reported tongue sores or lip oedema as a putative side effect following BT injection, nor have we ourselves observed similar muscle reactions in any of our cases. On the other hand, many viruses, predominately herpes simplex types, varicella zoster and various coccaceae types produce oral manifestations resulting in vesicles or ulcers, or both. Together with tetanus, these changes may or may not be pathogeno-
mic for a number of other infectious agents. Without further information, appearance of tongue sores and lip oedema in this case cannot further be clarified. It seems possible that they had appeared unre-
lated to a drug reaction as a common or uncommon stomatological infection, with or without upper pharyngeal/respiratory infection.

The authors use the sequence of clinical events and the neurophysiological findings as their main argument for a relationship between the injection of botulinum type A and the upper brachialplexopathy. From this sequence, however, the plexopathy could be considered unre-
lated to the BT injections as well. The fol-
lowing two arguments, however, do not preclude an immune-mediated mechanism for its occurrence.

Firstly, plexopathy started with irradiat-
ing neck pain that, after a free interval of 23 days, was followed by weakness of selected shoulder and arm muscles. Despite the gen-

erally assumed clinical similarity of immune to non-immune forms of brachial plex-
athy, this interval between pain and onset of weakness is frequently significantly longer in the non-immune form of brachial plex-
athy; a booster injection was therefore performed 15 days later. At that time, 2 ml of a solution of 200 LD 50 U/ml of saline were injected in both posterior cervical regions.

Tongue sores and lip oedema are con-
spicuous adverse events. The causality be-
 tween the use of a drug and their appear-
ance is difficult to establish. Although there is an long list of drugs that may cause these events, oth-
er aetiologies cannot be excluded. We admit that tongue sores would not be mentioned if not actively sought. Only after the patient had been informed of these events and their tongue sores were dis-
crete but in our patient the complaints were serious.

We admit that it is impossible to be sure of a causal relationship in the use of BT and the development of brachial plex-
opathy but the possibility does exist.

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Sampaio et al reply: In our patient we used 200 LD 50 U/ml per ml of saline and in the first treatment, we injected 4 ml saline distributed in eight points: two in the right sternomastoid; three in the right posterior cervical region (splenium capitis and trapezius); and three in the left posterior cervical region (splen-
ium capitis and trapezius).

After this first treatment there was no clinical improvement in the cervical dysto-

nia; a booster injection was therefore performed 15 days later. At that time, 2 ml of a solution of 200 LD 50 U/ml per ml of saline were injected in both posterior cervical regions.

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Delirium and quantitative EEG

In the recent report by Jacobson et al on conventional and quantitative EEG in the diagnosis of delirium in the elderly, the authors report that there are variables which distinguish normal from encephalopathic records (mini-mental State Examination and relative power in delta and an index of EEG slowing). Jacobson et al stated that EEG with quantitative analysis has the potential to provide important information to supplement the clinical examination, in making an appropriate and timely diagnosis. Our experiences generally agree with studies by Koponen et al and Jacobson et al—to our knowledge, the only recent reports of quantitative EEG in delirium. Nevertheless, we wish to stress some points.

Firstly, in 1990, Inouye et al reported a valid and reliable instrument in the detec-
tion of delirium, called the assessment method (CAM). This consists of nine crite-
ria from DSM-III-R and can be completed in less than five minutes.

Secondly, some qualitative changes seen in "true" EEG (triphasic waves and focal and diffuse epileptic discharges) may not be recognised if we used the quantitative EEG alone. In particular, specific EEG patterns, including periodic lateralised epileptiform discharges suggestive of focal intracranial causes of ACS, and diffuse abnormal EEG activity (triphasic waves, spikes, sharp waves, and spike and wave complexes) may not be recognised by using automated fre-
quency analysis alone.

Thirdly, quantitative EEGs followed seri-

ally over time seem to show that delirium is less transient than currently believed: in our preliminary study, six of 12 patients had focal diffuse increase of theta band power, or both, after the resolution of clini-
cal features of ACS.

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Diagnosis by axilla skin biopsy in an early case of Lafora's disease

Rubio et al reported a young girl with a family history of Lafora's disease, myoclonus affecting the upper limbs and head, EEG abnormalities, no evidence of dementia and the presence of Lafora bodies in skin axilar tissue. We describe two siblings with Lafora's disease: one with epilepsy, myoclonus, EEG abnormalities, severe dementia and numerous Lafora bodies in the muscle and skin tissue; the other without dementia complained of one myoclonic seizure of the upper arms, and had EEG abnormalities and atrophy in the muscle and skin tissues. We concluded that the diagnosis of Lafora's disease by skin and muscle biopsy is possible in the early stages of the disease, when there are myoclonic epilepsy and EEG abnormalities, and before the onset of dementia.

In our case of Lafora's disease the diagnosis was made in an earlier clinical stage.