reported tongue sores or lip oedema as a putative side effect following BT injection, nor have we ourselves observed similar mucosal reactions in any of our cases. On the other hand, many viruses, predominantly herpes simplex types, varicella zoster and various coxsackie types produce oral manifestations resulting in vesicles or ulcers, or both. Together with enanthemas, these changes may or may not be pathognomonic for a number of other infectious agents.[5] Without further information, appearance of tongue sores and lip oedema in this case cannot further be clarified. It seems possible that they had appeared unrelated to a drug reaction as a common or uncommon stomatological infection, with or without upper pharyngeal/respiratory infection.[5]

The authors use the sequence of clinical events and the neurophysiological findings as their main argument for a relationship between BT injections and upper brachial plexopathy. From this sequence, however, the plexopathy could be considered unrelated to the BT injections as well. The following two arguments, however, do not preclude an immune-mediated mechanism for its occurrence.

Firstly, plexopathy started with irradiating neck pain that, after a free interval of 23 days, was followed by weakness of selected shoulder and arm muscles. Despite the generally assumed clinical similarity of immune to non-immune forms of brachial plexopathy, this interval between pain and onset of weakness is frequently significantly longer in non-immune cases.[6] Persistence of pain at the onset of weakness, on the other hand, is seen more frequently in the serogenetic forms.[7]

Secondly, the haemagglutinin-toxin complex of the *Clostridium botulinum* type A administered has strong antigenic and biochemical similarities to the toxoid of *C. tetani*.[8] In accordance with everyday neurological experience, however, vaccine-induced plexopathies from the toxoid form of *C. tetani* are extremely rare.[6] Given the worldwide, billion-fold application of tetanus toxoid for many decades, it seems improbable that vaccine-induced complications following BT injections at the peripheral nervous system will occur at a conspiciously higher rate than with tetanus toxoid.

BT is a new therapeutic agent with a high level of medical surveillance. Medical observation, therefore, will link any evidence of a possible adverse event to the administration of such an agent; this is even more likely, if the event represents a condition with a generally ill-defined aetiology, such as the non-serogenetic or non-vaccine-induced forms of ("idiopathic") brachial plexopathy.[7] Analyses of such cases must take into account selection biases, before further conclusions are drawn.

In our opinion, the above documentation does not sufficiently rule out the mere coincidence between BT injections and the bilateral brachial plexopathy. As BT is one of the most important novelties of neurological treatment in recent years, possible adverse effects in its use merit close attention, but should be documented as completely as possible.

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Sampaio et al reply:

In our patient we used 200 LD 50 U/l 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 (splenium capitis and trapezius).

After this first treatment there was no clinical improvement in the cervical dystonia; a booster injection was therefore performed 15 days later. At that time, 2 ml of a solution of 200 LD 50 U/l per ml of saline were injected in both posterior cervical regions.

Tongue sores and lip oedema are conspicuous adverse events. The causality between the use of a drug and their appearance is difficult to establish. Although there is an long list of drugs that may cause these events,[1] other aetiologies cannot be excluded. We admit that tongue sores would not be mentioned if not actively sought. Only four patients of 102 confirmed their presence. Their tongue sores were discrete but in our patient the complaints were serious.

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

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Delirium and quantitative EEG

In the recent report by Jacobson *et al*[1] 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 the alpha frequency band), and delirium from dementia (theta activity, 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*[2] and Jacobson *et al*[1]—to our knowledge, the only two recent reports of quantitative EEG in delirium. Nevertheless, we wish to stress some points.

Firstly, in 1990, Inouye *et al*[3] reported a valid and reliable instrument in the detection of delirium, confusion assessment method (CAM). This consists of nine criteria from DSM-III-R and can be completed in less than five minutes.

Secondly, some qualitative changes seen in "raw 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,[4] and diffuse abnormal EEG activity (triphasic waves, spikes, sharp waves, and spike and wave complexes)[5] may not be recognised by using automated frequency analysis alone.

Thirdly, quantitative EEGs followed serially over time seem to show that delirium is less transient than currently believed: in our preliminary study, six of 10 patients had focal or diffuse increase of theta absolute power, or both, after the resolution of clinical features of ACS.

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

Rubio *et al*[1] 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 axillar tissue.

We describe[2] 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 Lafora bodies 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

than the Spanish case. The EEG, normal for background activity, did not differ from an EEG of primary idiopathic generalised myoclonic epilepsy and the patient had had only one isolated myoclonic seizure of the upper arms.

Rubio *et al* [1] have confirmed our reported data and it is interesting that the less aggressive skin and muscle biopsies are diagnostic in the early stages of the disease.

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Devic's neuromyelitis optica and Schilder's myelinoclastic diffuse sclerosis

I read with interest the article by Hainfellner *et al*[1] reporting the occurrence of Devic's neuromyelitis optica and Schilder's myelinoclastic diffuse sclerosis. I was surprised to find no information about anti-DNA antibodies or complement levels. Furthermore it was stated that the erythrocyte sedimentation rate was 27 mm in the first hour and that necropsy revealed perivascular lymphoid cell infiltrates of varying prominence. These findings, including the observation of cerebrospinal fluid immunoglobulin abnormalities, can be seen in systemic lupus erythematosus (SLE).[2] It is known that SLE may induce Devic's syndrome and may lead to a false diagnosis of demyelinating disease, especially when systemic manifestations of the disease are not yet present.[3,4] I followed a patient with SLE and Devic's syndrome who was reported elsewhere.[5] What are the arguments against SLE being the cause of the disease observed in the patient reported by Hainfellner *et al*?

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NOTICES

Riyadh Armed Forces Hospital Fourth International Course on Magnetic Resonance Imaging

The Departments of Radiology, Medical Physics and Medical Studies of the Riyadh Armed Forces Hospital are sponsoring the Fourth Course on Magnetic Resonance Imaging (MRI) on 2–5 October 1994. The course will provide an overview of MRI technology, and basic principles, current, and future applications of MRI in the whole body. Current and potential applications of MRI spectroscopy will also be discussed. Small group workshops on basic physics, neuro-applications, musculoskeletal and genitourinary systems will be provided for all participants. *Fees*: 1300 Saudi Riyals (1$ = 3·75 Riyals); 650 Saudi Riyals for medical staff-in-training; 325 Saudi Riyals for radiographers. *For further information contact*: Department of Medical Studies, Armed Forces Hospital, PO Box 7897, Riyadh 11159, Saudi Arabia. *Tel*: ++966 1 477 7714 (Ext. 2289/2269); *Fax*: ++966 1 477 7194/477 9168.

Third International Congress of Movement Disorders

The congress will be held on 8–12 November 1994 in Lake Buena Vista (Orlando), Florida, USA. The deadline for abstract submission is 1 April 1994. For further information contact: Central Headquarters Office, The Movement Disorder Society, PO Box 6, Clarastrasse 57, CH-4005 Basel, Switzerland. Tel: ++41 61 691 51 11; Fax: ++41 61 691 81 89.

BOOK REVIEWS

All titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the United Kingdom and for members of the British Forces Overseas, but overseas customers should add £2 per item for postage and packing. Payment can be made by cheque in sterling drawn on a United Kingdom bank, or by credit card (Mastercard, Visa or American Express) stating card number, expiratory date, and your full name.

Neuropsychology of the Amnesic Syndrome (Brain Damage, Behaviour and Cognition Series). By A J PARKIN and N R C LENG. (Pp 202 Illustrated; Price: £24.95). 1993. Hove (UK) Lawrence Erlbaum Assoc. Publishers. ISBN 0-86377-200-5.

For the purposes of this book the "amnesic syndrome" is defined in terms of permanent, non-progressive forms of amnesia linked to cerebral pathology. In practice the disorders considered as meeting this definition are amnesic phenomena associated with such things as the Wernicke-Korsakoff syndrome, temporal lobe damage, diencephalic and thalamic lesions, herpes simplex encephalitis and anterior communicating aneurysms. After introductory chapters discussing models of memory and the assessment of memory disorders, neuropsychological research into amnesia resulting from each of the above causes is described. The book concludes with a discussion of some underlying theoretical issues and a brief chapter on the remediation of memory disorder.

Because the neuropsychology of amnesia is a heavily researched area with a large volume of potentially relevant published work, this book attempts to encompass a great deal in little more than 150 pages. This is inevitably selective and occasionally skates a little thinly over some details that might suggest reservations about the picture that is being offered. Nevertheless, the selection is generally judicious and the discussion is almost always clear and easy to follow as well as being up-to-date.

Overall this book can certainly be recommended as a useful and clear introduction to the psychology of amnesic syndromes. As such it fills a definite gap and deserves to be widely read by members of the various disciplines concerned with the problem of amnesia and not just by psychologists.

E MILLER

Down Syndrome & Alzheimer Disease (Progress in Clinical and Biological Research Vol. 379). EDITED by LYNN NADEL and CHARLES J EPSTEIN. (Pp 318; Price $96.00). 1992. New York, Wiley-Liss Inc. ISBN 0-471-58841-5.

This book primarily serves as a permanent record of the proceedings of the 8th Science Symposium of the National Down Syndrome Society of the US, held in New York in January 1992. Its contents are 17 unedited, rapid reproduction manuscripts covering clinical aspects of dementia in Down's syndrome, chromosome 21, and animal models. Thus, there is considerable variation in the balance between review, and reporting of personal research work. A good proportion of the review material is also repeated from chapter to chapter.

The contents explore a number of associations. First, the development of features of Alzheimer's disease in Down's syndrome or trisomy 21, which pointed towards the second main observation of mutations of the amyloid precursor protein gene in some families with Alzheimer's disease. There is also an apparent increase in Down's syndrome in relatives of those with Alzheimer's disease. The observation that amyloid may be deposited in the brain in Down's syndrome and normal people as long as 30 years before the onset of dementia also appears to support amyloid as the prerequisite for neuronal loss and tangles. However the link between the amyloid gene mutations in Alzheimer's disease, and the pathogenesis of Alzheimer pathology in Down's and Alzheimer's remains obscure. Since preparation of the book, a cloud has descended over the validity of early claims that transgenic models with alterations of normal amyloid precursor protein expression develop neuronal loss and tangles.

This book will have a fairly limited appeal, mainly to budding research scien-