CORRESPONDENCE

To the Editor

Stroke Training in Canadian Neurology Residency Training Programs

Re: tPA use for Stroke in the Registry of the Canadian Stroke Network. Can J Neurol Sci. 2005;32:433-9.

Stroke patients now make up the majority of inpatient admissions to neurological wards and transient ischemic attack referrals, a growing number of outpatients. At our institution, for example, of the 911 inpatients admitted to the neurology clinical teaching unit in 2005, 611 were diagnosed with stroke. In the outpatient setting, 988 patients were assessed due to a possible transient ischemic attack. The Registry of the Canadian Stroke Network indicates that approximately 8.9% of patients presenting with an acute stroke in Canada are treated with thrombolysis.[1] It is, therefore, important for neurology trainees to develop competency in the diagnosis and management of cerebrovascular disease.

There are many challenges faced by training programs in providing adequate stroke education to neurology trainees. How much time should be allocated to training in stroke? How much training should take place in the inpatient and outpatient setting? At what point during residency should this training take place? Should trainees play an active role in the acute stroke team and in the administration of thrombolysis? How will a trainee's competency and procedural skills be assessed?

There is a paucity of studies focusing on education provided to medical trainees in stroke. In a recent US survey,[2] 27% of graduating neurology residents were not comfortable administering tPA independently and 20% had not personally treated patients with tPA. We were interested in determining the nature and amount of training in stroke provided to Canadian neurology residents. A questionnaire was developed and sent by email to all Canadian neurology residents in 2005 via the program directors of the 15 Canadian Adult Neurology Residency Training Programs. Despite a low response rate of 25% (34/136) limiting the interpretation of this study, we believe interesting and important information was still obtained.[3]

Of the 12 neurology training programs represented, six offered weekly educational stroke rounds. Half of the programs offered the opportunity for residents to undertake a local dedicated stroke elective. The duration of the stroke elective varied considerably from as short as a single month to several months in duration over the course of the residency program. A dedicated acute stroke service was present at 7 of 12 programs and residents participated in this service at all of these centers.

Twenty four of 34 residents (71%) had personally administered thrombolysis. Fifty percent of these residents (12/24), however, had administered thrombolysis on only three or fewer occasions. As expected, experience with thrombolysis increased with each post graduate year, with a median of 0 (range 0-2) cases in the first post graduate year to 7 (3-10) cases in the fifth post graduate year. There was one resident in the fourth post graduate year, however, who had never administered thrombolysis.

Despite the unevenness of dedicated training in stroke, 94% (32/34) of neurology residents were satisfied with their training in stroke. All trainees were comfortable or very comfortable with their knowledge of the modifiable risk factors for stroke and 91% (31/34) and 94% (32/34) were comfortable or very comfortable with the indications for anticoagulation and carotid endarterectomy, respectively. In addition, 74% of residents who had administered thrombolysis, stated they would be comfortable administering thrombolysis independently in the future.

Some trainees may have acquired additional knowledge by attending the annual Canadian Stroke Consortium residents course (www.strokeconsortium.ca), which has taken place for the past six years. However, it is striking that despite the inadequacy of cerebrovascular training in most programs, the great majority of residents were satisfied with it. If they only knew what they do not know. Given the increasing prevalence of cerebrovascular diseases and the fact that stroke is both treatable and preventable, this situation calls for redress.

Miguel Bussière, Vladimir Hachinski
London, Ontario, Canada

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To the Editor

Re: Triphasic Waves versus Nonconvulsive Status Epilepticus: EEG Distinction. Can J Neurol Sci 2006;33:175-80.

I wish to congratulate Dr Boulanger et al.[1] for their recent article regarding the electroencephalographic distinction between triphasic waves (TWs) and generalized nonconvulsive status epilepticus (GNCSE). These authors approached reasonably an unresolved problem with obvious therapeutic implications. They concluded that some subtle morphological criteria seen on the electroencephalogram (EEG) and, particularly, the response to stimulation may be helpful in distinguishing TWs from GNCSE.

Nowadays, only a few researchers pay attention to establish simple and general principles for improving the EEG diagnosis in the evaluation of patients with altered mental state. Toxic-metabolic encephalopathy and GNCSE are common causes of delirium and, frequently, to obtain an accurate diagnosis depends on a precise and correct electroencephalographic interpretation.

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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.

José L. Fernández-Torre
Cantabria, Spain

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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.

F. Maroun, G. Murray, J. Hutchinson, R. Avery, A. Engelbrecht
St. John's, Newfoundland, Canada

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