

LETTERS TO THE EDITOR

Sumatriptan and daily headache

Frequent use of ergotamine and simple analgesics is well recognised as a cause of chronic headache syndromes induced by drugs or drug withdrawal. Sumatriptan, a new and highly effective antimigraine drug, seemed not to produce this unpleasant side effect, as shown by long term studies.¹ Nevertheless, eight cases of sumatriptan misuse and daily chronic headache have been reported recently.^{2,3} Another 10 similar cases have been published in abstract form.⁴⁻⁶ The discrepancy between drug trials and clinical evidence may be due to the type of patients considered. In fact, eight of the 18 patients who used sumatriptan daily switched from misuse of analgesics or ergotamine to misuse of sumatriptan, 10 (nine of whom were migraineurs with a previous history of analgesic overuse) developed sumatriptan induced daily headache de novo. This kind of patient is not usually included in drug trials but is often found in clinical practice.

Sumatriptan has been reported to induce repeated recurrence of migraine attacks that respond to further doses of the drug.^{7,8} It may be possible that a similar mechanism occurs in drug induced migraine-like headache. In our opinion only one dose per week of sumatriptan should be prescribed to patients with either previous or current daily headache caused by drug misuse.

More reports of daily headache induced de novo by sumatriptan are needed to determine whether or not this new drug can transform migraine into chronic daily headache, as is already well documented for ergotamine and analgesics.

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Bilateral optic neuritis after hepatitis A

In adults bilateral optic neuritis is most commonly seen with demyelinating disease. An acute form exists in which both eyes are affected simultaneously. This acute simultaneous bilateral optic neuritis (ASBON) behaves differently from that associated with demyelinating disease and is characterised by a good visual prognosis and a low incidence of subsequent neurological disease.^{1,2} Here we describe a case of ASBON after hepatitis A infection.

A 22 year old female nurse presented in October 1992 with a three week history of malaise, lethargy, and abdominal pain, followed by the onset of jaundice. A full infectious screen detected hepatitis A specific IgM antibody only. One week later she developed pain around both eyes, which was exacerbated by movement. After a further week the visual acuity in her right eye deteriorated, followed two days later by her left eye. On presentation to the eye department, the visual acuity was 6/24 in the right eye and 6/18 in the left eye. There was a right relative afferent pupillary defect and colour vision was impaired in both eyes. Fundal examination showed bilateral but asymmetric optic disc swelling, with her right disc more swollen. Automated examination of the visual field showed diffuse field loss in both eyes. Apart from the presence of jaundice, general medical and neurological examination was normal.

Over the next three days the visual acuity fell to "no perception of light" and "perception of light" in the right and left eyes respectively. Head and orbit CTs showed no abnormality; MRI of the head did not show any white matter lesions and views of the optic nerves were normal. Visual evoked potentials showed prolonged latencies in both eyes, with averaged P2 latencies of 139 ms on the right and 121 ms on the left. Auditory and somatosensory potentials were normal. Lumbar puncture showed clear CSF, with an opening pressure of 10.5 cm H₂O. The CSF protein was 0.20 g/l, CSF IgG was 0.03 g/l, and there were no white cells or oligoclonal bands. Treatment with intravenous methylprednisolone and oral prednisolone was started.

Twelve days after the initiation of treatment the visual acuity had improved to "count fingers" and 6/5 in the right and left eyes. After one month the acuity had improved further to 6/18 and 6/5 respectively, and after four months to 6/12 and 6/5. One year after the onset of symptoms the patient had developed no further ocular or neurological symptoms. There was, however, temporal pallor of both optic discs and a persistent afferent pupillary defect.

ASBON occurs more often in children, and has been described after immunisation and after several viral and bacterial infections. Cases have been described in adults after fevers and also after chickenpox.^{2,3}

It is questionable, given the relatively good visual prognosis in adults with ASBON whether treatment with intravenous and oral steroids was indicated in this patient. Given the profound loss of vision, we thought that steroids were indicated to hasten visual recovery.⁴ Whereas the visual improvement began quickly, it continued for over six months after the cessation of treatment. A previous report of patients with unilateral optic neuritis has shown that even those with profound visual

loss initially may recover good vision, and this was the case here.⁵

In any case of adult bilateral optic neuritis the diagnosis of multiple sclerosis must be considered. Given the normal CSF examination, normal MRI, and the absence of further neurological symptoms after more than one year of follow up, we believe that multiple sclerosis is most unlikely but that hepatitis A was the causative factor.

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Eyelid opening apraxia in focal cortical degeneration

Tyrrell and associates recently described a novel neurodegenerative syndrome in which patients develop progressive restriction of speech output and orofacial apraxia.¹ All patients initially retained normal intellectual capacity. Positron emission tomography showed striking bifrontal hypometabolism, providing a functional neuroanatomical correlation with the clinical deficits. Despite considerable orofacial apraxia, the patients described by Tyrrell *et al*¹ apparently did not show abnormal ocular or eyelid movement.

We encountered a patient who had a virtually identical syndrome of progressive speech loss and orofacial dyspraxia. Whereas initial manifestations likely resulted from left frontal lobe dysfunction, she eventually developed pronounced lid opening apraxia, possibly reflecting progression of disease to homologous regions of non-dominant frontal cortex.

A 72 year old woman initially noted difficulty selecting and pronouncing words during telephone conversations in 1991. Her spouse noted that one of the earliest signs was a change in the way her mouth moved when they kissed. Despite a speech disturbance, the patient noted no dysphagia and recalled no other neurological symptoms. The problems gradually advanced to the point that she could only utter single, poorly pronounced words. Nevertheless, she understood spoken language and retained complete expressive communication through writing. Her memory also remained intact. By 1993, she noted difficulty initiating movement and her gait became slow and unsteady. No tremor or adventitious movements were ever witnessed. Within 12 months, the patient experienced difficulty

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Cavanagh replies:

Having read Wu's reply to my earlier criticism I still think that this case should not be regarded as anything more than "suspected triphenyltin intoxication". There are too many uncertainties for the conclusions to be anything firmer. One important uncertainty is the remarkably slow though sustained evolution of the signs of change in the nervous system. While ataxia and blurred vision were early events, it was two weeks before he slipped into semicomatose in November and he lay in coma virtually until the beginning of February. Signs of peripheral neuropathy developed two months after admission and persisted for several months more. The pattern of the neuropathy suggested an axonal mechanism whereas the electrophysiology gave evidence of myelin loss. Another uncertainty is the dose the subject absorbed, which is unknown, nor do we have any blood concentrations. Although it might seem from the reports that animal studies support the suggestion that triphenyltin can be neurotoxic, when such studies are unaccompanied by thorough morphological work interpretation is always very difficult and experience strongly suggests that these should be taken with the proverbial pinch of salt, especially when they have not been confirmed by others.

Triphenyltin compounds are widely used in the field and are generally considered to be free of serious neurological side effects, unlike trimethyl and triethyl compounds each of which produces its own pattern of affected cell types. On available evidence it is to be doubted whether there will be any future occasion when the claim of Wu and his colleagues will be supported, but should this happen I am content that this discussion and my initial reservations will be quoted.

JP CAVANAGH

NOTICES

Stanley Foundation Research Awards Program Announcement of available research funds for research on schizophrenia and bipolar disorder

The Theodore and Vada Stanley Foundation, in collaboration with the National Alliance for the Mentally Ill, wel-

come applications for the 1996 Stanley Foundation Research Awards Program. The purpose of the awards is to support research directly related to the causes or treatment of schizophrenia and bipolar disorder.

The research awards are intended to attract established scientists from other areas of biology and medicine (for example, biochemistry, immunology, virology, and neurology) into research on schizophrenia and bipolar disorder as well as to provide support for innovative research by scientists already in the field whose funding sources are limited. Applicants are invited from all stages of career development.

Awards are for one or two years. They may be up to \$75 000 per year for studies involving human subjects and up to \$50 000 per year for other studies. Funds may be used for salaries, supplies, and equipment, but it is the policy of the Stanley Foundation not to pay indirect costs for administration of the award. In 1995, 49 applications were funded out of a total of 220 received.

Deadline for receipt of applications is 1 March 1996. The 4 page application consists of a brief outline of the proposed project, a budget, and a list of current and pending sources of funding. Notification of awards is made in June and funding to award recipients begins in August.

The research award applications are reviewed by a professional selection committee.

Requests for applications and questions should be directed to: Research Awards Coordinator, Stanley Foundation Research Awards Program, c/o NAMI, 200 North Glebe Road, Suite 1015, Arlington, VA 22203-3754, USA. Tel (703) 524-7600; fax (703) 524-9094

Sixth Meeting of the European Neurological Society June 8-12 1996 Netherlands Congress Centre, The Hague, The Netherlands.

Administrative Secretariat ENS 1996, c/o AKM Congress Service, PO Box, 4005 Basel, Switzerland, Tel ++41 61 691 51 11, Fax: ++41 691 81 89.

British Neurosurgery Research Group Meeting together with the North American Research Society of Neurological Surgeons Meeting, 1996.

This joint meeting will be held in Newcastle upon Tyne, 23-25 May 1996.

For further information contact: Professor A David Mendelow, Newcastle General Hospital, Westgate Road, Newcastle upon Tyne NE4 6BE, UK.

World Federation of Neurosurgical Societies Awards to young neurosurgeons.

The World Federation of Neurosurgical Societies will give five awards to young neurosurgeons for the best papers submitted for presentation at the XI International Congress of Neurological Surgery to be held in Amsterdam, Netherlands 6-11 July 1997. This will be open to all neurosurgeons born after 31 December 1961. Each award will consist of an honorarium of US \$1500, a certificate for the Congress. The papers will be judged by a committee and must contain

original, unpublished work on basic research or clinical studies related to neurosurgery.

Young neurosurgeons should submit eight copies of the manuscript (not more than 10 double spaced typewritten pages exclusive of figures and tables) to: Albert L Rhoton, Jr, MD Chairman, WFNS Young Neurosurgeons' Committee, Department of Neurological Surgery, University of Florida Medical Center, PO Box 100265; 1600 SW Archer Road Gainesville, Florida 32610-0265, USA.

The submission should be accompanied by a supporting letter from the head of the candidate's neurosurgical department. The last date for submission is 1 October 1996.

Announcement from the British Neuropsychiatry Association: 1996 meetings

The 1996 Winter meeting—a joint meeting with The British Neuropsychological Society—will be held on Friday 19 January at the London Zoo. "Disorders of reasoning and perception" is the theme of the morning session and there will be presentation of short scientific papers and single case videos by members of both associations in the afternoon.

The 1996 Summer meeting will be held on 14-16 July at Robinson College, Cambridge. It will include topics on neurodevelopment, language, and the presentation of short scientific papers and single case videos by members. The Association's AGM will be held on 16 July.

For further details of these meetings please contact: Sue Garratt, Administrative Assistant, BNPA, 17 Clocktower Mews, London N1 7BB. Telephone/Fax: 0171 226 5949.

For details of membership of the BNPA, which is open to medical practitioners in psychiatry, neurology, and related clinical neurosciences, please contact: Dr Jonathan Bird, Secretary BNPA, Burden Neurological Hospital, Stoke Lane, Stapleton, Bristol, BS16 1QT. Telephone: 01179 701212 ext 2925/2929 or Sue Garratt at the address given above.

CORRECTIONS

Catarci T, Lenzi GL, Cerbo R, Fieschi C. Sumatriptan and daily headache. *J Neurol Neurosurg Psychiatry* 1995;58:508.

The reference to Osborne *et al* should be *BMJ* 1994;308:113.

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In table 2 (bottom line) the mean R2 index (range) in the third EMG subclass should be 31 (28-37).