Zeman A. Fatigue amnesia. Cortex. 2016;79:153–4.
300. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed, text revision (DSM-IV-TR). Washington: American Psychiatric Association; 2000.
301. Benon R. Les ictus amnésiques dans les démences "organiques". Ann Méd Psychol. 1909;67:207–19.
302. Benon R. Les ictus amnésiques dans la paralysie générale. Gaz Hôp (Paris). 1908;1335
303. Hall JA. Transient global amnesia in neurolues. Practitioner. 1982;226:953–5.

304. 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.
305. 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.
306. McCorry DJ, Crowley P. Transient global amnesia secondary to herpes simplex viral encephalitis. Q J Med. 2005;98:154–5.
307. Pommer B, Pilz P, Harrer G. Transient global amnesia as a manifestation of Epstein-Barr virus encephalitis. J Neurol. 1983;229:125–7.
308. Young CA, Humphrey PR, Ghadiali EJ, Klapper PE, Cleator GM. Short-term memory impairment in an alert patient as a presentation of herpes simplex encephalitis. Neurology. 1992;42:260–1.
309. Smithson E, Larner AJ. Glioblastoma multiforme masquerading as herpes simplex encephalitis. Br J Hosp Med. 2013;74:52–3.
310. Hussein HM. Transient global amnesia as a possible first manifestation of COVID-19. Neurohospitalist. 2021;11:84–6.
311. Nishizawa T, Kawakami A, Taguchi T, Osugi Y. Transient global amnesia with bilateral hippocampal lesions during the COVID-19 global outbreak. J Gen Fam Med. 2020;22:154–5.
312. Werner R, Keller M, Woehrle JC. Increased incidence of transient global amnesia during the Covid-19 crisis? Neurol Res Pract. 2020;2(1):26.