Electromyography and recovery of the blink reflex in involuntary eyelid closure: a comparative study

M Aramideh, J L A Eekhof, L J Bour, J H T M Koelman, J D Speelman, B W Ongerboer de Visser

Abstract
Electromyographic (EMG) activity of orbicularis oculi and levator palpebrae muscles was recorded to study the origin of involuntary eyelid closure in 33 patients. The evoked blink reflex in all patients and in 23 controls was also studied. To examine the excitability of facial motoneurons and bulbar interneurons in individual patients and to compare the results with EMG findings, R1 and R2 recovery indices were calculated in all subjects, as the average of recovery values at 0-5, 0-3, and 0-21 second interstimulus intervals. Based on EMG patterns, the patients were divided into three subclasses: EMG subclass 1, 10 patients with involuntary discharges solely in orbicularis oculi muscle; EMG subclass 2, 20 patients with involuntary discharges in orbicularis oculi and either involuntary levator palpebrae inhibition or a disturbed reciprocal innervation between orbicularis oculi and levator palpebrae; EMG subclass 3, three patients who did not have blepharospasm, but had involuntary levator palpebrae inhibition in association with a basal ganglia disease. The total patient group showed an enhanced recovery of both R1 and R2 components compared with controls. Although 30 out of 33 patients had blepharospasm (EMG subclasses 1 and 2), R1 recovery index was normal in 64% and R2 recovery index was normal in 54%. Patients with an abnormal R2 recovery index had an abnormal R1 recovery index significantly more often. All patients from EMG subclass 1 had an abnormal R2 recovery index, whereas all patients from EMG subclass 3 had normal recovery indices for both R1 and R2 responses. Seventy five per cent of the patients from EMG subclass 2 had normal recovery indices. The results provide further evidence that physiologically blepharospasm is not a homogeneous disease entity, and indicate that different pathophysiological mechanisms at the suprasegmental, or segmental level, or both are involved.

Keywords: dystonia; blepharospasm; blink reflex recovery

Blepharospasm is a form of focal dystonia, with an aetiology and underlying pathophysiological mechanisms that are still obscure. Blepharospasm may occur in diseases of the basal ganglia or in association with upper brain stem abnormalities. In most patients, however, it is idiopathic in origin.

One investigation technique that has contributed to our understanding of blepharospasm is the recorded blink reflex recovery, which indicates the state of excitability of the facial motoneurons and bulbar interneurons. Electrical stimulation of the supraorbital nerve elicits a blink reflex that consists of two components; an early ipsilateral response, R1, mediated through the pons by an oligosynaptic pathway and a late bilateral response, R2, relayed through a polysynaptic medullary pathway. Motoneurons of the facial nerve constitute the final common path for both responses. The recovery curve, also known as excitability cycle, of R1 and R2 responses can be obtained by applying two stimuli, conditioning and test, to the supraorbital nerve at different interstimulus intervals. The excitability of the blink reflex circuit may then be assessed by comparing the size of the test response with that of the conditioning response, which is partly dependent on the functional integrity of the suprasegmental structures. In healthy subjects, the R2 test response shows pronounced suppression at shorter interstimulus intervals, whereas patients with cortical lesions may exhibit an enhanced habituation and patients with basal ganglia disorders a diminished habituation (enhanced recovery) of the response.

Previous work has shown an enhanced recovery of the R2 response in patients with blepharospasm, which suggests hyperexcitability of the bulbar interneurons.

By simultaneous recording of the electromyographic (EMG) activities from the orbicularis oculi and levator palpebrae muscles, we have shown that patients with blepharospasm are not a homogeneous group, because differing abnormalities of EMG patterns are found among these patients.

In the present study, we examined the recovery of R1 and R2 responses and EMG activity of orbicularis oculi and levator palpebrae muscles in patients with involuntary eyelid closure. The aim of the study was: (1) to evaluate the state of excitability of facial motoneurons and bulbar interneurons in individual patients with a certain EMG abnormality; (2) to see whether there is a correlation...
Materials and methods

Thirty three patients, 22 women and 11 men (mean age 67 (range 37–88) years) with involuntary eyelid closure were studied. Table 1 summarises the clinical data of the patients. At the time of the study, seven patients were already under treatment with botulinum A toxin (Dysport) but in all patients voluntary and reflex activity of the orbicularis oculi could easily be recorded. The control group consisted of 23 subjects (six women and 17 men) with a mean age of 49 (range 18–73) years.

The method of EMG recording from orbicularis oculi and levator palpebrae muscles has been reported previously. 19 20 Blink reflex studies were performed with the subject lying supine. Electrical stimuli were applied to the supraorbital nerve at the supraorbital foramen. Surface recording electrodes were placed over the lower portion of orbicularis oculi ipsilateral to the stimulation site. A Grass stimulator was used to apply constant current pulses with a duration of 0·2 ms. The early and late blink reflexes were evoked with stimulus intensity adjusted to three times the threshold of the R2 response. Subjects were requested to close their eyes gently during stimulation, and responses with artefacts due to involuntary movements were rejected. Paired stimuli (conditioning and test) were delivered at interstimulus intervals of 0·5, 0·3, and 0·21 seconds and six trials were performed at each interval. Between successive trials, a rest period of at least 30 seconds was maintained to avoid habituation of the response. The low pass filter was set at 3 kHz and the high pass filter at 1 Hz (6 dB/oct). The sweep time was kept at 200 ms. All responses were stored digitally on a PDP 11/73 computer. In an off line analysis procedure, performed fully automatically, reflex responses were digitally band pass filtered within a range of more than 100 Hz to minimise DC offsets and slow eye drifts and below 900 Hz to reduce the high frequency noise. The responses were then full wave rectified and the average of six trials was computed for each interstimulus interval. Peak amplitude of R1 was calculated within a window from 10 to 25 ms to avoid stimulation artefact, and that of R2 within a window from 32 to 90 ms. The average rest activity level was established within a window from 150 to 200 ms and subtracted from the average response. For each subject, R1 and R2 recovery values were obtained by calculating the size of the test response as a percentage of the conditioning response at each interstimulus interval.

R1 recovery index was calculated in each subject as the mean value of peak amplitude recovery, obtained at interstimulus intervals of 0·5, 0·3, and 0·21 seconds. The R2 recovery index was calculated in the same way. The upper limit of normal for R1 and R2 was defined as the mean + 2·5 SD.

Table 1 General characteristics of the patients examined

<table>
<thead>
<tr>
<th>Group</th>
<th>No of subjects</th>
<th>Focal dystonia (n)</th>
<th>Mean age (y) (range)</th>
<th>Mean age at onset (y) (range)</th>
<th>Mean duration of illness (y) (range)</th>
<th>Type of dystonia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls Patients</td>
<td>33</td>
<td>22/11</td>
<td>67 (37–88)</td>
<td>57 (25–77)</td>
<td>9 (1–20)</td>
<td></td>
</tr>
</tbody>
</table>

Table 2 Mean values of R1 and R2 recovery indices in patients and controls

<table>
<thead>
<tr>
<th>Group</th>
<th>No of EMG subclasses</th>
<th>No of abnormal R1 indices</th>
<th>Mean R1 index (range)</th>
<th>No of abnormal R2 indices</th>
<th>Mean R2 index (range)</th>
<th>No of abnormal R2 indices</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls Patients:</td>
<td>23</td>
<td>3</td>
<td>78 (36–120)</td>
<td>22 (11–42)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whole group</td>
<td>33</td>
<td>17/13</td>
<td>125 (35–227)***</td>
<td>10†</td>
<td>42 (6–108)***</td>
<td>15</td>
</tr>
<tr>
<td>EMG subclass:</td>
<td>3</td>
<td>10/3/3</td>
<td>161 (73–278)***</td>
<td>5</td>
<td>60 (45–79)**</td>
<td>10</td>
</tr>
<tr>
<td>2</td>
<td>10</td>
<td>3/7/3</td>
<td>118 (35–215)*</td>
<td>5</td>
<td>32 (6–108) NS</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>20</td>
<td>14/6/0</td>
<td>96 (74–107) NS</td>
<td>0</td>
<td>60 (29–37) NS</td>
<td>0</td>
</tr>
</tbody>
</table>

The recovery indices are given in percentages. * p < 0·05; ** p < 0·001 v controls.
†R1 could not be recorded in five patients, two from EMG subclass 1 and three from EMG subclass 2.
discharges in orbicularis oculi, EMG recording in these patients disclosed either episodes of involuntary inhibition of tonic activity of levator palpebrae or a disturbed reciprocal innervation of orbicularis oculi and levator palpebrae. EMG subclass 3 consisted of three patients. Abnormality in these patients was characterised by solely involuntary inhibition of levator palpebrae, with normal activity of orbicularis oculi and normal reciprocal innervation of orbicularis oculi and levator palpebrae muscles. These three patients had, therefore, no blepharospasm.

**BLINK REFLEX FINDINGS**

**Controls**

There was pronounced variability in R1 recovery from one subject to another at different interstimulus intervals. The mean recovery of R1 at the 0-5 second interval was 69% and this increased to 78% at 0-21 seconds. Table 2 shows R1 and R2 recovery indices. The mean value of the R1 recovery index was 78% (range, 36%-120%), and the upper limit of normal was 135%. The R2 recovery curve showed less variability. By contrast with R1, recovery of R2 gradually decreased from 29% at the 0-5 second interval to 15% at 0-21 seconds. The R2 recovery index had a mean value of 22% (range 11%-42%), and the upper limit of normal was 43%.

**Patients**

In five patients, the R1 recovery index could not be calculated because the amplitude of R1 was too low with respect to the noise. Recovery of R1 gradually increased from 117% at the 0-5 second interval to 137% at 0-21 seconds. Table 2 shows R1 and R2 recovery indices for the total patient group and for the three EMG subclasses. The R1 recovery index in the patient group differed significantly from that in the control group (P < 0.001). Recovery of R2 gradually decreased from 50% at the 0-5 second interval to 31% at 0-21 seconds. R2 recovery index in the patient group was significantly higher than that in the control group (P < 0.001).

Considering the individual data, 18 of 28 patients (64%) had a normal R1 recovery index, and 18 of 33 patients (54%) had a normal R2 recovery index (table 2). A significant number of patients with an abnormal R1 recovery index also had an abnormal R2 recovery index (P = 0.01, Yates corrected test).

Considering the data from different EMG subclasses, the R1 recovery index differed statistically from control subjects in EMG subclass 1 (P < 0.001, table 2) and less so in EMG subclass 2 (P < 0.05). The R2 recovery index differed statistically from control subjects only in EMG subclass 1 (P < 0.001). All the patients from EMG subclass 1 had an abnormal R2 recovery index, and 50% of them also had an abnormal R1 recovery index. Dystonia of other cranial or cervical muscles was found significantly more often in EMG subclass 1 (P < 0.01, table 2). Figure 1 shows an example of abnormal R1 and R2 recovery curves in one of the patients from this group. All three patients from EMG subclass 3 had normal R1 and R2 recovery indices, whereas all had associated basal ganglia disease; one had progressive supranuclear palsy, and two had multiple system atrophy. Interestingly, 15 patients (75%) from EMG subclass 2 had a normal R2 recovery index. Figure 2 shows normal R1 and R2 recovery curves in one of the patients from this group.

**Discussion**

We investigated the recovery of R1 and R2 components of the blink reflex in patients with involuntary eyelid closure, as one single group or divided into three subclasses according to the EMG patterns of orbicularis oculi and levator palpebrae muscles. Patients with EMG subclass 1 had involuntary discharges solely in orbicularis oculi muscle. EMG subclass 2 consisted of patients with involuntary discharges in orbicularis oculi, accompanied by either involuntary inhibition of levator palpebrae motor activity, or a disturbed reciprocal innervation of orbicularis oculi and levator palpebrae. In an earlier report, we proposed the term "blepharospasm-plus" to designate thry patients with this type of EMG abnormality. The patients from EMG subclass 3 had solely involuntary inhibition of levator palpebrae,21,22 also known as apraxia of eyelid opening,23 with no clinical or EMG signs of blepharospasm.19,20 We included these patients in the present study because the differentiation between blepharospasm and involuntary levator palpebrae inhibition is not always easy to make clinically and synchronous EMG recording from orbicularis oculi and levator palpebrae is often required.24

In controls, R1 recovery showed some variability between subjects at all three interstimulus intervals. From 0-5 to 0-21 second intervals, R1 recovery showed a gradual enhancement, with relatively less suppression at 0-3 seconds. By contrast, R2 recovery showed almost no interindividual variability and from 0-5 to 0-21 second intervals there was a gradual increase in suppression. Also, in the total patient group the recovery of R1 differed from that of R2. There was often an absolute increase of R1 test response when the interval was shortened from 0-5 to 0-21 seconds, whereas the R2 test response diminished at shorter intervals. Such a difference in the properties of R1 and R2 responses has also been found in other studies.24-26 The same facial nerve motoneurons generate both responses and the final effent pathway is common. Therefore, the difference in the recovery behaviours of R1 and R2 responses, in both the controls and patients, should be caused by the bulbar interneurons. Yet it cannot be entirely explained by the difference in the number of bulbar interneurons through which these responses are relayed, and is probably also dependent on the individual properties of the interneurons, their relation to one another, and the suprasegmental drives on them. A difference in presynaptic...
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Figure 1 (A) Rectified and averaged (6 ×) EMG responses at intervals 0-5, 0-3, and 0-21 seconds between the conditioning and test stimuli in a patient from EMG subclass 1. Recovery curve for R1 (B) and R2 (C) in the same patient. The recovery of R1 and R2 responses are enhanced at all three intervals. ISI = interstimulus interval; t/c ratio = test response/ conditioning response.

inhibition at the primary cutaneous afferent terminals also cannot be excluded.

The enhanced recovery of the R2 response found in the total patient group confirms the results of previous authors. We also found an increased recovery of the R1 response, whereas Tolosa et al noted a normal R1 recovery and the other authors did not report on the features of R1 recovery in patients with blepharospasm. Although these conflicting results and the lack of information on this issue may require further investigation, some features of the recorded blink reflex are in favour of an enhanced excitability of the oligosynaptic pathway through which the R1 response is transmitted. Willer et al reported on the existence of a crossed trigemino-facial connection, and showed that a contralateral R1 response can easily be obtained in healthy subjects when the test stimulus was preceded by an adequate conditioning stimulus. Yet after single stimulation of the supraorbital nerve, Berardelli et al recorded a contralateral R1 response in six of 16 patients with blepharospasm. Based on these data, it seems that at least the crossed oligosynaptic route mediating the contralateral R1 response is hyperactive in some patients with blepharospasm. Therefore, we agree with the conclusion of Tolosa et al that the facial motoneurons themselves are not hyperactive in patients with blepharospasm. We presume instead that the interneurons involved in the generation of both the R1 and R2 responses may be hyperexcitable.

The pathways through which the basal ganglia can modulate the blink reflex are unknown. It seems, however, that the basal ganglia exert an inhibitory influence on the bulbar interneurons, even if it occurs...
indirectly via cortical pathways because lesions of these pathways can suppress the blink reflex.\(^{11,30,31}\) Evinger et al\(^{32}\) showed that apomorphine, a dopamine receptor agonist, and nicotine, which releases dopamine in the striatum, increase the latency of the R2 response without altering the latency of the R1 response. In their recent paper, Basso et al\(^{26}\) showed that partial or even complete lesions of the substantia nigra and ventral tegmental area in rats do not modify the R1 recovery at all, whereas R2 recovery shows pronounced excitability. Accordingly, in patients with Parkinson’s disease R1 recovery is normal,\(^{14,33}\) R2 recovery is enhanced,\(^{14,33,34}\) and R2 latency is shortened.\(^{14}\) Furthermore, patients with Huntington’s disease exhibit a decrease in blink reflex excitability\(^{34,35}\) and an increase in the latency of the R2 response,\(^{34,35}\) comparable with those findings in patients with hemispheric lesions. By contrast, R1 recovery was enhanced in our patients and the previous studies showed that the latency of R2 is normal in patients with blepharospasm.\(^{15-17}\) Based on these findings, two important conclusions can be drawn. Firstly, it is conceivable that dopaminergic activity of at least the substantia nigra may not control the state of activity of facial motoneurons directly or indirectly, through the oligosynaptic reflex pathway, and that the enhanced R1 recovery in our patients as a group should have some other origin. Secondly, it is possible that alterations in basal ganglia activity influence the bulbar interneurons somehow differently in patients with idiopathic blepharospasm from those patients with known diseases of the basal ganglia such as Parkinson’s disease.
A causal relation between hyperexcitability of internuclear neurons and the occurrence of involuntary discharges in orbicularis oculi becomes less evident when we consider the data of R1 and R2 recovery indices in individual patients or in patients from different EMG subclasses. Our results showed a normal R1 recovery index in 64% and an abnormal R1 recovery index in 54% of the total patient group, despite the fact that 30 out of 33 patients had involuntary discharges in orbicularis oculi, whether or not in combination with involuntary levator palpebrae inhibition (EMG subclasses 1 and 2). An abnormal R1 recovery index occurred significantly more often in patients with an abnormal R2 recovery index. An interesting finding was that all patients with blepharospasm alone (EMG subclass 1), had an abnormal R2 recovery index and 50% also had an abnormal R1 recovery index. On the other hand, those patients with involuntary discharges in orbicularis oculi with either involuntary levator palpebrae inhibition or a disturbed reciprocal innervation of orbicularis oculi and levator palpebrae (EMG subclass 2), often had normal R1 and R2 recovery indices. At the time of this study, seven out of 20 patients from EMG subclass 2 were already under treatment with botulinum A toxin. It is unlikely that this influenced the recovery values in these patients, because previous studies showed no difference in the blink reflex excitability in patients with blepharospasm before and after such treatment. Furthermore, it is also unlikely that patients with blepharospasm alone may have had a severe form of orbicularis oculi muscle dystonia, because as noted by Pauletti et al., there is no correlation between the severity of dystonia and the excitability of the blink reflex. The most probable explanation is a difference in underlying pathophysiological mechanisms. There may be a dissociation of the suprasegmental controls resulting in involuntary orbicularis oculi contractions, whether or not accompanied by disturbed levator palpebrae muscle activity, which cannot be examined by simply eliciting the blink reflex. Dissociation between innervation of emotional and voluntary movements of the facial musculature is well recognised in patients with central facial paresis, and withthalamic or pallidal lesions.

Symptomatic blepharospasm may occur in disorders of the basal ganglia but never, to our knowledge, as an isolated abnormality. Conversely, no study has yet provided clear evidence for any pathology of the basal ganglia and their involvement in the generation of involuntary contractions of the eyelids in patients with idiopathic blepharospasm. On the other hand, Siegel et al. reported on cells, located within a portion of the medial reticular formation, which showed discharges in relation to the closing movements of eyelids. The authors mentioned that some of these cells probably project directly to the facial cells and some may relay their output over multisynaptic pathways. In another report, Siegel et al. also proposed that local interaction of these medial reticular formation cell populations may allow synthesis of the simple reflex movements generated in the brain stem. We suggest that besides the abnormal drives exerted by the basal ganglia, disorders of the segmental structures may also contribute to the enhanced excitability of the blink reflex responses and to the generation of blepharospasm. This hypothesis may explain why the recovery of R1 and R2 responses was often enhanced in patients with blepharospasm alone, who also often had dystonia of other cranial muscles without additional clinical features pointing to abnormal basal ganglia activity.

All three patients from EMG subclass 3, with involuntary levator palpebrae inhibition, had normal R1 and R2 recovery indices. These patients also had abnormalities of, among others, the basal ganglia. Therefore, certain disorders of the basal ganglia may influence the tonic activity of levator palpebrae muscle without altering the excitability of the blink reflex. We are unaware of any previous reports on this issue.

In conclusion, the results of the present paper are in agreement with our earlier findings, and provide further evidence that, physiologically, blepharospasm is not a homogeneous disease entity. They indicate that various pathophysiological mechanisms at the suprasegmental, or segmental, level, or both, are involved.

We thank Drs L. Volger and A. Hilgendorf for their help in statistical analysis.

Cervicogenic headache: an early description

Despite the controversies surrounding the clinical sources and pathological basis for cervicogenic headache, this term was used by, and often is attributed to, Otis Sjøastad,1 although Berardelli and Riff had earlier described headache and vertigo, related to cervical arthritis, ascribed to stimulation of the vertebral nerve.2 However, an impressive, but little known description is found in the fourth of 18 lectures given between 1860 and 1862 on Rest and Pain, in John Hilton’s classical text:3

"Suppose a person to complain of pain upon the scalp, it is not very essential to know whether that pain is expressed by the fifth nerve or by the great or small occipital? Thus pain in the anterior and lateral part of the head, which are supplied by the fifth nerve, would suggest that the cause must be some-
Bipolar
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colleagues
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occasion when the claim
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many uncertainties
triphenyltin
regarded
cism
replies:

Gavanagh 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 but speculative. One of the uncertainly
is the remarkably slow though sustained evo-
lution of the signs of change in the nervous
system. While ataxia and blurred vision were
early signs, it was two weeks before he slipped into semicoma in November and he
lay in coma virtually until the beginning of
February. Signs of peripheral neuropathy
developed two months after admission and
progressed for several months more. The pat-
tern of the neuropathy suggested an axonal
mechanism whereas the electrophysiology
gave evidence of myelin loss. Another uncer-
tainty 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 unac-
compounded 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 con-
firmed 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 GAVANAGH

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, 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, bio-
chemistry, immunology, virology, and neuro-
logy) into research on schizophrenia and
bipolar disorder as well as to provide sup-
port 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 con-
stitutes a brief proposal into a pro-
ject, 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 commit-
tee.

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, 4007
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, 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 neu-
rosurgeons 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.

Young neurosurgeons Committee
Department of Neurological Surgery,
University of Florida Medical Center,
PO Box 100265; 1600 SW Archer Road
Gainesville, Florida 32610 USA.

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

Announcement from the British Neu-
ropsychiatry Association: 1996 meetings

The 1996 Winter meeting—a joint meet-
ing 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 pre-
sentation 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 neuro-
development, language, and the presenta-
tion 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 Gurratt, 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, Stone
Lane, Stapleton, Bristol, BS16 1QT.
Telephone: 01179 701212 ext 2925/2929 or
Sue Gurratt at the address given above.

CORRECTIONS

Cataci T, Lenzi GL, Cerbo R, Fieschi
C. Sumatriptan and daily headache. J
Neur Psychiagry 1995;58:308.

The reference to Osborne et al should be

Aramideh M, Eckhof JLA, Bour LJ,
Koehna NHM, Speelman JD, Ongerboer
de Visser BW. Electromyography and
recovery of the blink reflex in involuntary
closed eyelid: a comparative study. J

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