Brief Communication

Brain Stem Dysfunction in Transient Global Amnesia

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SUMMARY A patient with transient global amnesia also had transient bilateral gaze nystagmus which was detected both by conventional bedside examination and upon electronystagmographic recording. The nystagmus was absent one week later indicating recovery of the temporary brain stem deficit. The recording of objective evidence of brain stem dysfunction in the form of gaze nystagmus in a patient who had transient global amnesia, suggests that both were due to a transient ischemic attack involving the cerebral blood supply in the vertebrobasilar distribution.

THE SYNDROME of transient global amnesia (TGA) is well known. It was named by Fisher and Adams1 but described first by Bender.2 Typically, the patient reports having had a complete loss of memory, usually for a few hours. Relatives describe the patient during the attack, as being aware of having a problem, repeatedly asking the same questions about it, and then being unable to remember the answers. During the attack the patient also has retrograde amnesia for a period of days, weeks or even years. After recovery from the episode of TGA, the retrograde amnesia gradually shrinks but leaves a period of amnesia for the duration of the attack.

We describe a patient with TGA in whom we were able to obtain electronystagmographic (ENG) evidence of transient brain stem dysfunction.

Report of a Patient

A 70-year-old man had a history of 2 episodes of myocardial infarction 10 years and one year before. For the preceding year he had been taking propranolol isosorbide and quinidine. Because of diarrhea, the quinidine had been discontinued 8 days before the episode that prompted his referral to the hospital.

The patient was well on the morning of his attack. He went out for his usual afternoon walk with a letter to post. One hour later he returned home having failed to mail the letter and for the next 15 minutes was greatly disturbed by the fact that he could not remember events of the previous day or where he had been on his walk. This gradually subsided over the succeeding 20 minutes and he was then able to remember the previous day's events. He was still amnesic concerning the walk during which he had been seen in a store and was then noted to be acting normally. He was referred from the Emergency Department of Sunnybrook Medical Centre to the Stroke Unit.

On questioning he denied ingestion of sedatives or alcohol. No neurological signs were found and the admitting officer made no specific comment about presence or absence of nystagmus. The patient was noted to have some premature ventricular beats and was placed on cardiac monitoring. A random blood sugar was normal.

On the following day neurological examination revealed bilateral horizontal gaze nystagmus, more marked on gaze to left, as an isolated abnormality. An electroencephalogram at this time showed a normal background and occasional bursts of high voltage and bilateral theta activity during hyperventilation. An ENG recording was made on the following day (fig.). The patient remained well and was discharged home on the fourth hospital day after quinidine therapy was recommenced. On clinical examination 10 days after his attack there was no gaze nystagmus and an ENG recording three days later confirmed its absence.

Comment

TGA is generally believed to be caused by impairment of blood supply to both temporal lobes, particularly the hippocampi, or reduction of blood supply to one hippocampus with the other damaged by previous disease.5-10 The syndrome tends to occur in older people, many of whom have generalized vascular disease. There are a few reports in the literature of TGA patients with a history of epilepsy or EEG findings suggesting this diagnosis; in these the syndrome may be part of psychomotor epilepsy.1, 5, 6, 11, 12

In our review of the English language literature we found that nystagmus was rarely noted in TGA patients,* and ENG recording of the sign has not been previously described. Bilateral horizontal gaze nystagmus in the absence of drug ingestion is considered to be due to involvement of brain stem-cere-
Abnormal connections. Absence of the gaze nystagmus in our patient about 10 days after the initial illness is evidence of the temporary nature of the dysfunction. It is likely that impaired blood flow in the vertebrobasilar system resulted in bilateral hippocampal ischemia and TGA in our patient. The presence of bilateral gaze nystagmus after the TGA resolved is evidence of a more prolonged brain stem deficit. We consider that these findings support the growing evidence that the majority of patients with TGA have transient ischemia in the distribution of the vertebobasilar arterial system.

References
Brain stem dysfunction in transient global amnesia.
N S Longridge, V Hachinski and H O Barber

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