## Recurrent dysphasic status epilepticus: a case with an unusual clinical course

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During 1 year an 83-year-old male, with an unremarkable past medical history, was admitted six times with sudden onset of a prolonged language disorder consistent with fluent dysphasia. Showing no neurological sequelae after the first two hospitalizations, he developed an increasing residual deficit in the course of each consecutive episode. MRI studies showed repeatedly a small venous cavernoma in the left parieto-occipital region and no progressive vascular changes. Periodic lateralized epileptiform discharges (PLEDs) with temporo-occipital maximum and, at the last hospital stay, a transient sharp-slow-wave focus were noted. Tc-SPECT study in the acute phase revealed a temporal and parieto-occipital hyperperfused area changed to hypoperfusion after a week. Reversible ischaemic neurological deficit was diagnosed initially. PLEDs, epileptogenic focus, hyperaemic SPECT study and aspects of the clinical course suggest the diagnosis of symptomatic nonconvulsive simple partial status epilepticus. In this context the increasing residual deficit remains to be discussed.

## Transient epileptic amnesia

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Episodes of transient amnesia occurring in middle life are most often due to 'transient global amnesia' (TGA), a distinctive syndrome of amnesia for ongoing and recent events[1]. Attacks last about 4 hours and seldom recur. The pathophysiology of TGA is uncertain, but there is good evidence that it is *not* a form of epilepsy.

Recent reports[2] have suggested that temporal lobe epilepsy sometimes presents with episodes which are easily mistaken for attacks of TGA. Pointers toward a diagnosis of 'transient epileptic amnesia' (TEA) include episodes on wakening, recurrent episodes, an episode duration of less than 1 hour and the concurrent onset of more familiar manifestations of epilepsy. The distinction between TGA and TEA is important as TEA is occasionally symptomatic of structural lesions in the temporal lobes, responds to anticonvulsants and, unlike TGA, has implications for driving.

There are several unanswered questions about TEA: is it an ictal or a post-ictal phenomenon? Is it sometimes the *only* manifestation of epilepsy? Is the amnesia of TEA the only neuropsychological deficit or is it part of a more widespread confusional state? Eight recently encountered cases of putative TEA are tested against suggested criteria for diagnosis[1] and used to explore these issues.

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## Perioral myoclonia with absences: a further case report with video

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*Rationale*: Perioral myoclonia with absences (PMA) has been described by Panayiotopoulos. However, no other reports have appeared and it is not a recognized diagnosis of the ILAE.

*Case report*: We report a 10-year-old neurodevelopmentally intact boy who was diagnosed after review of an ictal home video recording. This demonstrated that what were reported as left-sided facial focal motor seizures were probably generalized absence seizures with perioral myoclonia. The ictal EEG confirmed the diagnosis by demonstrating generalized discharges. He responded well to sodium valproate treatment and has remained seizure-free with a normal EEG at 13 years of age, 1 year after stopping treatment.

*Conclusions*: PMA does seem to be specific variety of idiopathic absence epilepsy and ictal home video recording is a powerful diagnostic tool.