Correspondence

RE: DIFFERENTIAL ASPECTS OF SLEEP EPILEPSY

To the Editor

In the paper entitled "Differential aspects of sleep epilepsy" by Young et al., (November 1985 issue of the Canadian Journal of Neurological Sciences), it is stated that "focal seizures usually awaken patients"; this argument is used to support the assumption of the authors that focal seizures occurring during sleep were unlikely to remain unreported. Using this assumption and the data they collected, the authors find a predominance of generalized convulsions in patients with seizures exclusively during sleep.

We would like to point out that during the intensive video and EEG monitoring which is part of the presurgical evaluation of patients with medically intractable epilepsy, it is frequent to observe focal, partial and partial complex seizures during sleep; some of these seizures do not wake up the patient. Others wake up the patient but he or she may not be aware of them or may be amnestic for the episode. This situation has become particularly evident with the use of a computer system for the automatic recognition of seizures in the EEG (Gotman, 1982).

We do not have quantitative data to state how frequently this occurs, but we would like to emphasize that these seizures are not rare and they must be taken into consideration when evaluating the incidence and the types of seizures during sleep.

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REPLY

To the Editor

In our study (Young et al, 1985) the most commonly reported seizure type in patients with sleep epilepsy was the generalized convulsion. (We could not be certain in most cases whether such seizures were primarily or secondarily generalized.) This has been the experience of previous authors quoted in our paper. The observation that seizures which remain focal tend to waken patients from their sleep also comes from two previous clinical studies (Janz, 1953; Krischek, 1962).

Gotman et al raise a good point that some patients with partial complex seizures may not appear to wake up and if they do, they may forget their auras. The behaviour of a complex partial seizure, however, may be almost as likely to attract the attention of a spouse, parent or sibling as a generalized convulsive seizure.

Neither our study nor the previous works on sleep epilepsy have used the sophisticated computerized EEG monitoring developed by Gotman and his colleagues. Their methodology is more sensitive in detecting focal seizures than purely clinical studies. We feel it is important, however, to clearly distinguish between purely electrographic seizures (which have uncertain significance as far as the patient is concerned) from those which have clinical manifestations. Using the computerized EEG system developed by Gotman (1982), may be difficult or impossible unless there is clinical or visual correlation in every case.

Gotman et al do raise some interesting points and we hope that a careful prospective study will be done which utilizes both EEG and clinical monitoring together in an unselected population of patients with epilepsy.

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Young GB, Blume WT, Wells GA, Mertens WC, Eder S. Differential aspects of sleep epilepsy. Can J Neurol Sci 1985; 12: 317-320.

THE VALIDITY OF BINSWANGER'S DISEASE

To the Editor

We would like to bring to your notice the relatively scarce attention accorded by Huang, Wu and Luo (Can J Neurol Sci 1985; 12: 88-94) in their paper on Binswanger's disease to "imaging techniques" (Computerized Tomography and Nuclear Magnetic Resonance) and the possibilities they offer as regards the "in vivo" evaluation of the subcortical white matter alterations typical of this disease. The CT report in post-mortem verified cases of Binswanger's disease is that of a diffuse and symmetrical hypodensity of hemispheric white matter, located in the periventricular areas and in the central semiovalia, significantly associated with a variable grade of hydrocephalus. Lacunar infarcts are also commonly found in their elective locations.[1] This picture reflects the main pathological features as outlined by Binswanger:[2] pronounced subcortical white matter atrophy, of variable extension and intensity, enlargement of the cerebral ventricles and absence of cortical alterations. The subcortical white matter loss has subsequently been seen to be linked to demyelination and gliotic processes, possibly related to sclerojalinotic alteration of the small cerebral arteries. The presence of vast cortical infarcts, associated with severe atheromatous alterations of the large cerebral and extra-cranial arteries, implicates a diagnosis of Multi-Infarct Dementia rather than one of Binswanger's disease.[3] The main clinical feature of this CT-pathological picture is a progressive mental impairment of varying degree to the point of a subclinical expression, in patients with positive anamnestic report of arterial hypertension,