Unfortunately, the boundaries between GNCSE and encephalopathy may be imprecise and vague. Thus, relevant authors have used the term "allied ictal states" for defining a category of nonconvulsive ictal states in which borderline-NCSE versus TW toxic encephalopathies have been similarly categorized.[2] It is not surprising, therefore, that the same clinical condition had been considered as encephalopathy or GNCSE depending on authors' view. Both clinical resolution of the confusional state and electroencephalographic abolition of the epileptiform discharges after the administration of intravenous benzodiazepines is the method of choice to diagnosis GNCSE. However, it is well-known that this approach has numerous limitations on the clinical practice: i/ Both TWs of toxic-metabolic origin and generalized epileptiform discharges may be suppressed with intravenous benzodiazepines; ii/An immediate clinical improvement can be difficult to evaluate in a patient under the hypnotic effects of benzodiazepines (the patients frequently fall deeply asleep); iii/ The absence of a clinical improvement after intravenous benzodiazepines is not always a definite sign of encephalopathy because a delayed normalization of the mental state may occur in GNCSE.

The electrographic differentiation between TWs and genuine generalized epileptiform discharges is particularly important in the differential diagnosis between toxic encephalopathy and drug-induced GNCSE.[3] Several medications such as ifosfamide, cefepime and baclofen have been related to confusional states. In those cases described as drug-induced GNCSE, the onset of antiepileptic treatment is always accompanied by the withdrawal of the potentially neurotoxic medication. Therefore, it is not possible to establish a unique mechanism responsible for the recovery. In addition, the increased concentration on the central nervous system of the most of these drugs seems to lower the seizure threshold by decreasing brain inhibition mediated by gamma aminobutyric acid. Therefore, a subjacent epileptic mechanism cannot be completely ruled out. Under this scenery, the utilization of simple tools, as those proposed by Boulanger et al[1] is very welcome.

A minor criticism is that due to the inclusion of an elevated number of patients with anoxic encephalopathy. Apart from the discussion whether these subjects are suffering from a genuine GNCSE or severe encephalopathy with irreversible cortical injury (some authors consider more probable this last option),[4] it is likely that electroencephalographic patterns associated with hypoxic-anoxic cerebral damage might have some differences respect to those due to toxic, metabolic or septic origin.

To summarize, as demonstrated by Boulanger et al,[1] the evaluation of the EEG response to sensory stimulation may be helpful to distinguish TWs and GNCSE. Therefore, this aspect should be carefully analyzed when suspecting encephalopathy or nonconvulsive status epilepticus.

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## To the Editor

### Re: Propionibacterium Acnes Infections after Cranial Neurosurgery. Can J Neurol Sci. 2006;33:292-5.

We read with interest the article by Michael E. Kelly et al, on "Propionibacterium Acnes Infections after Cranial Neurosurgery" published in your journal. We would like to share our experience of 21 post-operative neurosurgical infections spanning over ten years due to Propionibacterium acnes which we presented at the Canadian Congress of Neurological Sciences meeting in Montreal, June, 2006 (Post-operative neurosurgical infections due to Propionibacterium acnes).

Our series comprised of 17 brain tumours (9 gliomas, 8 meningiomas) 1 aneurysm; 2 VP shunts and 1 post traumatic. Dural grafts were performed in 16 cases (9 with galea and 7 with allodura).

Certain special features of this type of infection were noted. First, the interval between surgery and infection averaged 14.6 months but if we exclude the two VP shunts infections which occurred over three and five years after the initial surgery, then the average interval between surgery and onset of infection decreases to 4.9 months.

Secondly, the most common site of infection was in the frontal region where, seemingly, the P. acnes is more prevalent than in any other area of the scalp. There were characteristic appearances on CT scan consisting of enhanced epidural collection weeks after surgery together with the presence of air. The predilection of the infection was in the overwhelming majority in the epidural space.

We agree with the authors regarding the management of those cases with surgical debridement and removal of bone flap with antibiotic coverage for a few weeks.

It is suggested that dural graft and gelfoam may act as a culture media for the P. acnes and thus explain the frequent involvement of the epidural space.

Finally, we feel that Propionibacterium acnes is an infection which may be on the rise and this anaerobic gram positive bacillus may be less indolent than it was originally thought.

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