J Neurol (1999) 246 : 53–54

A. Rösler
G. J. Mrass
A. Frese
I. Albert
F. Schnorpfeil

Precipitating factors of transient global amnesia

Received: 11 November 1997
Received in revised form: 15 May 1998
Accepted: 19 May 1998

Sirs: In the past, the precipitating factors of transient global amnesia (TGA) have been mostly reported anecdotally ("amnesia at the seaside"). Only in the last 2 decades have systematic studies of these factors been performed. The proportion of patients who reported unusual precipitating activity have ranged between 30 and 69% [2, 3, 9, 11]. We undertook a retrospective study of the precipitating factors in 72 patients with TGA seen at our centre between January 1982 and March 1997; all fulfilled the diagnostic criteria of Hodges and Ward [5]. Two of us (AF, IA), who were not aware of the aims of the study, interviewed 61 of these patients directly and talked to relatives who witnessed the amnesic attacks in 3 other patients.

Patients and relatives were asked eight standardized questions concerning the situation at the onset of the attack, e.g. contact with water, emotional stress, pain, physical exercise, car driving, sexual intercourse, coughing fits and whether they were inside or outside a building. In 44 patients, precipitating factors were also clearly stated in the clinical records. Results were compared with the answers of 70 control patients with transient ischaemic attack (TIA) who were admitted to our hospital in 1997.

Fifty-eight of the TGA patients (81%; 95% confidence interval: 71–90%) described precipitating factors such as contact with cold or hot water (13); emotional stress, e.g. job interview or escape of bulls from a stable (20); physical exercise (14); pain, e.g. tooth extraction (9); sexual intercourse (1); and a coughing fit (1) immediately before the attack. Only 3 of the TIA patients (4%) could describe similar situations before the attack ($P < 0.01$). Driving a car has been mentioned by Caplan [2] as a situation often preceding TGA, but the incidence of car driving did not differ between the TGA and the control group in our sample (2 patients in each group). There was also no difference between the two groups in the location of the patients before the attack (i.e. inside or outside the building).

We report a larger proportion of precipitating factors in TGA than other investigators [3, 9, 11]. However, none of the other investigators has reported systematic questioning about the precipitating events. On the other hand, there might be an overestimation of precipitating factors in our study owing to rather wide definitions of "extraordinary emotional stress" or "strenuous physical exercise". However, exploring the situation immediately before the attack provides critical diagnostic information and aids in distinguishing the condition from TIA.

The cause of TGA is still unknown. Possible explanations range from TIAs [4], including a high incidence of paradoxical embolism [7], to migraine [5], epileptic discharges [3] and attacks owing to vertebral angiography [6]. The precipitating factors found in our study may be considered to be factors stimulating the vegetative nervous system. This is also true for the four patients who developed TGA after a Valsava manoeuvre, described in the study of Melo et al. [9]. They experienced vegetative rather than emotional triggers, proposed by Fisher as being the "common thread" of precipitating factors in TGA [3].

Spreading cortical depression (SD) [8], which has already been implicated in the pathogenesis of TGA, can be provoked by vegetative stimuli [10]. Experimental SD can cause amnesia [1], and SD has also been used to explain cortical blindness, which has been linked with TGA after vertebral angiography [6]. Hence, SD may provide a unifying explanation for TGA [10], because it is able to link the "migraine hypothesis" and the "vascular hypothesis" owing to secondary vasomotor changes.

In conclusion, the high proportion of precipitating factors in TGA in comparison with TIA underscores the need for a precise evaluation of the events before the attack. Furthermore, our findings may lend further weight to a role of SD in TGA.

References

1. Bures J, Buresova O, Krivanek J (1974) The mechanisms and applications of Leao's spreading depression of electroencephalographic activity. Academic Press, New York
2. Caplan LR (1990) Transient global manesia: Characteristic feature and overview. In: Markowitsch HJ (ed) Transient global amnesia and related disorders. Hogrefe & Huber, Göttingen, pp 15–27
3. Fisher CM (1982) Transient global amnesia. Precipitating activities and other observations. Arch Neurol 39:605–608
4. Frederiks JAM (1990) Transient global amnesia: An amnesic TIA. In: Markowitsch HJ (ed) Transient global amnesia and related disorders. Hogrefe & Huber, Göttingen, pp 28–47
5. Hodges JR, Ward CD (1989) The aetiology of transient global amnesia. Brain 112:595–620
6. Jackson A, Stewart G, Wood A, Gillespie JE (1995) Transient global amnesia and cortical blindness after vertebral angiography: further evidence for the role of arterial spasm. AJNR Am J Neuroradiol 16:955–959
7. Klotsch C, Sliwka U, Berlit P, Noth J (1996) An increased frequency of patent foramen ovale in patients with transient global amnesia. Analysis of 53 consecutive patients. Arch Neurol 53:504–508

8. Leao AAP (1944) Spreading depression activity in cerebral cortex. J Neurophysiol 7:359–370
9. Melo TP, Ferro JM, Ferro H (1992) Transient global amnesia. A case control study. Brain 115:261–270
10. Olesen J, Jorgensen MB (1986) Leao's spreading depression in the hippocampus explains transient global amnesia. Acta Neurol Scand 73:219–220
11. Zorzon M, Antonutti L, Mase G, Biasutti E, Vitrani B, Cazzato G (1995) Transient global amnesia and transient ischemic attack. Stroke 26:1536–1542

A. Rösler (✉)[1] · G. J. Mrass · A. Frese
I. Albert · F. Schnorpfeil
Department of Neurology
University Hospital, Philipps-University
Marburg, Germany

Mailing address:
[1] Cognitive Neurology and Alzheimer's Disease Center, 320 E Superior
Searle 11–453, Chicago, IL, 60611, USA
e-mail: a-rosler@nwu.edu
Tel.: +1-312-908-9769