LETTERS

‘Who came with you?’ A diagnostic observation in patients with memory problems

The importance of obtaining collateral history when assessing patients attending the neurology clinic complaining of memory difficulties is well known. Patients developing amnesia in the context of Alzheimer’s disease may underplay their difficulties because of cognitive anosognosia, whereas those with purely subjective memory complaints (the ‘worried well’) may overemphasise difficulties. Memory complaint, preferably corroborated by an informant, is one of the suggested diagnostic criteria of mild cognitive impairment (MCI). Misdiagnosis of memory complaints may occur when no collateral history is available.

For these reasons, all patients referred to my cognitive function clinic are sent, as part of their clinic appointment letter, a request asking them to bring a relative, friend, or carer from whom additional clinical information may be obtained; this is printed in bold type and in a separate paragraph. Despite this, some patients attend the clinic alone. A study was undertaken to measure the diagnostic value of this observation.

As part of an audit of referrals over a 2 year period (September 2002 to August 2004 inclusive), attendance or non-attendance of a relative or friend at each consultation was noted. Diagnosis of dementia was based on DSM-IV criteria, established by clinical interview, neuropsychological assessment and structural neuroimaging. Diagnosis of dementia subtype (Alzheimer’s disease, vascular dementia, dementia with Lewy bodies, frontotemporal dementia) and of MCI followed widely accepted diagnostic criteria. All patients had minimum follow up of 6 months.

Of 183 new referrals seen, 150 (82%; 95% confidence interval [CI] 76 to 88%) followed the written information in the clinic appointment letter and attended with a relative, friend or carer; the remaining 33 (18%; 95% CI 4 to 31%) attended alone. In this cohort, 90 patients were diagnosed with dementia and 93 were not demented; three had MCI. Of the 150 patients accompanied to the clinic, 90 (60%; 95% CI 52 to 68%) had dementia; of the 60 not demented, one had MCI. None of the 33 patients attending alone had dementia, although two had MCI.

Hence, if attending the clinic with a relative, friend, or carer (that is, following the instructions given in the appointment letter) were considered a diagnostic test for dementia, it would have a sensitivity of 100% (95% CI 96 to 100%, Wilson method), specificity of 35% (95% CI 26 to 46%), and positive and negative predictive values of 60% (95% CI 52 to 67%) and 100% (95% CI 95 to 100%) respectively. Positive likelihood ratio was 1.55 (95% CI 1.33 to 1.80, log method), judged unimportant, but negative likelihood ratio (0) was large.

Although not absolute, as those unaccompanied patients with MCI might yet evolve to dementia, the period of follow up for some patients is brief, and clinically established diagnoses may require revision (for example, when neuropathological data become available), these findings nevertheless support the belief that attending the neurology clinic alone, despite written instructions to the contrary, is a robust sign of the absence of dementia.

A J Larner
Cognitive Function Clinic, Walton Centre for Neurology and Neurosurgery, Liverpool, UK

Correspondence to: Dr A J Larner, Cognitive Function Clinic, Walton Centre for Neurology and Neurosurgery, Fazakerley, Liverpool, L9 7LU, UK; a.larner@waltoncentre.nhs.uk
doi: 10.1136/jnnp.2005.068023

Competing interests: none

References

Laryngeal abductor paralysis can be a solitary manifestation of multiple system atrophy

Laryngeal abductor paralysis (LAP) and stridor are well known features that occur in one third of patients with multiple system atrophy (MSA). The pathogenesis of LAP is thought to be crico-arytenoid abductor muscle denervation, although there is a lack of consistent evidence of motor cell loss in the nucleus ambiguus. More recently, dystonia of the laryngeal adductor muscle has also been proposed. LAP/stridor usually occurs in the advanced stages of the disease, and is considered to be a poor prognostic feature. In contrast, some MSA cases have shown LAP initially, with most of these reported by otolaryngology departments. However, there are no systematic surveys as to the extent to which MSA patients initially present with LAP. We describe the result of a survey of 200 MSA patients conducted in a neurology department.

We reviewed the case records of 200 consecutive ‘probable’ MSA patients who met the inclusion and exclusion criteria.[4] They were 119 men and 81 women, mean age 60 years; 29 had the Parkinsonian form (MSA-P) and 171 the cerebellar form (MSA-C). Among these, eight patients (4%) (four MSA-P, four MSA-C) were shown to have stridor as the initial manifestation (table 1). Stridor was the solitary manifestation in six of the patients, though it was combined with minimal laryngeal signs in two of these six patients (inspiratory gasp in one, hoarseness in one) and REM (rapid eye movement)-sleep related behavioural disorder (“night terror”) in one. In the remaining two patients stridor occurred together with bladder dysfunction or gait ataxia. In the former six patients, stridor was followed by bladder dysfunction in four, constipation in three, tremor/akinesia in one, ataxia in one, and postural hypotension in one. The average interval between the development of stridor and these later symptoms and signs was 3.3 years (range 1 to 6). The average interval between stridor and hospital admission was 5.4 years (1–10). In all eight patients, laryngoscopy confirmed that the stridor was caused by LAP. The grade of LAP at the first admission to our hospital (using the grade to Isozaki’s laryngoscopy classification, was moderate (abductory paresis during waking; paradoxical adduction during sleep) in three and severe (complete paralysis) in five. Among these patients, continuous positive airway pressure was introduced in three, laser incision of the vocal fold was carried out in one, and subsequent tracheostomy was necessary in five.

In the cases presented, it proved true that LAP/stridor can be a solitary manifestation of MSA. The interval between LAP/stridor and hospital admission was rather long (on average 5.4 years), suggesting that the progression of LAP was not very rapid in those patients. Although the initial presentation of LAP/stridor was not common (it occurred in only 4% of all MSA patients), it is clinically relevant because patients with LAP/stridor but without obvious neurological symptoms may see general physicians or otolaryngologists first. Laryngeal stridor also occurs because of local inflammation or tumours, or from distant causes that affect the vagal nerves, such as upper thoracic or nasopharyngeal carcinoma. If such conditions have been excluded, central neurological causes should be considered. Co-morbid bladder dysfunction (particularly urinary incontinence and post-voiding residual volume of more than 100 ml), postural and postprandial syncope, parkinsonism, and ataxia are all red flags suggestive of MSA. In our eight patients, bladder dysfunction was an early sign and was chronologically correlated with LAP/stridor; this finding is in line with a previous report. These atypical features for a local pathology suggest that further studies of the brain are necessary to confirm the diagnosis of MSA.

A Uzawa, R Sakakibara, N Tamura, M Asahina, Y Yamanaka, T Uchiyama, T Ito, T Yamamoto, Z Liu, T Hattori
Department of Neurology, Chiba University, 1-8-1 Inohana Chuo-ku, Chiba 260-8670, Japan

Correspondence to: Dr R Uzawa Sakakibara; sakakibara@faculty.chiba-u.jp

www.jnnp.com

J Neurol Neurosurg Psychiatry 2005;76:1739–1752

1739
Table 1  Patients with multiple system atrophy who initially presented with laryngeal abductor paralysis/stridor

<table>
<thead>
<tr>
<th>Patient*</th>
<th>Years from the onset of illness</th>
<th>Laryngoscopy findings†</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1 2 3 4 5 6 7 8 9 10 11 12 13</td>
<td></td>
</tr>
<tr>
<td>1. 62/M</td>
<td>Laryngeal stridor</td>
<td>BD</td>
</tr>
<tr>
<td>MSA-P</td>
<td></td>
<td>Decreased sweating</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Akinesia, cerebellar</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ataxia, PH</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Admission:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>tracheotomy</td>
</tr>
<tr>
<td>2. 74/F</td>
<td>Laryngeal stridor</td>
<td>Constipation,</td>
</tr>
<tr>
<td>MSA-P</td>
<td></td>
<td>BD, PH, akinesia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Admission:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>inspiratory gasp,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>hoarseness</td>
</tr>
<tr>
<td>3. 83/F</td>
<td>Laryngeal stridor, inspiratory</td>
<td>Atoxic gait,</td>
</tr>
<tr>
<td>MSA-C</td>
<td>gait</td>
<td>BD, constipation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PH</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Admission:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Akinesia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Admission:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>tracheotomy</td>
</tr>
<tr>
<td>4. 59/F</td>
<td>Laryngeal stridor, RBD</td>
<td>Ataxic gait</td>
</tr>
<tr>
<td>MSA-C</td>
<td></td>
<td>BD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Admission:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CPAP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt; tracheotomy</td>
</tr>
<tr>
<td>5. 62/M</td>
<td>Laryngeal stridor</td>
<td>Constipation,</td>
</tr>
<tr>
<td>MSA-C</td>
<td></td>
<td>BD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Admission:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ataxic gait,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>akinesia,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>inspiratory gasp,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CPAP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Admission:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>tracheotomy</td>
</tr>
<tr>
<td>6. 57/F</td>
<td>Laryngeal stridor, hoarseness</td>
<td>Hand tremor</td>
</tr>
<tr>
<td>MSA-P</td>
<td></td>
<td>Dysphagia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>admission:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ataxic gait,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>akinesia,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>inspiratory gasp,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CPAP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Admission:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>laser incision</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt; tracheotomy</td>
</tr>
<tr>
<td>7. 53/M</td>
<td>Laryngeal stridor, hoarseness</td>
<td>PH, decreased</td>
</tr>
<tr>
<td>MSA-P</td>
<td></td>
<td>sweating,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ataxic gait</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Admission:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>dysphagia</td>
</tr>
<tr>
<td>8. 63/F</td>
<td>Laryngeal stridor, ataxic gait</td>
<td>Admission:</td>
</tr>
<tr>
<td>MSA-C</td>
<td></td>
<td>hoarseness,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>akinesia;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CPAP</td>
</tr>
</tbody>
</table>

*Age, sex, diagnosis.
†Laryngeal abductor paralysis: + (mild), ++ (moderate), +++ (severe), Isozaki’s classification.

AHI, apnoea hypopnoea index; BD, bladder dysfunction; CPAP, continuous positive airway pressure; F, female; HUT, head up tilt (60˚ for 10 min); M, male; MSA-C, cerebellar form of multiple system atrophy; MSA-P, parkinsonian form of multiple system atrophy; ODI, oxygen desaturation index (dips per hour); PH, postural hypotension; RBD, REM sleep behavioural disorder; SAS, sleep apnoea syndrome.
PostScript

References

Disseminated \textit{Pseudallescheria boydii} infection successfully treated with voriconazole

A 56 year old, right handed African-American man with past history of left knee osteoarthritis, remote intravenous drug use, remote alchoholism, and seropositivity for hepatitis C was admitted to a local hospital for fatigue, chest pain, 13.6 kg weight loss, night sweats, and vision loss. On examination, a loud systolic murmur was present. An electrocardiogram (EGC) displayed T wave alternans and a transoesophageal echocardiogram revealed severe mitral regurgitation with mitral valve vegetations, ruptured chordae tendineae, and left ventricular ejection fraction of 75%. He was diagnosed as having endocarditis and cytomycelial infection in the USA is as mycetoma, a chronic limited subcutaneous infection in immunocompetent individuals engendered by minor trauma, and is characterised by grain formation and local tissue destruction. However, \textit{P. boydii} has recently emerged as an agent of invasive fungal disease as well, a phenomenon linked to the increasing prevalence of immunosuppression in the community. Although endocarditis and endophthalmitis have been described, lung, bone, joint, or central nervous system (CNS) involvement is more typical of this organism. Infections are classically acquired through penetrating trauma or massive inoculation through inhalation, such as may occur in near drowning in stagnant or polluted water. Disease subsequently results from contiguous extension and haematogenous dissemination. It is likely that our patient acquired his infection through prior intravenous drug use, resulting in endocarditis with secondary dissemination to eye, kidney, extremities, and brain.

Among the various types of invasive fungal disease attributable to \textit{P. boydii}, survival rates...
appear to be particularly poor with central nervous system and/or valvular involvement. A relatively recent literature review revealed that of 39 patients with documented CNS disease, only nine were known to have survived; no prior reports exist of survivors of endocarditis. Intrinsinc resistance to amphotericin B, a mainstay in the treatment of most invasive fungal diseases including aspergillosis and mucormycosis, has been reported repeatedly, a trait undoubtedly associated with poor survival in patients in whom the diagnosis is delayed. We describe the first immunocompetent survivor from P. boydii native valve endocarditis complicated by multiple ischaemic and haemorrhagic strokes and peripheral embolisation.

Successful treatment of invasive disease due to P. boydii hinges upon surgical resection with institution of appropriate antifungal therapy. Miconazole, an imidazole derivative used topically for many dermatophyte infections, was previously the treatment of choice in the light of this organism’s resistance to many commonly used systemic antifungals, including amphotericin B and flucanazole. However, its poor CNS penetration, toxicity profile, and unavailability in the USA as an intravenous formulation render it less than desirable. Of the azole antifungals, voriconazole, and particularly itraconazole have been used successfully in treatment of pulmonary pseudallescheriasis, their poor CNS penetration significantly impairs their therapeutic utility in the treatment of brain abscess.

L G Apostolova
Department of Neurology, University of California, Los Angeles, CA, USA

E K Johnson
Saint Luke’s Hospital, Chesterfield, MO, USA

H P Adams Jr
Department of Neurology, University of Iowa, Iowa City, IA, USA

Correspondence to: Dr L G Apostolova, Tichi Willkerson-Kassel Dementia Fellow, Department of Neurology, University of California at Los Angeles, Rex Neurological Research Center 2-218, 710 Westwood Plaza, Los Angeles, CA 90095, USA; lapapostolova@mednet.ucla.edu

The patient described in this letter consented to his details being published

doi: 10.1136/jnnp.2005.067322

Competing interests: none declared

References


Ocular tilt reaction and anterior inferior cerebellar artery syndrome

The ocular tilt reaction (OTR) is an eye-head postural reaction consisting of ipsilateral head and neck tilt, skew deviation, and ocular torsion. OTR indicates either a unilateral peripheral vestibular deficit (inner ear or vestibular nerve) or a unilateral lesion of brain stem pathways from the vestibular nuclei to the interstitial nucleus of Cajal in the rostral midbrain.

The anterior inferior cerebellar artery (AICA) supplies the lateral inferior pontine tegmentum and middle cerebellar peduncle, vestibulocochlear nerve including the root entry zone, inner ear, and anterior inferior cerebellum. Although there has been one report of skew deviation owing to an AICA infarction, the cardinal features of the OTR have not previously been documented. We describe two patients with AICA infarction, each of whom had ipsiversive OTR—one with complete OTR, the other with skew deviation and tonic ipsiversive ocular torsion.

The first was a 58 year old man with long standing hypertension who presented with sudden vertigo and imbalance. On neurological examination, he had bilateral gaze evoked horizontal nystagmus, left peripheral facial weakness and numbness, dysmetria of the left limbs, and gait ataxia. There was no caloric response on the left side. Pure tone audiometry showed 65 dB sensorineural hearing loss on the left side. The subjective visual vertical with binocular viewing was tilted 17 degrees to the left (that is, counterclockwise from the patient’s point of view). Fundus photography showed 25° extension of the left eye and 12° intorsion of the right eye. He had a skew deviation with a right hypertropia of 20 prism diotors in primary gaze (fig 1). Magnetic resonance imaging (MRI) including diffusion images showed acute infarcts in the left middle cerebellar peduncle and the left lateral inferior pontine tegmentum (fig 1).

The second patient was a 58 year old woman with type 2 diabetes mellitus and hypertension who developed severe vertigo, hearing loss, tinnitus on the left side, dysarthria, and imbalance. She had bilateral gaze evoked nystagmus with a horizontal-rotatory component. There was left peripheral facial weakness and numbness, dysmetria of the left limbs, and gait ataxia. Pure tone audiometry showed a 65 dB sensorineural hearing loss on the left side. Fundus photography showed 14° extension of the left eye and 3° extension of the right eye. Prism testing showed a skew deviation with a right hypertropia of 6 dioptr in the primary position. Subjective visual vertical with binocular viewing was tilted 13° to the left (that is, counterclockwise from the patient’s point of view). Caloric response was absent on the left side. MRI showed new infarcts in the left middle cerebellar peduncle, left lateral inferior pontine tegmentum, and anterior inferior cerebellum, possibly including the flocculus. Two months later the subjective visual vertical was normal. Fundus photography

Figure 1 Tonic ocular tilt reaction in patient 1. Note sustained head tilt and concurrent vertical divergence of the eyes (skew deviation). T2 weighted axial magnetic resonance imaging of the brain showed acute infarcts in middle cerebellar peduncle and lateral inferior pontine tegmentum. There is conjugate leftward torsion of the eyes (that is, counterclockwise from the patient’s point of view): a 25° extension of the left eye and a 12° intorsion of the right eye. HT, hypertropia; LT, left; RE, right eye; RT, right. Patient consent was obtained for publication of this figure.
now showed 1° of extorsion of the left eye, indicating that the left eye had been exorted by 13° at the first examination (that is, by 14° minus 1°) and 9° of extorsion of the right eye, indicating that at the first examination the right eye had in fact been intorted by 6° (that is, by 3° minus 9°).

Comment

Most earlier reports of AICA infarction have focused on the brain stem or cerebellar findings. Recently, there have been several reports describing the clinical importance of inner ear symptoms, vertigo and/or sudden deafness. However, a detailed description of OTR as a sign of AICA infarction has not been reported previously. OTR, a sign of vestibular dysfunction in the roll plane, is characterised by a triad of conjugate ocular torsion, skew deviation, and head tilt. It results from destractive or irritative lesions of central or peripheral graviceptive vestibular pathways. Although head tilt is a common component of OTR, skew deviation with conjugate ocular torsion often occurs without head tilt as in our patient. Thus the pathophysiology of a partial OTR (that is, skew deviation and conjugate ocular torsion without head tilt) is the same as that of a complete OTR, and skew deviation with conjugate ocular torsion is sufficient for the diagnosis of OTR.

In addition to lesions of the central and peripheral vestibular pathways conveying graviceptive signals, lesions of the cerebellum may also result in OTR. Skew deviation is commonly seen with cerebellar infarction. Mossman and Halmagyi described two patients with cerebellar infarction. Mossman S, Halmagyi M. Partial ocular tilt reaction due to unilateral cerebellar lesion. Neurology 1997;49:491.

Video assessment of rTMS for Tourette syndrome

In a recent study, subthreshold 1 Hz repetitive transcranial magnetic stimulation (rTMS) over left motor or premotor cortex failed to improve tics in patients with Gilles de la Tourette syndrome (GTS) as determined by self assessment scores.1 However, video ratings of this study had not been analysed. Here, we present the results of blinded analysis of the video of GTS patients who participated in the previous study. We show that rTMS has a placebo effect and confirm that low intensity motor or premotor rTMS does not have a specific effect on tics in GTS.

In a placebo controlled cross-over study of 16 patients with GTS, subthreshold 1 Hz rTMS (2400 stimuli delivered on 2 consecutive days) were applied under three conditions in random order: left motor, left premotor, and left motor sham stimulation. Videotapes were recorded before and after each rTMS intervention in eight patients. One of the authors (AHS) who did not know the patients and was blinded to the treatment conditions, rated the video recordings. Data were analysed using two different rating scales, the Modified Rush Video Scale (MRVS)2 and an “Adapted Yale” Video Scale (AYVS) which was developed for this study. With the MRVS, the following five tic domains are rated from 0 to 4 according to severity: number of body areas involved with tics, motor tic severity, phonic tic severity, frequency of motor tics, and frequency of phonic tics. The sum of the five domain scores provides a total tic impairment score (0–20). As the MRVS does not consistently score the complexity, intensity, and interference of tics, we devised an additional scale using the categories of the Yale Global Tic Severity Scale (YGTSS).3 This new scale, the AYVS, rated the following five domains from 0 to 5 according to severity: number of different tics, frequency of tics, intensity of tics, complexity of tics, and interference of tics. Each domain was rated separately for motor and vocal tics. The sum of the five domains gave a total motor tic score and a total vocal tic score; these scores combined yielded the total tic impairment score (0–50).

Table 1 Mean differences (before and after rTMS) and confidence intervals of clinical scores

<table>
<thead>
<tr>
<th>Score</th>
<th>Motor rTMS</th>
<th>Premotor rTMS</th>
<th>Sham rTMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>MRVS total score</td>
<td>Difference</td>
<td>1.5</td>
<td>1.1</td>
</tr>
<tr>
<td>95% CI</td>
<td>−0.8 to 3.8</td>
<td>−0.5 to 2.8</td>
<td>−1.2 to 2.0</td>
</tr>
<tr>
<td>AYVS total score</td>
<td>Difference</td>
<td>0</td>
<td>0.3</td>
</tr>
<tr>
<td>95% CI</td>
<td>−0.3 to 8.3</td>
<td>0.2 to 4.0</td>
<td>2.6 to 3.1</td>
</tr>
<tr>
<td>AYVS motor score</td>
<td>Difference</td>
<td>1.1</td>
<td>1.4</td>
</tr>
<tr>
<td>95% CI</td>
<td>−1.3 to 3.5</td>
<td>0.5 to 0.9</td>
<td>−0.9 to 2.4</td>
</tr>
<tr>
<td>AYVS vocal score</td>
<td>Difference</td>
<td>1.1</td>
<td>1.4</td>
</tr>
<tr>
<td>95% CI</td>
<td>−0.4 to 3.5</td>
<td>0.4 to 5.3</td>
<td>−1.1 to 2.6</td>
</tr>
</tbody>
</table>

AYVS, Adapted Yale Video Scale; CI, confidence interval; MRVS, Modified Rush Video Scale.

Competing interests: none declared

Acknowledgements

This study was supported by grants of the Oriental Medicine R&D Project (03-PJ9-PG6-S002-0001), Ministry of Health and Welfare, Republic of Korea.

H Lee
Department of Neurology, Keimyung University School of Medicine, 194 Dongsan dong, Daegu, 700-712, South Korea

S-Y Lee
Department of Ophthalmology, Keimyung University School of Medicine

H Lee, S-R Lee
Brain Research Institute, Keimyung University School of Medicine

B-R Park
Department of Physiology, Medicine and Harbong Brain Disease Research Centre, Wookwang University School of Medicine, Iksan, South Korea

References


In conclusion, this is the first report of well-documented OTR with AICA infarction. The ipsiversive OTR in these patients probably resulted from infarction of the inner ear or the root entry zone of the eighth nerve.

www.jnnp.com
Figure 1  Video scores before and after each intervention (means and standard error of the mean). (A) Modified Rush Video Scale (MRVS) total score; (B) Adapted Yale Video Scale (AYVS) total score; (C) AYVS motor score; (D) AYVS vocal score.

appears to be a valid and comprehensive tool to assess tic severity in GTS patients, but it needs to be evaluated further.

We conclude that left motor or premotor low intensity 1 Hz rTMS does not improve tics in GTS patient as assessed by blinded video scoring. Further studies, perhaps using higher intensity rTMS, longer rTMS trains, or bilateral stimulation, are needed to delineate the usefulness of rTMS in GTS patients. In these studies, blinded and independent video rating should be used.

Acknowledgements
The authors thank all patients who participated in this study.

A H Snijders
Sobell Department of Neuropsychology, Institute of Neurology, Queen Square, London, UK

B R Bloom
Department of Neurology, University Medical Centre St Radboud, Nijmegen, The Netherlands

M Orth, J C Rothwell
Sobell Department of Neuropsychology, Institute of Neurology, Queen Square, London, UK

M R Trimble, M M Robertson
Department of Neuropsychiatry, The National Hospital for Neurology and Neurosurgery, London, UK

A Münchau
Sobell Department of Neuropsychology, Institute of Neurology, Queen Square, London, UK

Correspondence to: Dr Alexander Münchau, Department of Neurology, University Medical Centre Hamburg-Eppendorf, Martinistrasse 52, 20246 Hamburg, Germany; muenchau@uke.uni-hamburg.de
doi: 10.1136/jnnp.2004.058321

A Münchau and M Orth were supported by the Tourette Syndrome Association (USA) and the Raymond Way Unit, Institute of Neurology, Queen Square, London, UK. A H Snijders was supported by the Hensenrichting Nederland.

Competing interests: none declared

References

A case of superior cerebellar artery syndrome with contralateral hearing loss at onset

Deafness is rare in ischaemic stroke but sometimes occurs as a result of lower pons infarction. The main cause of such deafness is occlusion of the anterior inferior cerebellar artery (AICA); occlusion of the superior cerebellar artery (SCA), which perfuses the ipsilateral posterior fossa. The latter causes SCA syndrome and also results in deafness but is extremely rare.1 In the present report, we describe a patient with SCA syndrome, whose initial complaint was contralateral hearing loss.

Case report
A 64 year old male with untreated hyperglycaemia and hypertension was admitted 4 h after the sudden onset of deafness in his right ear. Hearing loss was the only complaint and no neurological signs were absent. Right sensorineural hearing loss was revealed by a hearing test (fig 1A). After several examinations including stapedial reflexes and speech discrimination, an initial diagnosis of sudden deafness was made. However, 8 h later the patient complained of diplopia, vertigo, and nausea. Impaired abducens function in the right eye and bilateral lateral gaze nystagmus were observed. Finger-nose and heel-knee tests suggested left cerebellar ataxia. Hence, a diagnosis of SCA syndrome was made, and argatroban, an anti-coagulant, was adminis-
Figure 1 (A) Right sensorineural hearing loss. ‘o’ and ‘X’ indicate air conduction without masking in the right and left ears, respectively. ‘[]’ and ‘[]’ indicate bone conduction with masking in the right and left ears, respectively. (B, C) Axial diffusion weighted MRI images (B, C) 12 h after the onset of deafness. Note high signal intensity is physically separated by the left cerebellum (B, arrow) and left lateral superior pons (C, arrow). (D) Magnetic resonance angiography showing occlusion of the lower basilar artery (arrows). (E) Axial FLAIR showing high intensity areas at both occipital lobes (arrows).
Edited by Reinhard Konig, Peter Heil, Eike Most clinical neurologists scarcely spare a so far, so good. Part II ("Coding of sounds"), the brain. We move on to voices, and speech: fundamental claims about the organisation of is truly a closed book for most clinicians, both neuroscientist, the book provides a state of discussion petrified to the more sober experience while reading it of hearing in my limens it is but a few synapses to Mozart and hallucinations. But by any criterion this is Nevertheless the content of the book and its formative influence on the neurologist's thought may serve as a reminder that the five-sensed cane of mind paraphrase that poem of Kemp's, the in daily life, that the eyes (human eyes, at that) are the sole windows of the brain. To paraphrase that poem of Kemp's, the unguessed glories of the auditory cortex remind us that the five-sensed cane of mind, it is after all, five-sensed.

The Auditory Cortex, A Synthesis of Human and Animal Research


As a general rule, it is safe to assume that any textbook entering a seventh edition does so on its merits and must be worth reading. The latest edition of the classic Mechanism and Management of Headache by Lance and Goadsby does not disappoint. It is occasioned not just by the passage of time but also by advances in the field of headache that make a new edition necessary. Among these, the long-awaited revision of the International Classification of Headache Disorders is the most important, as it has created some entirely new headache entities and significantly altered criteria for others. New information about the structural consequences of seemingly benign headache disorders – iron deposition in the brainstem, an increased prevalence of clinically silent ischaemic brain lesions in migraineurs, for example – and impressive gains in understanding of the basic pathophysiology of headache also demand explication for practising physicians. So too do new "hot topics" such as the possible connection between patent foramen ovale and aura, or the use of nerve stimulators to treat various forms of headache. And finally, the treatment landscape for headache disorders, notably migraine, has changed considerably since publication of the sixth edition: new triptans, the advent of topiramate for prophylaxis, the increasing popularity of preemptive treatments for menstrual migraine, to name just a few.

Readers who turn to this book for up to the minute information about the pathophysiology of headache, recent advances, and the latest treatment recommendations will find it all here, as the book has been thoroughly and thoughtfully updated. That this has been done without sacrificing the time-tested order and the names of many of headache's best and brightest. There is no better testament to the enduring legacy and influence of both authors.

The new edition is slightly smaller than the previous one, and the cover design has been updated. Those are cosmetic changes only, but appreciated nonetheless. Chapter titles and order have not been changed, save for the welcome insertion of a new chapter on chronic daily headache. The book begins with an historical overview of headache that serves as a useful reminder of just how far we have come from the days when holes were drilled in the skull to relieve headache. Throughout the book, photographs have been updated or replaced, and in general are clearer than those in previous editions. Horner's syndrome, third nerve palsy, and other physical findings are nicely and usefully illustrated, and are a true asset to the chapter on examination.

Not unexpectedly, the chapters dealing with headache classification and pathophysiology have undergone the most extensive revision. Updated, timely information about natural treatments has also been added, reflecting the reality of patient interest and enthusiasm for such things. A new table on clinical stratification of acute, specific migraine therapies has been added to the treatment chapter, and other tables in this chapter are more carefully organised, larger, and more readable than those in previous editions. A small oversight is the retention of the former term "tension headache" in many places – perhaps done to save space, but if so at the expense of the subtle but important implications conveyed by the longer term "tension-type."

The new edition of Mechanism and Management of Headache is a well-deserved tribute to the enduring legacy of Lance and Goadsby. It is a welcome update and a true asset to the chapter on examination.

Reference

Oxford handbook of psychiatry


Psychiatry, third edition, Oxford core texts


The Oxford Handbook of Psychiatry is a wonderful little book. The "little" applies to its size...
Volume 76 Assessors

The Editor is grateful to the following, who have assisted in the assessment of papers during the past year.

D Aarsland
G Abbruzzese
K Abe
J Acheson
L Ada
M Adamaszek
C Adler
S Agapejev
Y Agid
N Agrowal
J Y Ahn
T Ala
S Alarowitch
A Albanese
A Alberti
M Alberts
A Al-Chalabi
M P Alexander
J Alsiky
R Allan
S Allder
S Allen
R Al-Shahi
M Altiere
J C Alvarez Uzabeaga
T Alves
A Amato
M Aminoff
P Anand
F Andermann
N Anderson
S Anderson
V Anderson
N Andrews
J Andrich
C Angelini
J C Antoine
A Antonini
M D A S Antonio
P Appelros
D Araujo
F Arbabi
M Arena
A Argyriou
K Arimura
C Armon
E Arnaiz
S Arnadottir
P Arnett
E Arnold
M Arnold
I Arnulf
S Artero
A Aruin
A Arzimanoglou
S Asano
R Asherson
K Ashkan
A Atkinson
C Avezaot
T Aziz
P Azouvi
H S Bachelard
J Baehringer
H Baezner
I Baguley
A Bahra
P Bain
S Bakchine
G Baker
R Bakshi
K Bala
R Balaji
L Balcer
R Baldwin
R W Baloh
O Bandmann
R Barclay-Goddard
F Baringarremerentia
F Barkhof
L Barnes
M Barnes
T Barnes
G Barnett
R J Barohn
J-C Baron
P Barone
W Barr
C Bass
A Bastian
M Bastin
A Bateman
D Bates
R Baumgarner
S Baxendale
G Beaumont
J G Becher
E Beghi
L Beglinger
L Belovlev
B Bell
E Bellissant
E Bellone
H Benamer
O Benavente
M Bendszus
M Benedicic
T Bengner
T Benke
E Ben-Menachen
C Bennett
Y Ben-Shlomo
J Berciano
H Berendse
D Berg
S Berilgen
S Berkovic
G Berlot
G E Berrios
F Besag
G Besson
F Bethoux
S Betmouni
J Bevilaquada
B B Bhakta
K Bhattia
F Biagi
G Jan Biessels
E Bigler
K Bihari
P Bill
M Billiard
G Binetti
A Biondi
A Bizzi
F Blaes
J Bland
R Blankenstein
A Bleasel
S Blecic
K Blennow
O Blin
B Bloem
C Booke

E Joyce

CORRECTION
doi: 10.1136/jnnp.2004.059824corr1
D J Mahad, A Helldén, J Jarvis, et al. Aciclovir induced posterior leucoencephalopathy (J Neurol Neurosurg Psychiatry 2005;76:1308–9). The authors of this letter were mistakenly grouped according to their affiliations. The correct ordering of the authors is: D J Mahad, A Helldén, J Jarvis, D Mitra, A Gholkar, P P Chinnery.
H-C Koennecke
M Koepp
M Koga
M Koltzenburg
D Kondziella
M Kopelman
A D Korczyn
R Korinthenberg
C Kosinski
P Koudstaal
N Kovač
P Krakau
J Kramer
C Krarup
A Kreisler
I Kreitschmann-Andermahr
H Kremer
J Kreutzer
E Krishnamoorthy
P Krystkowiak
J Kulisevsky
D Kullmann
S Kumar
V Kumari
A Kurz
S Kuwabara
M Laddoucheur
P Laloux
M Lambert
A Lammertsma
P Lamont
C Lampi
C Lamy
J Lance
R Lane
D Langdon
G Lange
S Lanius
M Lanteri-Minet
A Larner
V Larrue
E Lartigau
P Lasjaunias
H Lassmann
L Latiní Corazzini
S Laureys
M Lavoie
M C Lawden
S M Lawrie
F Lazaro Perlado
T Le Tourneau
J P Leach
B R F Lecky
X Leclerc
T Ledin
H Lee
K L Leenders
A Leentjes
A Lees
J-P Lefaucheur
C Lefebvre
A Leff
J-M Leger
B Legros
D J Lemmann
J Leigh
P Leigh
J-P Lejeune
T Lempert
G Lennox
F Lenz
G L Lenz
J Leonard
R Lesser
S Lewis
K-K Liao
C Lichy
D Liebeskind
E T Lim
M Lima
P Limousin
K W Lindsay
H Lipton
G Livingston
T Loddenkemper
J Loder
E Loder
G Logroscino
A Lombes
C Loo
O Lopez
N Losseff
S Love
S Lovestone
P Low
C Lubetzki
B Lubicz
C Lucas
J Luchsinger
H Luders
G Lundborg
M Lunn
L M Luxan
T Lynch
R Macdonald
C Machado
M Mackowiak-Cordoliani
P Maddison
A Maertens de Noordhout
O Malik
R A Malik
L Mallet
C L. Mallucci
G Mallucci
F Manes
P Manganotti
H Manji
D M A Mann
R Manni
J Mant
P Maquet
H J Markowitsch
H Markus
L Marsh
F MARTIN
R Martin
P Martinez-Martin
A Martins da Silva
C Martyn
J-L Mas
J Mesdue
S Massengou
C Masson
F Mastaglia
C Masters
D Mataix-Cols
M Mataro
C J Mathias
T Mathiesen
P S Mathuranath
R Matsumoto
A Matsumura
H Matsunaga
P Matthews
H P Mattle
A Mauskopf
A Meyes
T McAllister
A McCadden
D McFarland
N McGregor
C McGuigan
P McGuire
I McKeith
P J McKenna
J G McLeod
T McLellan
E Medeiros de Bustos
N Medford
M M Mehdifarad
J Mellers
A Mendelow
M F Mendez
M Menken
G Meola
R Metz
C Meyer
M Meyer
P Michel
R Mielke
E Mignot
J Mink
P Misra
A Mitchell
J D Mitchell
P Mitchell
M Mizuguchi
H Mizusawa
G Modi
C Monaca
L Montalbetti
J J Moxie
M Moonis
D Moore
A Moosa
T Moreau
R Morecraft
J Morgan
C Morganti-Kossman
K Morgen
J Moriarty
A Morini
C Morris
J Morrow
F Motlaffdy
T Moulin
M Mouradian
K Muir
P Muir
C Mulder
M Mumenthaler
C Mummery
N Munro
M Muñiz
Y Muragaki
R Muri
D Murphy
N Murray
D Na
Y Nagahama
H Nagayama
G Nagels
Z Nagy
D Neary
P Nestor
T Neumann-Haefelin
P Newman
S P Newman
E T Ngan
A Nicolson
N Nighoghossian
C Nimsky
M Nishimura
K Nohara
A Nordberg
J Noseworthy
J Nott
H Nukada
T Nurmi
G Nys
M Oertel
E O'Hearn
M Okun
M Olde Rikkert
A Olivier
T Olsen
D O’Neill
S O’Neill
H Ophenshaw
J Opitz
C Oppenheim
J-M Orogogazo
R W Orrell
M Orth
K Ostergaard
C Ostertag
P O’Sullivan
C Ouellet
J-C Ouellet
O Outterscy
F Ovsiew
A M Owen
L Ozellus
M Paciaroni
C Paolavo
L Padua
M-C Pai
J Palace
C Papayiopoulou
L Panioti
L Pantoni
M Panza
S Pappata
L Parntett
J Parvisi
A Pascaud-Leone
F Pasquier
G Pasquier
M Pasquini
P Patrick
P Patonneri
J M S Pearce
R Peatfield
J Pedersen
E Peitersen
S Peker
J Peltoha
D Perani
Volume 76 Reviewers

The Editor is grateful to the following for reviewing books during the past year.

A Bahra  J Ball  P Boon  R Botell  S Crutch  K Fotopoulou  C Gardner-Thorpe  E W Loder  A Schrag  S Tabrizi  J Warren  T A Yousry

J Ball  A Bahra  P Boon  R Botell  S Crutch  K Fotopoulou  C Gardner-Thorpe  E W Loder  A Schrag  S Tabrizi  J Warren  T A Yousry

www.jnnp.com