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,

focal neurological dysfunction and pseudo-bulbar palsy.[4] This last aspect as the CT-pathological ones related to the sequence demyelination and gliosis — white matter loss and hydrocephalus, is in agreement with the slow progression of the disease (10 years and more). The clinical CT-pathological diagnosis of Binswanger's disease considered above does not apply to the cases presently by the Authors and to the subcortical alterations they found, these latter rightly attributed by the same to terminal episodes of hypoxic-ischaemic damage in patients with multi-infarctual cortical alterations. The Authors reflections on the opportunity of not considering Binswanger's disease as a separate entity from M.I.D. do not therefore appear to be conclusive. On the other hand the studies above reported[1,3,4] suggest a possible separation, among cerebro-vascular patients, of those cases with clinical-CT-pathological features of localized or diffuse, acute or chronic, subcortical ischaemia due to sclero-jalinotic alterations of the small cerebral arteries. The classical pathological pictures of "état lacunaire", "état criblè" and Binswanger's disease, often overlapping, could then be valued in this nosological context.

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REPLY

To the Editor

We thank Drs. Inzitari and Mascalchi for reminding us of the "in vivo" evaluation of the subcortical white matter alterations in Binswanger's Disease. According to Binswanger[1] and other authors, this disease is characterized: clinically, by progressive deterioration of intellect, often associated with a variety of focal symptoms and signs in clinical plateaux or even with improvement; pathologically by large patches of demyelination of cerebral white matter with sparing of the arcuate fibres and also frank infarcts; and etiologically, by arteriosclerosis of the cerebral arteries. Binswanger was a pathologist who first described this disease by gross appearance without microscopic investigation. Later reports with postmortem examination produced many controversies: some cases without dementia[2,3] and some without arteriosclerosis of the cerebral arteries[4,5] were described.

CT findings are, after all, shadows due to various sorts of lesions, such as oedema, softening, demyelination, etc., the true nature of which can only be determined by pathological investigation or long follow-up with CT study. The hypodense area around a cerebral haemorrhage is generally thought to be due to oedema by many CT specialists, but in our study, seven of twelve haemorrhages surrounded by a ring of hypodensity on CT were later proved to be necrosis of different ages histologically.[6] Laizou et al[7] performed clinical and radiological investigations of subcortical arteriosclerotic encephalopathy with CT and cited two cases showing a low density area in the white matter which subsequently subsided, so that the hypodense areas could not be the basis of subcortical arteriosclerotic encephalopathy.

One must not neglect the anatomy of the arteries of the cerebral white matter which are supplied by long and short medullary branches centripetally and ventricular branches centrifugally, without anastomoses.[8] Changes in these arteries do not produce large patches of demyelination with sparing of the arcuate fibres; this pattern of involvement is caused only by obstruction of venous drainage.

Our denial of the existence of this disease requires further confirmation to be conclusive. We plan more work to support our contention, and hope that Drs. Inzitari and Mascalchi also will continue their follow-up studies and perhaps secure pathological evidence proving it to be a true disease.

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