BSNO

British Society of Neuro-otology
www.bsno.org.uk

Second Meeting of the British Society of Neuro-Otology

Monday, 28 October, 2002

Leicester University,
Gilbert Murray Hall.

ABSTRACTS

ORAL PRESENTATIONS:

Visual Symptoms in Vestibular Patients
Adolfo M. Bronstein
Academic Department of Neuro-Otology, Imperial College, Charing Cross Hospital, London

In this presentation I will discuss two syndromes with visual components that are seen by clinical neuro-otologists. These are the syndromes of oscillopsia and visual vertigo. Oscillopsia is the illusion of movement of the visual surrounding. If it is related to a patient's head movement the cause is likely to be bilateral absence of vestibular function. If the oscillopsia is triggered by a specific movement, or position of the head, it may be part of a positional vertigo, peripheral or central. If the oscillopsia is fairly continuous it is more likely to be due to a nystagmus resulting from a disorder of the central nervous system (down beat nystagmus, torsional nystagmus, pendular nystagmus, and others). The main treatment is aimed at the underlying condition but in addition specific exercises and medications (Clonazepam, Gabapentin, Carbamazepine) can be useful. The second syndrome, Visual Vertigo, essentially is the sense of unsteadiness or dizziness that certain vestibular patients experience in "busy" visual surroundings such as supermarkets, crowds, traffic, disco lights. Recent research has shown that the symptom of visual vertigo develops in vestibular patients who have increased visual dependence. This visual dependence can be assessed in the laboratory or the clinic by the "rod and frame" test or the "rod and disc" test. A recent trial has demonstrated that the addition of prolonged optokinetic stimuli and tasks involving visual vestibular conflict are clinically helpful for the management of these patients.

The Effect of Round window Catheterisation and Gentamicin infusion in Meniere's disease
Leicester Balance Centre Experience
G.Dhanasekar, J.A.Cook

Background: Meniere's disease is a complex disorder of the inner ear, characterized by aural fullness, fluctuating hearing loss, tinnitus and episodes of vertigo. Round window microcatheter (Durect, Denver, CO, USA), administering gentamicin is a relatively new and exciting treatment for Meniere's disease.
Patients and Methods: Between May 1998 and June 2002, 21 (11 male and 9 female) patients with Meniere's disease underwent Round window catheterization with gentamicin infusion at the Leicester Royal Infirmary. All the patients underwent 10 days of continuous treatment with 5 microlitre/hour of gentamicin (10mg/ml). The results were analysed by the AAOO-HNS (1995) guidelines. The median follow up was 19.5 months.
Results: In 17 of 21 patients (80.9 %) had complete resolution of their vertigo, 3 (14.3 %) have substantial improvement with only infrequent attacks of vertigo. One patient still has rotatory vertigo. The hearing was improved in 2, unaffected in 16 (76.2%) patients and worse in 3. One had a mild deterioration and the other two developed profound sensorineural deafness, but all 3 already had pre-morbid sensorineural hearing loss, 40, 55 and 65dBA respectively. Aural fullness and tinnitus was improved in 65 % of patients.
Conclusions: In our series, we have shown that by delivering gentamicin directly to the round window in a controlled manner with a Round Window microcatheter, we can successfully control the symptoms of vertigo in Meniere's disease without affecting hearing in most patients.

Ill health in vertigo: motion or emotion?
Davies RA*, Premachandra P***, Le Pira A#, Luxon LM*, McKee M**
*Department of Neuro-otology, NHNN, Queen Square, London WC1N 3BG
**Health Services Research Unit, London School of Hygiene and Tropical Medicine
***Department of Audiology, GOSH, London WC1N 3BG
#MSc Student, Institute of Child Health, London WC1N 3BG

Self-report questionnaires are increasingly used to measure outcome in dizzy patients following rehabilitation intervention. However there are few studies which show how general measures of health are impaired as a result of the anxiety and somatisation often associated with vertigo in patients with "poorly compensated vestibular dysfunction" (PCPVD).
In the following study, 114 consecutive patients with a typical history of PCPVD awaiting vestibular testing, completed the Vertigo Symptom Scale (VSS, Yardley et al. 1992), which measures autonomic and

somatisation scores as well as duration and frequency of vertigo, and the Short Form 36 (SF36), a well validated questionnaire which measures eight domains of health.
Results: patients with PCVD had significantly lower scores on all 8 domains of ill health of the SF36 compared to the equivalent age and sex-matched European population.
Patients were then grouped into high and low scorers (50% in each group) for each of the four VSS domains. Factor analysis and linear regression were used to assess the relationship between the two subgroups for each of the 4 domains of the VSS and the eight domains assessed by the SF36. High scores for "autonomic" and "somatisation" were significantly correlated with poor health in 6/8 SF36 domains as compared to high scores of vertigo frequency (3/8), and vertigo duration (0/8).
There was no difference between patients with canal paresis and/or a directional preponderance on vestibular testing on any of the domains on SF36 and those with normal tests
Conclusion: the results confirm that ill health as judged by the SF-36 is poorly correlated with vestibular test findings, and suggest that autonomic and somatisation symptoms associated with PCPVD are more strongly correlated with measures of ill health than duration and frequency of vertigo.

The Effect Of Acute Vertigo On Objective And Subjective Autonomic Measures In Dizzy Patients: A Case Series

Barry M. Seemungal, MRCP [1], Ros Davies, FRCP[2], Peter Rudge, FRCP [2], Michael A. Gresty,PhD[1], Adolfo M. Bronstein, FRCP[1].
[1]Academic Department of Neuro-Otology, Imperial Coll., Charing Cross Hosp., London W6 8RF, [2]National Hosp. for Neurol. and Neurosurg., Queen Sq., London WC1N 3BG.

We report three patients with dizziness and autonomic symptoms in whom questionnaires, blood pressure, pulse and respiration recordings during caloric testing were obtained. Patient 1 had caloric-induced hyperventilation, hypertension and anxiety but no nausea; they improved with vestibular physiotherapy. This finding documents that vestibular symptoms can precipitate panic attacks. Patient 2 had caloric-induced migraine with anxiety and nausea, suggesting that the connection between vertigo and migraine is bi-directional. Patient 3 had an intense caloric-induced nausea yet showed no sympathetic or anxiety response. These cases demonstrate that the responses to vertigo of anxiety, nausea and increased cardio-respiratory output can be mutually exclusive.

Meningo-encephalitis associated with myringitis bullosa haemorrhagica

DC Wild* & PDR Spraggs [+]
*Leicester Royal Infirmary [+]North Hampshire Hospital

An unusual case of a patient presenting with myringitis bullosa haemorrhagica, who subsequently developed meningo-encephalitis is described. Myringitis bullosa haemorrhagica is an acute ear infection characterised by haemorrhagic vesicles in the tympanic membrane. Myringitis bullosa haemorrhagica is a poorly understood disease and its incidence, pathology and aetiology are still unclear. Intracranial complications of otological infections are well recognised, but our literature search showed that a specific association of myringitis bullosa haemorrhagica and meningo-encephalitis has not been described before. The aetiology of myringitis bullosa haemorrhagica is discussed and recommendations for the management of patients with myringitis bullosa haemorrhagica or acute otitis media are made.

Stress, Cortisol levels and Vestibular disorders.

Linda M Luxon, Bronte Holt, Borka Ceranic, Hannah Derry, Mayank Dutia*
The Department of Neuro-otology , The National Hospital for Neurology and Neuro-surgery, Queen Square, London WC1N and
*Department of Biomedical Science, University of Edinburgh, Edinburgh

Psychological factors, such as anxiety or stress may delay recovery from vestibular symptoms in labyrinthine disorders and there is some evidence to suggest that this maybe consequent upon glucocorticoid dependent mechanisms. Moreover, connections between the vestibular system and the hypothalamic-pituitary-adrenal axis suggest that vestibular disorders may affect the HPA system, possible triggering psychological effects and further affecting vestibular compensation.

Salivary cortisol measures before, during and after vestibular stimulation (caloric and rotation testing) were used to investigate the stress response in patients with peripheral vestibular disorders (n = 12), as compared to normal subjects (n = 6). In both groups, diurnal cortisol responses were also assessed using five saliva samples taken over one day, within one week of vestibular testing. Both groups completed vestibular and psychological questionnaires.

Patients had significantly higher anxiety and dizziness handicap inventory scores, with a trend towards increased depression. Statistical analysis of all the stress and diurnal cortisol responses found no significant differences between the patients and normal subjects. However, a group of patients (n=3) with high cortisol responses to vestibular stimulation was identified, demonstrating significantly different stress and diurnal responses, with higher morning cortisol rise and anxiety levels, together with a trend towards higher awakening and 45 minute cortisol levels. Similar findings have been reported in studies of chronically stressed groups.

Thus, while all patients with vestibular disorders experienced higher levels of anxiety and handicap than normal subjects, there is a subgroup of patients with high stress and diurnal cortisol responses, which may be relevant in terms of symptomatic compensation from peripheral vestibular pathology.

Is the Visual Vertical specific to Otolith Function?
M. Pavlou, N. Wijnberg, M.E. Faldon, A.M. Bronstein
Academic Department of Neuro-Otology, Imperial College London

The subjective visual vertical (SVV) is usually considered as a functional measure of otolith mediated verticality perception. In order to investigate the influence of semicircular canal stimulation (SCC) on the SVV, 8 normal subjects were rotated in yaw about an earth-vertical axis, with velocity steps of +/- 90°/s, for 60s. The SVV was assessed by setting a chair-mounted illuminated line to perceived earth vertical in the dark, during both rotation and a post-rotary period. In order to vary the amount of vertical SCC stimulation, 4 different head positions were tested: head normally upright, head tilted 30° forwards (chin down) or backwards from the normal head position, and head tilted as far forwards as possible (approximately 40°). During head normal and head tilted backwards conditions a significant SVV tilt (p<0.01) in the opposite direction to the rotational stimulus was found (e.g. yaw rotation to the right induced SVV tilt of the top of the line to the left). The time constant of decay for the SVV response was found to be approximately 30s. The SVV tilt reversed during the post-rotary response. Rotation with the 30° forward head tilt produced no effect on the SVV. When the head was tilted the furthest possible forwards, the SVV tilt was reversed, compared to the head normal/head backward responses. Correlations between individual canal efficiencies at different angles of head tilt and mean SVV tilts were 0.79 for posterior, 0.34 for anterior and –0.80 for horizontal SCCs. Simultaneous SVV and torsional eye position measurements (3D video-oculography) were highly cross-correlated (0.83) with a mean lead of torsional eye position of 3.6s. The direction of the observed ocular and SVV tilts was in a direction opposite to that of the slow phase of the torsional vestibulo-ocular reflex. Conclusions: 1) The SVV is influenced by SCC stimulation. 2) The modulation in SVV tilt by various head positions indicates that the effect is mediated by the vertical SCC, particularly the posterior SCC. 3) The rotationally induced changes in the SVV are due to changes in torsional eye position. 4) The torsional and SVV changes occur in the 'anticompensatory' (fast phase) direction of vestibular nystagmus. 4) In the clinical context, abnormal tilt of the SVV cannot be considered as an absolute indication of otolith system asymmetry.

The Effect of Endolymphatic Sac excision in Meniere's disease
Leicester Balance Centre Experience
G.Dhanasekar, J.A.Cook

Background: Meniere's disease is a complex disorder of the endolymphatic compartment of the labyrinth, characterized by aural fullness, fluctuating hearing loss, tinnitus and episodes of vertigo. Most patients are initially managed medically together with dietary control and/or vestibular rehabilitation. Surgery is performed when medical treatment fails to control the most debilitating symptom - vertigo. Current surgical

procedures include grommets, labyrinthectomy, endolymphatic sac surgery, selective vestibular neurectomy and intratympanic gentamicin therapy.

Patients and Methods: Removal of the extraosseous portion of the endolymphatic sac without any drainage procedure is one of the treatment options for Meniere's disease. This procedure was performed in the affected ear in 17 (9 male and 8 female) patients with Meniere's disease between October 1995 and July 2002 at the Leicester royal Infirmary. The results were analysed by the AAOO-HNS (1995) guidelines. The median follow up was 48 months.

Results: In 6 of 17 (35.2%) patients there was complete resolution of vertigo, 10 (58.8%) have infrequent attacks of vertigo. One patient still has vertigo. The hearing was improved in 2, unaffected in 11 patients and worse in 4 (3 of whom had pre-existing sensorineural hearing loss). Tinnitus improved in 4 (23.5%) patients. Aural fullness cleared in 24% of patients.

Conclusions: Our results suggest this procedure has reasonable success rates in terminating the attacks of vertigo and a significant reduction in the frequency of the vertiginous episodes. Hearing remained stable in most of the patients.

MRI Screening for acoustic neuroma: An analysis of published protocols.

Rupert Obholzer*, Peter Rea, Jonathan Harcourt
Department of Otorhinolaryngology Head and Neck surgery, Charing Cross Hospital, London

Objective: Patient selection for Magnetic Resonance imaging (MRI) to exclude a cerebellopontine angle (CPA) lesion is complicated by the variable presentation of such lesions. The BAO-HNS recommends that all patients with asymmetrical sensorineural hearing loss be screened to exclude acoustic neuroma without defining what constitutes significant asymmetry. We have assessed the specificity and sensitivity of published selection criteria and compared them with individual and combinations of pure tone thresholds.

Methods: All MRI IAMS requested in the year 2000 were reviewed. The audiograms and symptoms on which the decision to scan were made were reviewed for the 40 'positive' scans and the last 100 negative scans. This information was analysed using a database containing seven published protocols to assess their relative sensitivity and specificity, and compared to analysis of individual and paired pure tone thresholds.
Results: Four of the protocols analysed had a sensitivity of greater than 97%, of these the highest specificity (48%) was found using a threshold of a 15dB interaural difference at two adjacent frequencies. One patient with an acoustic neuroma would not have been scanned by these criteria. This patient was investigated because he developed Trigeminal parasthesia, an indication for investigation irrespective of hearing loss. Unilateral tinnitus was not significantly associated with acoustic neuroma.
Conclusion: Our data support the adoption of a threshold for MRI screening of 15dB interaural difference at two adjacent frequencies in the absence of other indications (e.g. other cranial nerve palsy) or a strong degree of clinical suspicion. Its application to this patient group would have reduced the number of scans performed from 392 to 168 without missing any pathology. More elaborate protocols incorporating tinnitus and vertigo do not increase sensitivity or specificity.

The Immunohistochemical Localisation Of Somatostatin Receptors 1-5 In Acoustic Neuromas

Luke T Condon[1], Michael JC Rogers[1], Daniel Crooks[2], Stephen L Atkin[3], Nicholas D Stafford[1].
Academic departments of : [1]Otolaryngology Head & Neck Surgery, [2]Neuropathology, [3]Endocrinology, Hull Royal Infirmary, East Yorkshire, HU3 2JZ.

Background: Somatostatin is a naturally occurring peptide, which exerts antiproliferative and antiangiogenic effects via a family of 5 membrane-bound receptor subtypes (SSTRs1-5). Somatostatin receptor subtype-specific analogues are in clinical use for endocrine conditions, and are now undergoing clinical trials in a variety of neoplastic conditions.
Methods: Archival samples of 8 acoustic neuromas were dewaxed, microwaved, blocked and incubated with rabbit polyclonal antibodies raised against SSTR 1-5 specific peptides. Signal was amplified with a biotinylated tyramide kit and visualised with the HRP-Strep ABC method using DAB as the enzyme substrate. Staining intensity was graded independently by two observers.

Results: All of the acoustic neuromas examined showed expression of at least one SSTR subtype. Overall receptor 2 was the most prevalent, with intermediate levels of SSTRs 1,3 & 5 and low levels of expression of receptor 4. Strong or moderate Schwann cell expression of SSTR2 was observed in 7/8 cases, whilst all 8 tumours expressed SSTR2 in blood vessels. Receptor expression density was generally higher towards the tumour periphery and diminished towards the centre.
Conclusions: This is the first study to demonstrate the expression of somatostatin receptors in acoustic neuromas. Stimulation by SSTR subtype specific analogues has a potential therapeutic role in inhibiting tumour growth and angiogenesis in acoustic neuromas.
Acknowledgement: L. Helboe, Dept. Neurobiology, H. Lundbeck A/S, Valby, Denmark.

Mosaicism in the neurofibromatoses presenting as unilateral vestibular schwanommas-clinical and diagnostic implications.
C.N.Marnane, G.M. Weiner, P.D.Turpenny*
Department of Otolaryngology and *Department of Clinical Genetics
The Royal Devon and Exeter Hospital

Mosaicism describes the situation in which an individual who arose from a single genetically homogenous zygote has more than one genetically distinct cell line. It arises through mutational errors arising during somatic or cell division.
Mosaicism in the neurofibromatoses may lead to atypical presentations of type 1(NF1) or type2 (NF2) neurofibromatosis, such that only isolated features of each type may occur. A case is described where a patient originally presenting with isolated stigmata suggestive of NF1 went on to develop a unilateral vestibular schwannoma and was subsequently diagnosed as an NF2 mosaic. The diagnosis of a mosaicism of NF2 has important implications in regard to the management, prognosis and genetic counselling of these patients. Indivduals with the mosaic form are less likely to have severe disease and they also have lower offspring recurrence risk than individuals with the non-mosaic form. As a mosaic for NF2 the potential for existing vestibular schwannoma to increase in size or the possibility of developing a contralateral schwannoma is not known, but must be considered when deciding future management. The importance of considering mosaicism in individuals who present with only isolated stigmata of the neurofibromatoses, especially in reference to the risk of developing vestibular schwannomas is emphasised and discussed.

Progressive Hearing Loss And Auditory Neuropathy In Patients With Leber's Hereditary Optic Neuropathy
Borka Ceranic and Linda M. Luxon
Neuro-otology Department,
The National Hospital for Neurology and Neurosurgery
Queen Square,
London WC1N 3BG

Leber's hereditary optic neuropathy (LHON) is a maternally transmitted disease leading to bilateral severe visual loss. It is caused by a mitochondrial DNA mutation, most commonly occurring at nucleotide positions 11778, 3460 and 14484.The association between the characteristic bilateral sequential optic neuropathy and other neurological abnormalities has been suggested in the past, although inconsistently, particularly in cases of LHON and multiple sclerosis-like illness (Harding's disease). Auditory neuropathy, with or without hearing loss, may also be associated with this disease, although, this has not been previously recognised.
Two cases of LHON (point mutation at 11778) will be presented. In the first case, a female patient aged 46, with LHON and a multiple sclerosis-like illness, who developed visual loss in her mid thirties, presented initially with bilateral intermittent tinnitus, and subsequent difficulty in hearing. Two year follow-up indicated a slowly progressive mild sensorineural hearing loss bilaterally. In the second case, a male patient aged 60, with the onset of bilateral severe visual loss at the age of 15, presented with a right-sided severe-profound hearing loss, which developed initially, very gradually over a number years, but, in the last 3-4 years, with more progressive deterioration and fluctuation.
In both cases, auditory findings were consistent with auditory neuropathy and the hearing loss appeared to have a slowly progressive character, vestibular function was unaffected, and, in the first case, the olivocochlear reflex was also preserved. Further studies are required to establish whether this is a consistent association and to what extent the auditory system is affected in this disease.

You Rang M'lord?

Arani Nitkunan, Herman Nees, Ali Al-Memar
Department of Neurology, Atkinson Morley Hospital, Copse Hill, London SW20 0NE

Auditory agnosia is the inability to recognise verbal or non-verbal sounds despite seemingly normal perception, memory, language and general intellectual ability. The lesion responsible for auditory agnosia is due to damage to the bilateral temporal cortices, including the primary auditory cortex or bilateral transverse gyri of Heschl.
We present a 52 year old with a three month history of progressive hearing problems. She was unable to recognise the doorbell although she could hear it. She had difficulties with speech and memory. Her husband noted loss of personality traits. The signs were compatible with auditory agnosia. The MRI revealed lesions in sites that correlated to the symptoms. Brain biopsy confirmed the diagnosis of CJD.
In conclusion we report a confirmed diagnosis of CJD in a patient presenting with auditory agnosia.

Auditory event related potentials in the assessment of auditory processing disorders: a pilot study.

Alki Liasis[1,2], Doris-Eva Bamiou [3,4], Pauline Campbell [3], Stewart Boyd[2], Anthony Towell [2,5]
[1]Department of Ophthalmology, [2]Department of Clinical Neurophysiology, and [3]Audiological Medicine, Great Ormond Street Hospital for Children, [4]Neuro-otology Department, National Hospital for Neurology and Neurosurgery, [5]University of Westminster, London.

The aim of this pilot study was to investigate whether children with a suspected auditory processing disorder (sAPD) in the presence of normal hearing, differ significantly from normal age-matched controls on particular parameters of auditory event-related potentials. We assessed nine children (mean age 9.5 years) in whom the clinical profile and the results in a screening test for auditory processing disorder (SCAN/SCAN-A) suggested the presence of an auditory processing disorder, and nine age -matched normal control subjects, using auditory event-related potentials (AERP) to phonemes /ba/ and /da/. Analysis of the auditory event-related potentials recordings revealed an enlarged P85-120 and attenuated N1 and P2 in all sAPD children compared to controls. We also found significantly increased N1 peak latency, and a larger peak to peak amplitude of the P85-120-N1 and P2-N2 and smaller peak to peak amplitude of the N1-P2 in the sAPD children. Our results indicate that neurophysiological measures may identify a group of children with specific problems suggestive of an auditory processing disorder in the absence of an obvious structural or functional lesion who warrant further study in order to assess whether these findings reflect delayed CNS myelination.

Smoking as a risk factor for noise induced hearing loss

DC Wild*, MJ Brewster[+], AR Banerjee*
Department of Otorhinolaryngology at Leicester Royal Infirmary*, Industrial Diagnostics Ltd[+]

Objective: To analyse the influence of long-term smoking on the vulnerability of the cochlea to noise.
Design: Prospective observational cohort study on noise exposed employees in the brick manufacturing industry, collecting data on hearing thresholds, noise exposure, smoking habits, medical and otological history.
Participants: A study group of smokers and a control group of non-smokers were identified from a population of 227 noise-exposed employees. Inclusion criteria for the study group (n=30) were male gender, employment for 10 years or more at the manufacturing plant with significant noise exposure and a smoking history of 10 years or more with 10 or more cigarettes per day. Male non-smokers with equal work history were included in the control group (n=58). Exclusion criteria were asymmetrical or conductive hearing loss, significant recreational noise exposure, uncontrolled systemic illnesses and history of head injury, chronic middle ear pathology or major ear surgery. Ex-smokers or individuals smoking for less than 10 years were also excluded.
Main outcome measure: Pure tone audiometry.
Results: Both groups had similar mean age and total duration of occupational noise exposure. The mean age corrected hearing thresholds at 3 and 4 kHz in the smoker group were significantly higher than those in the non-smoker group ($p < 0.001$ at 3 kHz and $p<0.05$ at 4 kHz; Wilcoxon Signed Ranks Test). No statistical difference in the hearing thresholds between both groups was found in any other of the tested frequencies (0.5, 1, 2, 6, and 8 kHz).
Conclusion: Long-term smoking increases cochlear vulnerability to noise.

Diagnosis of sensorineural hearing loss through neural network modeling of distortion product otoacoustic emission patterns

Nausica Ziavra,[1] Ioannis Kastanioudakis,[1] Thomas A. Trikalinos[2] Antonios Skevas,[1]
John P.A. Ioannidis, [2,3]
[1]ENT Department, University Hospital of Ioannina and Section on Neurosciences and Sensory Organs, University of Ioannina School of Medicine, Ioannina, Greece; [2]Clinical and Molecular Epidemiology Unit, Department of Hygiene and Epidemiology, University of Ioannina School of Medicine, Ioannina, Greece; [3]Division of Clinical Care Research, Tufts-New England Medical Center, Tufts University School of Medicine, Boston, USA.

Objective: We investigated whether complex modeling with artificial neural networks of distortion product otoacoustic emissions (DPOAE) may achieve an accurate diagnosis for sensorineural hearing loss (SNHL) of cochlear origin.
Materials and Method: 256 ears (90 with SNHL and 166 with normal hearing) belonging to 132 people were evaluated audiologicaly with pure tone audiometry, impedance audiometry, speech audiometry and DPOAE. Ears were split into training (n=176) and validation (n=80) sets. Input variables included gender, age, time-length of the examination, absolute DPOAE intensity at F2 frequency of 593, 937, 1906, 3812, and 6031 Hz, and respective values corrected for noise levels.
Results: In the validation dataset, an aggregate (average) network had an area under the receiver operating characteristic curve of 0.86. The network correctly identified all ears with sudden hearing loss, congenital hearing loss, head trauma, nuclear jaundice and otoxocity, and 2/5 ears with acoustic trauma, but missed all 3 ears with Meniere's disease and 6/8 ears where SNHL was diagnosed purely based on pure tone thresholds on audiometry, but no accompanying clinical findings. For SNHL exceeding 45 dB HL on a pure tone threshold, sensitivity was 83% (15/18).
Conclusion: Neural networks-based analysis of the pattern of DPOAE across a range of frequencies offers a promising approach for the objective diagnosis of moderate and severe SNHL.

Evidence of sub-clinical cochlear deficit in patients recovered from bacterial meningitis with no sensorineural hearing loss.

*Mark N Johnston FRCSEd (ORL-HNS), +Martin Wiselka FRCP & !Mike Mulheran PhD. *Department of Otolaryngology, Queens Medical Centre, Nottingham; +Department of Infection and Tropical Medicine, Leicester Royal Infirmary, & !Medical Research Council, Centre for Mechanism of Human Toxicity, Leicester University.

Objectives: Sensorineural hearing loss (SNHL) of greater than 30 dbHL occurs in upto 30% of patients after acute bacterial meningitis. Do post-bacterial meningitic (PBM) patients with no apparent clinical SNHL have any evidence of subtle sub-clinical cochlear deficit?
Design: Prospective case controlled clinical trial.
Setting: Departments of Otolaryngology in Nottingham and Leicester, UK.
Patients: 58 controls and 20 PBM patients aged between 18 to 38 were screened by a questionnaire and tympanometry to exclude hearing loss attributable to other causes. All participants fell below the 90th centile according to Lutman and Davis UK data sets.[1,2]
Main outcome measures: Standard (0.25-8 kHz) and high frequency (10-16 kHz) pure tone audiometry (PTA) with distortion product otoacoustic emissions (DPOAE'S) at 2, 4 and 6 kHz were measured.
Results: Mean thresholds over the range of standard PTA (analysed independently) for the PBM patient's were significantly elevated ($P<0.05$ to $P<0.001$) between 4-7 dB in both ears above control group values. There was little evidence of significant high frequency threshold elevation (10-16 kHz) with only one frequency reading significance ($p<0.05$). The mean iso-DP values across all frequencies was elevated in the meningitis group, significantly at four of the six frequencies tested ($p<0.05$ to $p<0.01$), the remaining two frequencies $p<0.1$.
Conclusion: PBM patients with hearing below the 90th centile range have a mild but significant sub-clinical threshold elevation over the standard PTA. Threshold elevation may be as a result of the decrease in cochlear outer hair cell function reflected in the significantly reduced DPOAE'S generation.

1. Davis AC. Hearing in adults. London: Whurr publishers Ltd, 1995.
2. Lutman ME, Davis AC. The distribution of hearing threshold levels in the general population aged 18-30 years. Audiology 1994; 33: 327-50.

GUEST LECTURE

Vestibular Neuritis and Benign Positional Vertigo: Anatomic and Physiologic Considerations

Joel A. Goebel
Professor and Vice Chairman of Otolaryngology - Head and Neck Surgery,
Washington University School of Medicine, USA

Vestibular neuritis (VN) is a clinical diagnosis comprising severe vertigo, nausea, vomiting and a distinct absence of significant audiologic or central neurologic symptoms. Known in the literature by a variety of names, VN tends to recover spontaneously without serious sequelae in the majority of cases. In some instances, however, prolonged dysequilibrium, recurrent severe vertigo and brief positional vertigo attacks are described. Current theory implicates a viral ganglionitis as the cause with herpes simplex virus- 2 (HSV-2) as the putative infectious agent. A predilection for superior vestibular nerve damage with sparing of posterior semicircular canal function is seen in both histological and physiological studies. Indeed, this selectivity is curious and to date remains somewhat a mystery. However, recent anatomical studies have revealed consistent differences in anatomy between the branches of the vestibular nerve which might help explain the bias towards superior nerve injury and subsequent development of benign positional vertigo (BPV). In this presentation, we will explore the historical evidence regarding VN and propose a plausible anatomical explanation for the sparing of inferior vestibular nerve function and appearance of BPV in a percentage of cases. Implications for treatment options will also be addressed.

.....

POSTER PRESENTATIONS

Simulating Space Sickness in the European Space Agency (ESA) Zero-G Aircraft: Zero-G flights in March 2002

John F. Golding (1), Claude Gaudeau (2), Yves Lucas (3)
(1) Dept. Psychology, 309 Regent Street, University of Westminster, London W1B 2UW email: goldinj@wmin.ac.uk ; (2) Soc. Bio-Informatiques, Univ. Tours, France ; (3) Inst. Univ. Technologie, Bourges, France.

Background. Space sickness is a form of motion sickness which affects approximately 70 % of novice astronauts and cosmonauts to some degree in the first 24-48 hours of a mission. It results in loss of performance and can pose potentially lethal hazards if it occurs to the extent of vomiting in a space suit outside of a spacecraft. Aims (A) to examine the evolution of behavioural + physiological responses and investigate predictors of resistance to motion sickness during Zero-G flights; (B) perform a motion sickness survey on first time flyers. Methods & Interim Results (A) Three flights were performed with 3 subjects per flight in the ESA / Novespace Zero-G plane. Each flight exposed subjects to 31 parabolas during which periods of weightlessness were experienced. Behavioural measures included self-report checklists, inflight observation & video recordings, physiological measures included electrogastrogram (EGG), electocardiogram (ECG), sweating skin conductance (SC) & salivary cortisol. Some extra cortisol samples were taken from other zero-g volunteers. These results are currently under analysis. (B) In addition, an opportunity survey was performed on first time zero-g flyers. Self-report questionnaires of first flight exposure experiences were obtained from n=23 participants. Their intrinsic motion sickness susceptibility (mean percentile score 29) was less ($P<0.001$) than that observed in the general population (mean percentile score 50 by definition) on a standardized susceptibility questionnaire, probably indicating self-selection by volunteers for zero-g flights. Despite this low susceptibility and the use of antiemetics by the majority, one third of the subjects had nausea at some stage on their first flight and five vomited. Although those first time flyers medicated (n=17) with antiemetic (Scopdex) experienced significantly ($P<0.05$) less nausea (mean scale score 2.2) than those medication-free (n=6) (mean scale score 4.4), this protection was far from complete with 3/17 versus 2/6 vomiting respectively. Informal questioning suggested that those individuals who performed further flights appeared to acquire some degree of protection by habituation. Conclusions The snapshot survey illustrates that pharmacological protection is far from 100% complete even in intrinsically less motion sickness susceptible individuals. The main results when analysis is completed should contribute to the development of non-pharmacological countermeasures to space

sickness, including personnel selection and biofeedback systems to speed natural adaptation to the nauseogenic aspects of microgravity.

Glomus Tympanicum, Persistent Stapedial Artery and Dehiscent Facial Canal

Sudhakiran Kalavagunta MS FRCSGlasg, FRCSEng (Oto).
Off - E N T Department, University Hospital Aintree, Longmoor Lane, Liverpool, L9 7AL.

A 75-year-old man presented to the E N T clinic with complaints of left-sided deafness & pulsatile tinnitus since 6 months. ENT examination revealed a reddish hue of the left tympanic membrane. Rest of the ENT examination was normal. Audiogram revealed a conductive hearing loss on the left side with a 20-dB air-bone gap. MR scan revealed a soft tissue mass filling the left middle ear cavity. This mass was enhancing and appeared to be vascular in origin. A provisional diagnosis of glomus tympanicum was made. Pre-operative angiography revealed a 'tumour blush' and the main feeder was the stapedial artery, a branch of the occipital artery. This was embolised. Intra-operatively a reddish soft tissue mass was seen filling the entire middle ear cavity and engulfing all the ossicles. A persistent stapedial artery was found entering into the tumour mass and the tympanic segment of the facial nerve canal adjacent to the oval window was found to be dehiscent. The tumour was excised and the biopsy confirmed it to be a glomus tumour.
Conclusion: This case highlights two important facts. Persistent stapedial artery represents an arrest in middle ear development and is frequently associated with a variety of middle ear defects. Dehiscent facial canal and glomus tympanicum are the anomalies found in this case described. A neuro-otologist dealing with these tumours should be well aware of the association of the glomus tumour with the persistent stapedial artery and the implications of finding this embryological marker.
References
* Arch Otolaryngol Head Neck Surg 1990 Jul;116(7):852-4 Persistent stapedial artery supplying a glomus tympanicum tumor. Boscia R, Knox RD, Adkins WY, Holgate RC.
* Acta Otorrinolaringol Esp 1996 Nov-Dec;47(6):471-3 [Tympanomastoid glomus tumor and persistent stapedial artery][Article in Spanish] Arellano B, Ramirez-Camacho R, Pinilla MT, Garcia Berrocal JR.

Contralateral Benign Positional Vertigo. A complication of treatment

D. Golding-Wood, Bromley Hospitals NHS Trust

Objective: Outcome measure of benign positional vertigo treated by Epley manoeuvre.
Method: Case series analysis of consecutive patients with posterior canal BPV.
Results: A series of 124 patients with clinically diagnosed benign positional vertigo is reported (27 men and 97 women age ranging from 24 to 86 years with symptom duration ranging from 4 weeks to 12 years). All patients demonstrated fatiguing short duration dependent torsional nystagmus on Hallpike testing and were treated by Epley manoeuvre. Within this consecutive series treated by the senior author from December 1996 to December 2001 there were 8 patients who failed to respond to conservative treatment and 19 patients whose symptoms recurred between 2 weeks and 2 years after first treatment that responded to repeat Epley manoeuvre. Of the 19 patients with relapsing symptoms 4 (3%) showed contralateral symptoms with positive opposite Hallpike tests on review 2 weeks after initial treatment. The remaining 97 (78%) patients remained well without further symptoms for a minimum follow up period of 4 months after only one treatment.
Conclusions: Benign Positional Vertigo is successfully treated by Epley Manoeuvre.
A significant proportion of patients relapse following successful treatment. That the site of symptoms may easily be converted to another semi-circular canal of the same labyrinth is well recognised. Conversion of symptoms to the contralateral ear is less well known and we have not found previous reports of this phenomenon. The manoeuvre to successfully treat a given labyrinth must impart the reverse process to the unaffected side. An Epley manoeuvre applied to a given symptomatic ear will necessarily displace otoconia in an asymptomatic ear to the posterior semicircular canal introducing symptoms in the other ear.
This series shows a previously unreported number of induced contralateral BPV following Epley Manoeuvre that is in turn successfully treated after an interval of two weeks from the first treatment.
References:
Recurrence of symptoms following treatment of posterior semicircular canal benign positional paroxyamsl vertigo with a particle repositioning manoeuvre.
Beynon GJ, Baguley DM and da Cruz MJ Journal of Otolaryngology 29:2-6, 2000.
Complications of the canalith repositioning procedure.
Herdman SJ and Tusa RJ. wArchives of Otolaryngology - Head and Neck Surgery 122: 282-286, 1966

Hearing assessment of clinically unaffected individuals with the mitochondrial mutation for maternally inherited diabetes and deafness (MIDD).

Doris-Eva Bamiou, Pamela Rath, Sharon Jenkins, Alan Bird, Borka Ceranic, Linda Luxon.

Maternally inherited diabetes and deafness (MIDD) is a mitochondrial disease with a variable clinical presentation. Hearing loss in MIDD is progressive and is reported to be of cochlear origin. In most studies on MIDD, patients were recruited for the study based on the presence of diabetes and deafness, which may exclude a large percentage of patients with the mutation.
Auditory function was assessed in 11 clinically unaffected individuals with the mitochondrial mutation for maternally inherited diabetes and deafness and in 3 probands for the disease. We found evidence of a cochlear hearing loss of varying degree in all probands, but also in the majority of unaffected individuals with the MIDD mutation. While the probands had significantly worse hearing, than their unaffected relatives who harboured the mutation, we found that the unaffected individuals with the MIDD mutation had raised audiometric thresholds and poorer otoacoustic emissions than age matched normal controls.
Our findings highlight the difference between the audiometric findings in a disorder of mitochondrial inheritance, in which "clinically unaffected" family members with the mutation are quite strikingly abnormal in terms of audiometric tests, while in autosomal recessive hearing loss, unaffected, but obligate carriers of the mutation usually show very subtle, if any audiometric abnormality. The need for audiometric screening of all individuals with the MIDD mutation is, therefore, emphasised.

Distortion product otoacoustic emissions. Multiple to single tone pairs presentation method in adults

Ioannis Kastanioudakis MD[1] Assistant Professor of Otolaryngology
Nausica Ziavra MD[1] Otolaryngologist
Dimitrios Anastasopoulos[2] Associate Professor of Physiology
Antonios Skevas[1] Professor of Otolaryngology
[1]ENT Department, School of Medicine, University of Ioannina, Ioannina, Greece; [2]Department of Physiology, School of Nursing, University of Athens, Athens, Greece.

Objective: The reduction of the examination time of the Distortion Product Otoacoustic Emissions (DPOAE)
Materials and Method:
We investigated the applicability of the multiple-tone pairs method by comparing the DPOAE evoked by a combination of 4 and 3- tone pairs (Sim) with the conventional 1-tone pair (Seq) in 116 ears with normal hearing (NH) and 86 ears with sensorineural hearing loss (SNHL) of human adults.
Results: We found that: 1) DPOAE amplitudes in the NH group, obtained with the Sim condition were strongly correlated with those of Seq. 2) DPOAE amplitude and noise level mean values, obtained with Seq condition, were significantly higher than those obtained with Sim. 3) DPOAE amplitude was independent of the stimulation side. 4) Both conditions can equally distinguish ears with NH and low degree of SNHL from those with higher degrees of SNHL. 5) Examination time was shorter with Sim condition in both groups.
Conclusions: In conclusion Sim condition produces results similar with those of Seq. Despite this, a significant difference between the two methods according DPOAE amplitudes and noise levels was found. For this reason, data from normal individuals on both DPOAE amplitude and noise level are required to improve the sensitivity of the Sim technique. The mean examination time in both groups (NH and SNHL) was shorter with the Sim condition and for this reason Sim condition seems to be more suitable when is used as an auditory screening test.

Pharmacological Protection Of Auditory Function From Noise With Lamotrigine

D Selvadurai[1], S Etheridge[2], J.A Cook[1] & M Mulheran[3].

[1]Department of ENT Surgery Leicester Royal Infirmary, [2]GlaxoSmithKline, Harlow and [3]MRC Centre for Toxicology, Leicester University

Pharmacological protection against noise induced hearing loss (NIHL) is an exciting prospect for inner ear medicine. However, the potential for adverse reactions make it necessary to administer potential

neuroprotectants either through a cochleostomy or onto the round window (in vivo) limiting future clinical uses. Lamotrigine (LTG) is a "use dependent" sodium channel blocker, known to be orally active as an anti-epileptic agent, with a well-defined safety profile in clinical use. The aim of this experiment was to see whether LTG, administered orally, could provide protection against NIHL in the albino guinea pig.

Four groups were studied; control (n=9) and noise control (n=7) receiving 100dB freefield bandpass noise (4-16KHz) for 15 minutes. The remaining two groups received 20mg/kg LTG po (n=11) or LTG co-exposed with noise (n=7). Approximately 2 hours after LTG dosing, as determined by pharmacokinetic data, ECochG threshold recordings were determined pre and post treatment at 8, 16, 24 and 30 KHz using the Round Window E Cath (Durect Inc. USA) implanted through a trans-bulla approach. Control threshold recordings were obtained over 60 minutes. Threshold shifts from pre-treatment were analysed (Student's t-test).

Mean ((se) control thresholds remained very stable (20±6.1, 10.1±1.5, 6.4±1.0 and 15±1.0dB SPL (8, 16, 24 and 30KHz) and were comparable to the LTG group indicating no significant effect on auditory function. Exposure to noise caused increases in thresholds but compared to the noise/LTG group, the mean threshold change at 8KHz was significantly reduced (25.7 vs. 16.6dBSPL, $p<0.05$). Threshold differences were also observed at 16KHz, but did not attain statistical significance (27.0 vs. 17.4dB SPL $p<0.11$). No differences were seen at 24 and 30KHz.

Lamotrigine is believed to regulate neuronal firing in neurones undergoing bursts or persistently high frequency firing whilst having little action on normal low frequency synaptic transmission. Data from this study suggest that this is also the case in the auditory nerve. Orally administered Lamotrigine may therefore provide partial protection against NIHL, suggesting that this class of compound may find clinical applications in protection against NIHL or cochlear excitotoxicity more generally.

An accelerated model of acute NIHL: the effects of 100 dB SPL bandpass noise in combination 12% hypoxia on mammalian cochlear function.

M Mulheran[1], S Etheridge[2], D Selvadurai[3], J Cook [3] and P Jones[2].
[1] MRC Toxicology Unit , University of Leicester, Leicester, UK.
[2] GlaxoWellcome, Ware, Herts, UK,
[3] Department of ENT Surgery,Leicester Royal Infirmary, Leicester UK.

Introduction. Noise levels in excess of 90 dB SPL are well recognised as a cochlear insult. However, the sites and mechanisms underlying the resulting deficit are still being determined, with current consensus being that there are physical, biochemical and excitotoxic components. The aim of this study was to provide a robust acute model of accelerated noise damage with moderate noise levels in combination with marginal hypoxia. In particular, the aim was to develop a model with an enhanced excitotoxic component. This was achieved by reducing the likelihood of physical damage by using moderate broadband noise. Marginal hypoxia was employed to compromise the re-uptake the cochlear excitatory neurotransmitter glutamate.

Methods: Anaesthetised albino guinea pigs were implanted with cochlear recording electrodes via a trans-bulla approach. Systemic monitoring of heart rate, core temperature, respiratory rate and blood O2 saturation was carried out. Closed field acoustic stimuli were delivered using a reverse driven Bruel & Kjaer 4134. Cochlear compound action potentials (CAP) thresholds were determined at 8,16, 24 and 30 KHz at t=0, 15, 30 and 45 min. Animals were divided into four groups: Group 1 Control; Group 2 exposed to 15 mins hypoxia (12% atmospheric O2) at t=15 min; Group 3 exposed to 15 mins 100 dB SPL bandpass (4-16 kHz) noise at t=15 min; Group 4 treatment combination of Groups 2 and 3. Repeated measures ANOVA was used to analyse the data.

Results: No significant changes in threshold were seen in Group 1 or 2 over the recording period. During hypoxia, O2 saturation was estimated to fall between 75-80%, whilst other systemic function remained within normal limits. In Group 3, post noise mean threshold elevations at 8, 16, 24 and 30 were 25, 26, 12 and 9 dB respectively. These elevations were significantly ($p<0.001$)above Control values at 8, 16 and 24 kHz.

In Group 4, post noise/hypoxia threshold elevations at these same frequencies were 42, 38, 32, 19 dB respectively. These elevations were significantly ($p<0.001$) elevated above both Group 1 control and Group 3 noise alone thresholds. The greatest relative shift was 20 dB at 24 kHz.

Conclusion: This model shows that substantial acute threshold elevation in the albino guinea pig can occur with moderate noise exposure alone. Moreover, this elevation can be profoundly synergised using marginal hypoxia. This synergy is particularly marked at frequencies (24-30kHz) experiencing less severe noise exposure. The mechanism is highly likely to involve an increase in excitotoxicity due to compromised re-uptake of glutamate. Secondary damage due to compromised metabolic function in the cochlear nerve is also likely to be involved (see Selvadeurai et al. this meeting).

A Mismatch Between Visual And Vestibular Derived Displacement Alters Perception Of Motion Duration .
Barry M. Seemungal,, Silvia Buenning, Adolfo M. Bronstein, Michael A. Gresty,.
Academic Department of Neuro-Otology, Imperial Coll., Charing Cross Hospital, London W6 8RF.

We hypothesised that since displacement may be derived by the time integral of the vestibular signal, then an observed visual displacement different from that expected on the basis of internal estimates of duration and velocity of motion, could affect the perception of these estimates.
Methods: 18 Subjects sat on a motorised rotating chair in which vision of the surrounding drum was allowed only at the beginning and end of motion. The drum was decorated with images separated by 22.5°. Unknown to the subjects, during some chair rotations the curtain was rotated. After each rotation, and prior to vision of the drum being allowed, subjects indicated their perceived position with an analogue indicator. Following a pair of rotations, subjects indicated if the first movement was longer or shorter in duration than the second. A pair of movements (raised cosine velocities of peak 20-90°/s, duration 2-7s, and displacement 30°-180°) could either be of equal duration or differ by (1 1, 2, 3 or 4 seconds presented in balanced unpredictable sequences. There were 20 _018baseline_019 trials where the drum was stationary. On 20 of 40 subsequent trials, the drum rotated during one of the subject movements by amplitudes up to 100% of those of the chair. Thus, e.g. a drum rotation in the same direction as the subject was intended to make the rotation seem shorter in duration since when the light came on the subject would see that he had apparently made a small angular displacement.
Findings: The probability of subjects making a correct comparison of duration of baseline rotations was 0.9. With drum rotations, the probability of making correct comparisons of rotation durations were 0.1 for 1s ($p < 0.0001$) and 0.82 for 3s ($p=0.014$); i.e. shorter durations were made to appear longer by false feedback of displacement and vice versa for time differences up to 3s (but not for 4s which was not different from control). Subjects developed no suspicions that the drum rotated and made the required comparisons with ease.
Comment: That static visual information can recalibrate the internal perception of time, vestibularly-derived displacement and peak acceleration, has not been previously shown in humans (static visual cues recalibrate rat head direction cells). The perception of small differences between movement durations may be influenced by visual recalibration of the already integrated vestibular signal. This influence may compete probabilistically with other systems that generate an internal estimate of time.

Managing Motion Sickness Without Medication
Yen Pik Sang F [1], Billar JP [2], Golding JF [2], Ziavra N [1], Bronstein A [1], Gresty M [1]
[1] Academic Dept of Neuro-Otology, Imperial College Faculty of Medicine, London
[2] Dept Psychology, University of Westminster, London

Background: Data on the efficacy of non-pharmacological countermeasures for motion sickness (MS) mostly relates to astronauts, pilots and military personnel working in extreme motion environments. Efforts to develop simple behavioural methods of managing disorientation and malaise evoked by quotidian motion for the 'ordinary traveller' have been minimal yet they would be of value, not least because of the side effects of anti-emetics. Accordingly, we report the results of a series of experiments which examined a range of non-pharmacological methods for alleviating MS in healthy individuals, using controlled, within subjects, counterbalanced, cross over trials.
The behavioural measures investigated with the rationale for their selection were:
1] Effect of controlled breathing. Controlled breathing has been reported both in a comparison group trial and anecdotally by pilots, to reduce MS.
2] Listening to a commercially available anti-motion sickness audio-tape of music (Travelwell). Claimed by the company to be an effective counter to MS.
3] Breathing supplemental oxygen. Shown in a comparison group trial to reduce nausea and vomiting attributed to MS in patients during ambulance transport.

Methods: In Exp 1, 12 subjects rated their well being on a 4 point sickness scale whilst undergoing pitch visual-vestibular conflict stimulation (oscillating in pitch whilst viewing the image of an external visual scene which oscillated in counter-phase). Once mild nausea (rating 3) was achieved subjects were asked to employ either controlled breathing or a counting task. Motion was stopped when subjects developed moderate nausea (rating 4) or 30 minutes elapsed, whichever occurred first. In Exp 2, cross-coupled motion (head movements in pitch and roll made during incremental steps in rotational velocity) was used to provoke sickness. At nausea rating 3, 24 subjects either commenced controlled breathing or initiated the Travelwell tape or did nothing (control). In Exp 3, 20 subjects were exposed to off-vertical-axis-rotation (OVAR) and commenced breathing 100% oxygen versus air at nausea level 3. In each experiment, tolerance was defined as the duration of exposure necessary to evoke moderate nausea and the duration of mild nausea.

Results: Exp 1: The mean motion exposure time required to produce moderate nausea was significantly longer ($p<.009$) with controlled breathing (21.3 ± 9.6 min) versus counting (15.10 ± 10.6 min). With controlled breathing tolerance to mild nausea was increased by nearly 70%. Exp 2: Cross coupled motions induced sickness faster under control (9.2 ± 5.9 min) compared with controlled breathing (10.7 ± 5.6 min, $p<.01$) and Travelwell (10.4 ± 5.6 min, $p<.01$). MS was defended against equally well with controlled breathing and with Travelwell. Exp 3: There was no significant difference between breathing 100% oxygen (11.55 ± 7.79 min) or air (10.7 ± 8.26 min) on prolonging tolerance to OVAR challenges.

Conclusion: None of these techniques are any more effective in increasing tolerance to motion than one would expect of "distraction". In comparison with standard doses of oral Scopolamine, the "industry standard" for managing MS, these non-pharmacological countermeasures are only half as effective as drugs.

Short-term plastic changes of the cervico-ocular reflex after optokinetic stimulation

D. Mandellos and D. Anastasopoulos,
Department of Physiology, School of Nursing, University of Athens

Neck-proprioceptive stimulation in the horizontal plane induces weak eye movements (cervico-ocular reflex, COR) in humans, first described by Barany. While these movements are small and variable in normal adults, they become functionally relevant in patients with absent labyrinthine function, contributing to gaze stabilization during head movements. However, the mechanism of COR gain enhancement in the compensatory direction is not clear.
We studied the horizontal COR during passive trunk oscillations of (16 deg under the stationary head at 0.1 Hz, before and immediately after combined neck-proprioceptive and optokinetic stimulation for 45 min. During the adaptation procedure, subjects 'stared' at an optokinetic pattern moving in-phase with their trunk, while their head was stationary in space. This stimulus combination was thought to simulate the pattern of visual and proprioceptive input entering the CNS, when the head of patients without vestibular function is passively moved.
In all 6 normal subjects (aged 24-30 years) the gain and phase of COR were not consistently modified after the adaptation procedure (between -0.4 and +0.12, -94 and -25 respectively, i.e in the 'anticompensatory' direction). Whereas the COR in 2 patients with insulin-dependent diabetes mellitus (VOR gain between 0.35 and 0.44) and one with unilateral canal paresis after vestibular neuritis (VOR gain 0.65) did not differ from the normals before adaptation, a significant gain increase (0.36-0.42) and phase modulation in the compensatory direction was observed thereafter. Our results indicate that visual information can modify the COR in less than an hour, only when the vestibular input is, at least partially, reduced.