LETTERS TO THE EDITOR

Sumatriptan and daily headache

Frequent use of ergotamine and simple analgesics is well recognised as a cause of chronic headache in patients disorders 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, 8 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 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 misuse of sumatriptan, 10 (none 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. It may be 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 pattern of misusing drugs.

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

T CATARCI G L LENZI C FIESCHI
Department of Neurosciences, La Sapienza University, Via dell'Universitá 100, Rome, Italy

Correspondence to: Dr T Catarci.


Bilateral optic neuropathy 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. Two recent similar case reports of bilateral optic neuritis (ASBON) has been shown by two different associated with demyelinating disease and is characterised by a good visual prognosis and a low incidence of subsequent clinical visual loss.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 generalized pain, followed by the onset of jaundice. A full infectious screen detected hepatitis A specific IgM antibody only. One week later she developed right eye pain, 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 6/6 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 asymmetric optic discs, one with a bright 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 abnormalities. MRI of the brain 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 white cell count of the CSF 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 afferent loss 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.3 4 It is questionable, given the relatively good visual prognosis in adults with ASBON whether she both oral steroids and 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.5 Whereas the visual improvement was seen 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.6

In any case of adult bilateral optic neuritis the diagnosis of multiple sclerosis must be considered. Given the normal CSF protein, cerebrospinal fluid findings, 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.

M MCKIBBEN P G CLELAND Department of Neurology, Sunderland General Hospital, Knotty Road, Sunderland, Tyne and Wear SR4 7TP, UK
S J MORGAN Sunderland Eye Infirmary, Queen Alexandra Road, Sunderland, Tyne and Wear SR2 2NP, UK

Correspondence to: Dr M McKibben.


Eyelid opening apraxia in focal cortical degeneration

Tyrell 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 correlate.2 Despite occasional pathological Drs, considerable orofacial apraxia, the patients described by Tyrell et al 3 apparently didn't show normal ocular or eyelid movement.

We encountered a patient who had a visually identical syndrome of progressive speech loss and orofacial apraxia. 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, the patient's difficulty initiating movement and her gait became slow and unstable. No tremor or adventitious movements were ever witnessed. Within 12 months, the patient experienced difficulty

Downloaded from http://jnnp.bmj.com/ on June 24, 2017 - Published by group.bmj.com
Bilateral optic neuritis after hepatitis A.

M McKibbin, P G Cleland and S J Morgan

*J Neural Neurosurg Psychiatry* 1995 58: 508

doi: 10.1136/jnnp.58.4.508-a

Updated information and services can be found at:
http://jnnp.bmj.com/content/58/4/508.2.citation

These include:

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Errata**

An erratum has been published regarding this article. Please see [next page](http://jnnp.bmj.com/content/59/6/662.2.full.pdf)

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/
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 Mentality Ill, will be seeking 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 statement of the object, 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 22203-3734, USA. Tel (703) 524-7600; fax (703) 524-9094

Sixth Meeting of the European Neurological Society June 8-12 1996

Administrative Secretariat ENS 1996, c/o AKM Congress Service, PO Box, 4004Z Bazel, 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, 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 young 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.

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


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


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