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Transient Global Amnesia: The Electroencephalogram at Onset

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A middle-aged man experienced onset of transient global amnesia while undergoing an electroencephalogram. No important change in electroencephalographic activity occurred. This case provides evidence against the hypothesis that epileptic discharge is responsible for transient global amnesia.

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Transient memory loss may occur in association with temporal lobe seizures, stroke, migraine, temporal lobe encephalitis, drug intoxication, and hysteria [1].

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Transient global amnesia (TGA) is a specific stereotyped syndrome of altered behavior dominated by amnesia that occurs without other neurological symptoms or signs and resolves within hours [2].

Several etiological hypotheses have been advanced to explain TGA. Many reports have suggested a vascular mechanism [3]. Others have proposed an epileptic origin [4-7]. Some have suggested that ischemia may cause epileptic discharge, which in turn results in TGA [8, 9]. Cardiac arrhythmia [10], concussion [11], anesthesia [12], possible benzodiazepine intoxication [13], and migraine [14, 15] have all been implicated in specific cases.

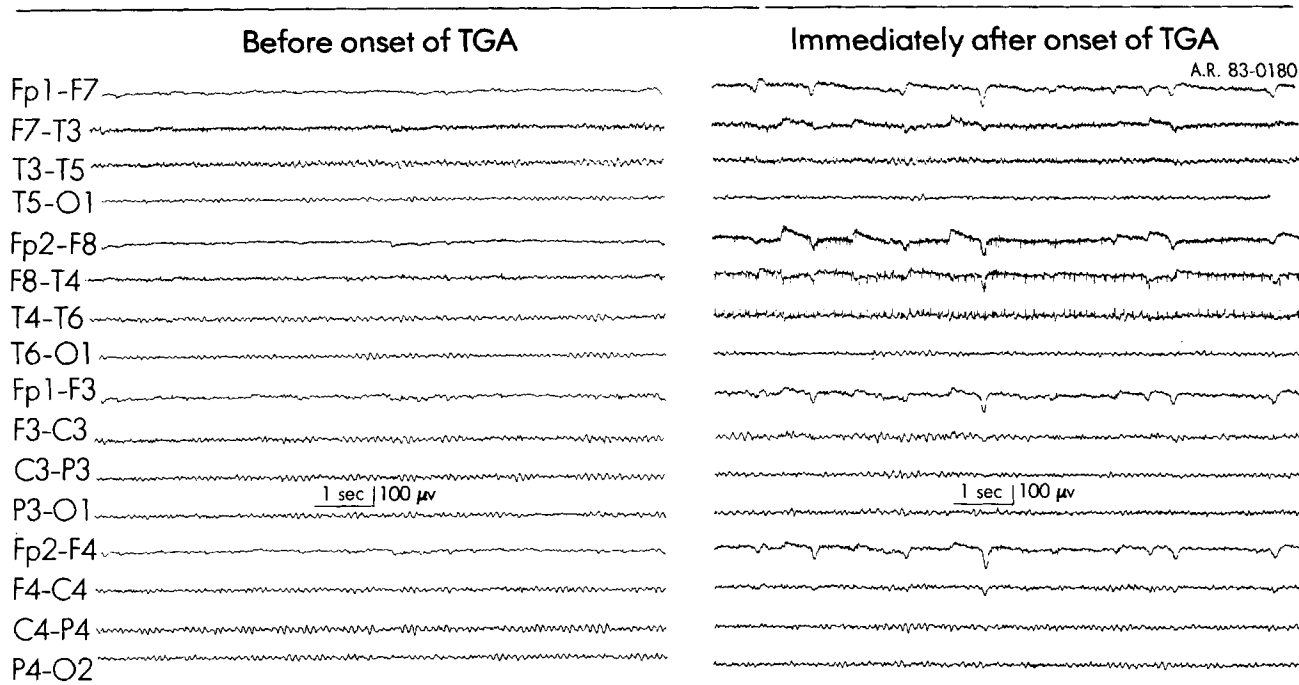
This report describes the fortuitous observation of a patient whose electroencephalogram (EEG) was being recorded at the time his attack began. No evidence of epileptic discharge either prior to, during, or after the attack was found.

Case Report

A 53-year-old right-handed man was referred for an EEG in the course of investigation of migraine. Midway through the EEG examination the technician spoke to the patient, who was able to carry on a normal conversation. The technician described the subsequent events: "When I entered the room to change to the last montage, the patient (who had acted perfectly normally until that time) suddenly said that he didn't know what he was doing there, or how he got there. He remembered the year he was born, but couldn't say how old he was. He remembered what he'd had for breakfast, but couldn't remember whether he'd driven here or come to town with a friend."

Examination by a neurologist demonstrated complete amnesia extending back to his arrival in the EEG laboratory. He was confused about how he arrived at the hospital. He asked repeatedly whether he had been to work that morning. He was oriented to person, place, and time, but was able to remember only 1 of 4 objects after 5 minutes. Digit span was 7 forward and backward. His recall of events over the preceding 3 days was patchy. His remote memory was intact, as was his calculation ability. The remainder of the neurological and systemic examination was normal. Investigations including electrocardiogram and cranial computed tomographic scanning were normal. Over the next 24 hours the patient completely recovered.

The EEG immediately prior to the onset of the attack and during the initial hour of the attack was normal. Background activity consisted of 9.5 Hz alpha rhythm dominant over posterior head regions, which was suppressed with eye opening (Figure, left panel). Hyperventilation resulted in generalized bursts of bilaterally synchronous slow waves with bifrontal predominance, which did not outlast the period of overbreathing. Immediately after the onset of TGA the background activity was unchanged, but superimposed were frequent eye blinks and some muscle artifact related to restlessness (see Figure, right panel). An EEG recorded 4 years earlier for evaluation of possible drop attacks was also normal.



Electroencephalogram recording immediately before the beginning of an attack of transient global amnesia (TGA) (left panel), and after the onset of an attack (right panel). Electrode positions conform to the standard international 10-20 nomenclature.

Discussion

Several reports of the EEG findings during attacks of TGA have described sharp discharges over one or both temporal regions [8, 10, 16]; however, in none of these reports was the onset of an attack recorded. A number of other reports [5-7, 9, 16] have emphasized the high incidence of EEG abnormalities, including temporal slowing and temporal spikes, after resolution of attacks of TGA. Many of these recordings were performed with nasopharyngeal electrodes. The resemblance of the spikes to benign epileptiform transients of sleep has been commented upon in several reports [6, 9] and raises a serious question as to their significance [17].

The fortuitous recording of the surface EEG at the onset of a classical attack of TGA casts doubt on the possibility of its epileptic etiology. The presence of ongoing normal background alpha activity over the lateral convexity is usually not seen during ictal discharge arising from mesial temporal regions. During hundreds of temporal lobe seizures recorded with sphenoidal electrodes in our laboratory, background activity over the convexity is almost invariably disrupted. Only the chance recording of the onset of an attack while sphenoidal or nasopharyngeal electrodes were in place could provide more convincing evidence that this puzzling condition is not due to epileptic activity in the limbic structures.

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