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ABSTRACT BOOKLET

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Guest Lecture:

VESTIBULAR COMPENSATION
The Brain Orchestration of Neurobiological and Behavioural Melodies
Implications for Pharmacotherapy and Rehabilitation

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On the basis of our experimental works in animal models and clinical investigations in vestibular defective patients, we propose the concept of *"brain orchestration of neurobiological and behavioural melodies"* as a basis for better understanding the vestibular compensation process. And we will show the clinical implications of this concept for both pharmacotherapy and rehabilitation of patients with vestibular loss.

The vestibular syndrome seen after unilateral vestibular loss (oculomotor, posturo-locomotor and perceptive deficits) is made of both static (at rest) and dynamic (during head/body motion in space) deficits. The static functions are fully recovered with time due to plastic events occurring in the vestibular nuclei (VN). In contrast, the dynamic deficits remain poorly compensated, but they are restored by means of substitution processes and vicarious strategies including the whole brain, that show inter-individual differences.

First key concept: the recovery mechanisms responsible for the compensation of the static deficits constitute a "deafferentation code", i.e., a complex set of molecular and cellular events re-expressed in the VN. These plastic events are reactive signatures that can be seen as "neurobiological melodies" orchestrated by the brain. Interestingly, we have demonstrated that these melodies depend on the real nature of the VN deafferentation using three different animal models of vestibular lesion. Depending on the vestibular loss characteristics (sudden, acute and total loss *versus* progressive, partial and/or reversible loss), i.e., on the different aetiologies encountered in vestibular pathology (vestibular neuritis, Menière's disease, ototoxicity, ageing process,...), the orchestration may be drastically changed and different melodies will be played. This has direct implications for pharmacotherapy, for drug treatment of vertigo and dizziness.

Second key concept: For a given vestibular pathology, the strategies and substitution processes underlying the recovery of the dynamic vestibular functions show a great inter-individual variability and different degrees of complexity. Such vicariant idiosyncratic strategies have been evidenced for the compensation of the vestibulo-ocular reflex and posture control in vestibular loss patients. They can be seen as "behavioural melodies" orchestrated by the brain and depending on the patients themselves. Some of them are very simple strategies, not really adapted to daylife situations, while others are more sophisticated and better adapted to the environmental constraints and ecologic conditions. One of the major goal for the physiotherapist is therefore to help the patient to switch from his/her maladaptive strategy to a more appropriate one on the basis of an early and active motivating rehabilitation program. Rehabilitation must be adapted to the patient's profile (age, professional activity, perceptive style,...), done in changing environments, and addressing interactively all the relevant sensorimotor cues. The notions of habituation *versus* adaptation are discussed also.

AuD and Aud-iers; the USA – UK connection

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The "invention" of the 'AuD" more than ten years ago was the brainchild of a PhD audiologist [RN et al] for the sole purpose of calling thyself 'Doctors" has somehow grew exponentially in the USA . The actual training has not changed significantly from its prior MA, CCC-A programes. Since then, non medical audiologists, have been aggressively pushing their scope of practice in the USA. The public image of being "doctors" has been exploited for advertising as "hearing health doctors". It also impacted the decision of insurance companies to issue a "provider number" for the AuDs and non AuD audiologist in the USA. Essentially they were given a permission to practice ear medicine without proper training or licensing. That move has been transplanted to the UK market over the last 4-5 years. Other global markets have also been tapped. The Impact is not as it is in the USA yet. This presentation will address the impact of this situation on Otology – Neurotology and the practice of Auditory and Vestibular Medicine globally.

Ref: *Hamid MA. The scope of practice of auditory and vestibular medicine. Curr Opin Otolaryngol Head Neck Surg. 2010 Oct;18(5):405-6. PubMed PMID: 20827084.*

Characterisation and optimization of the ocular vestibular evoked myogenic potential

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Ever since the vestibular evoked myogenic potential (VEMP) was first introduced nearly twenty years ago the level of interest in this elegant and highly informative test has continued to grow. Now referred to as the cervical VEMP or c-VEMP it has become a popular tool for assessing saccule and inferior vestibular nerve function and is used as a powerful adjunct to the vestibular test battery. More recently the c-VEMP has been complimented by the introduction of the ocular VEMP or o-VEMP. Although in its infancy, this closely related test offers the

potential to interrogate the utricle and hence provide a valuable method of more fully understanding inner ear function. Whilst the c-VEMP and o-VEMP may share many common features, to date the o-VEMP has not been fully optimised in terms of the eliciting stimulus or response characteristics. In this paper we present data which highlight the key parameters associated with accurate and repeatable o-VEMP recordings as well as some early promising clinical applications.

Vestibular Activation Differentially modulates Human V1 and V5/MT Excitability and Response Entropy

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Head movement imposes two additional burdens upon the visual system, both alleviated by vestibular input i.e., maintaining visual-acuity and determining the origin of retinal image motion (viz. self-motion versus object-motion). Although maintaining visual acuity during self-motion is effected by minimising retinal slip via the vestibular-ocular reflex, higher-order mechanisms also contribute. Disambiguating self-motion versus object-motion also invokes higher-order mechanisms and a cortical visuo-vestibular reciprocal antagonism is propounded. Hence one prediction is of a vestibular modulation of visual cortical excitability and indirect measures have variously suggested none, focal or global effects of activation or suppression in human visual cortex. Using transcranial magnetic stimulation-induced phosphenes to probe cortical excitability, we observed decreased V5/MT excitability versus increased V1 excitability, during vestibular activation. Non-specific factors (e.g. arousal) may affect cortical excitability hence response specificity was assessed using information theory, specifically response entropy. Vestibular activation significantly modulated phosphene response entropy for V5/MT but not V1, implying the vestibular effect was specific for V5/MT responses. This is the first demonstration that vestibular activation modulates human visual cortex excitability. Furthermore, using information theory, not previously used in phosphene response analysis, we could distinguish between a specific vestibular modulation of V5/MT excitability from a non-specific effect at V1.

Improving impaired balance function: real-time versus carry-over effects of prosthetic feedback.

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Aim: Given the correlation between the frequency of falls and excessive trunk sway, this study investigated whether training with real-time prosthetic biofeedback (BF) of trunk sway induces a carry-over improvement in balance control once BF is removed. Methods:12 healthy older adults and 8 chronic uncompensated vestibular loss patients were tested. All participants performed a battery of 14 balance and gait tasks (pre-test) upon their initial lab visit during which trunk angular sway in the pitch (AP direction) and roll (ML direction) was measured at L1-3 with a SwayStar™ system. They then received balance BF training on a subset of 7 tasks, three times per week, for two consecutive weeks. BF was provided using a multi-modal biofeedback system with graded head vibrotactile, bone-conducting auditory, and visual cues in relation to subject-specific angular displacement thresholds. Performance on the battery of the 14 balance and gait tasks was re-assessed without FB immediately after the 2 week training period, as well as 1 and 4 weeks later to examine BF carry-over effects.
Results: Significant 30-40% reductions in trunk angular displacement were observed with the real-time BF, compared to the pre-test trials. The effects of BF persisted when BF was removed immediately after the final training session. BF carry-over effects were slightly less evident at 1 and 4 weeks post-training.
Conclusions: This evidence supports the potential short-term effects of BF training in a number of stance and gait tasks after the BF is removed in healthy elderly subjects and those with vestibular loss. However, the prospect for longer term (>4 weeks) effects of prosthetic training on balance control remains currently unknown. Developing a more effective real time feedback device and improving its carry-over effects both seem to be viable options.

Enhanced Gaze Stabilization and Dynamic Visual Acuity Testing in Normal Subjects and Patients with Unilateral Vestibular Dysfuunction

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Objective: To compare results of the dynamic visual acuity test (DVAT), gaze stabilization test (GST), minimal perception time (MPT) and two novel oculomotor tests (target acquisition test- TAT and target following test-TFT) in patients with unilateral vestibular loss (UVL) and control subjects using a new computerized testing system prototype.
Study design: Cross-sectional study
Setting: Tertiary academic referral laboratory.
Patients: Seventeen patients (mean age, 63 years; range 40-86 years) with ≥ 49% bithermal caloric asymmetry or ablative surgery; 20 controls (mean age, 33 years; range 23-66 years).

Intervention(s): Diagnostic test protocol using a computerized system of target presentation and head velocity monitoring.
Main outcome measure(s): Comparison of optotype size (DVAT) and achieved peak head velocity (GST) during ipsilesional and contralesional self-generated head movement using randomly presented transient targets.
Results: Participants in the patient group had significantly lower GST scores in both the affected ($p<0.001$) and unaffected side ($p=0.004$) with median values 86.3 and 105.7 degrees/sec respectively compared to those participants in the control group (140.1 degrees/sec). The DVAT showed that the patient group had greater loss in visual acuity in both directions (affected side=0.31 ΔlogMAR, $p<0.001$; unaffected side=0.26 ΔlogMAR, $p=0.018$) compared to the control group (0.13 Δlog MAR). ROC curve analysis identified optimal thresholds of ≥0.3 ΔlogMAR decline for DVAT and ≤90 degrees/sec for GST. DVAT and GST had the same sensitivity (65%) for detecting the patient group's affected side compared with the control group, but GST had a slightly higher specificity (95% versus DVAT 90%). When either GST or DVAT was abnormal, the sensitivity increased (82%) while maintaining specificity (90%). There were no differences in the minimal perception time, target acquisition test or target following test results between the patient group and control group.
Conclusions: Both GST and DVAT individually demonstrated reduced gaze stabilization towards the affected ear in the patient group compared to the control group. Using the tests in combination resulted in higher sensitivity and specificity compared to either test alone. Tests of minimal perception time and target acquisition and following were not significantly different between the two groups.

Recurrent vestibulopathy: natural course and prognostic factors

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Objective: To evaluate the natural course of recurrent vestibulopathy.
Study design: Retrospective analysis.
Patients: One hundred and five adult patients with attacks of vertigo without auditory or neurological symptoms.
Methods: A structured interview was conducted over the telephone, 12 to 62 months after the patient's first visit to the out-patient department.
Results: Two-thirds of patients experienced spontaneous resolution of vertigo, while one-third continued to have symptoms. The diagnosis was subsequently changed to migraine in 2 per cent of patients and to Meniere's disease in 1 per cent.
Conclusion: The prognosis for patients with recurrent vestibulopathy is good. In a few cases, the diagnosis is provisional and will be subsequently changed to migraine or Meniere's disease.
Reference: Journal of Laryngology & Otology (2010,124:19-22)

Particle Repositioning is effective and safe when performed in a Falls and Syncope Unit – a consecutive series of 85 patients

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Objective: To report on efficacy and adverse outcomes in a consecutive series of particle repositioning manoeuvres performed in a Falls and Syncope unit on older patients who presented with falls, dizziness or syncope.
Study Design: Review of consecutive patients treated with a particle repositioning manoeuvre in the Falls and Syncope Unit. This unit sees >700 new patients per year with falls, syncope and dizziness. It is operationally distinct from the ENT/Vertigo service in the trust. Previous work from our unit shows that BPPV is a common diagnosis in the elderly who present with postural dizziness or falls with postural change (ref 1). Older persons with BPPV are less likely to complain of the sensation of vertigo with BPPV than younger patients and may not therefore be referred to an ENT service. Because of the increasing numbers of patients seen with BPPV we actively trained a balance physiotherapist and doctors in repositioning manoeuvres.
Method: Presenting complaint, semicircular canal involved, number of particle repositioning manoeuvres required for resolution of symptoms and adverse outcomes were recorded. Effective treatment was recorded if symptoms resolved and repeat Hallpike testing at review at 4 weeks was negative.
Results: 85 patients had particle repositioning manoeuvres in the Falls and Syncope Unit over a 15 month period. Mean age 74.6 years. Age range 45- 92, 17 males.
43 presented with falls and of these 62% (27) had fallen as a result of BPPV e.g getting up, looking up. Resultant injuries required hospital admission in 19% (8) and included head injuries, fractures, soft tissue bruising to chest wall or face. All had posterior canal BPPV, only one patient developed horizontal canal BPPV after treatment.
Posterior canal BPPV was present unilaterally in 73 (right side 42, left side 31) and bilaterally in 12.11 were treated while in-patients, the remainder as outpatients.
2 patients had such significant restriction in neck movements and discomfort from their neck that we performed only 2 treatments without resolution of symptoms.
5 patients remain under review, 5 DNA to follow up appointments therefore 73 have completed treatment and follow up.
Overall 63 (86%) received effective treatment for BPPV in the falls and syncope unit.
50 (66%) were successfully treated on one visit.8(10%) required 2 treatments
3(4%) required 3 treatments with 2 (3%) requiring 4 treatments
We referred two patients to the regional vertigo service in whom we had performed 5 manoeuvres, and one of these had a further 2 treatments before symptom resolution.

No significant adverse events were recorded post manoeuvre. No patients required admission. 3 complained of nausea. No patient reported falling within 4 weeks after manoeuvre. We saw one patient urgently 3 days post manoeuvre who was more unsteady but he was known to have an existent cerebellar infarct and down-beat nystagmus in addition to BPPV. Repeat hallpikes was negative and assessment of balance was unchanged from initial presentation.
Conclusion : BPPV is a common diagnosis made in a falls and syncope service and should be actively pursued and treated if present to prevent falls. This series shows that the rate of successful particle repositioning manoeuvres is similar to published series from traditional vertigo services and ENT departments.(Ref 2) Training in Hallpike testing and repositioning manoeuvres is important for those working within falls services. No adverse events were identified in this group of older, complex patients.

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Neurology 2008;70;2067

Vestibular function in unexplained falls

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Introduction
One in three people over the age of 65 fall annually, with incidence increasing with advancing years.
Vestibular hypofunction is associated with falls in older adults and also fractures of both the hip and wrist of the affected side. Vestibular function declines with age; approximately 50% of people in their 60's are unable to maintain balance when only vestibular cues are present. This proportion rises to approximately 70% and 85% of those in their 70's and 80's respectively.
However, the prevalence and types of vestibular dysfunction in older adults experiencing multiple unexplained falls is unknown, and may be useful in designing new rehabilitation programmes.
Subjects
Age matched patients referred for a) vestibular function testing at The National Hospital for Neurology and Neurosurgery or b) patients experiencing 2 or more unexplained falls who attended a falls clinic within the Southwark and Lambeth Integrated Care Pathway for Fallers (SLIPS).
Results
Vestibular dysfunction is common in a cohort of unexplained fallers (75%). Subjects referred for vestibular function testing report significantly less falls than those referred into the SLIPS pathway ($p<0.01$).Undiagnosed vestibular fallers are not significantly different from vestibular subjects across all vestibular outcome measures ($p>0.05$), but have a higher PPA falls risk ($p<0.05$).
Conclusions
The high prevalence of vestibular dysfunction in individuals experiencing unexplained falls indicates that the addition of vestibular function tests to older adult falls assessment may improve efficacy and that techniques to improve vestibular function in falls rehabilitation may be indicated.

Utricular function in normal subjects and patients with vestibular dysfunction

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Objective: To measure utricular function, using the unilateral centrifugation technique, in normal subjects and patients with vestibular dysfunction.
Methods: Subjects were securely seated on a rotating chair (Neuro Kinetics Inc, Pittsburgh) which slowly accelerated in darkness about a vertical axis to reach a constant velocity of 400°/s. During the constant velocity rotation, the subject was shifted slowly sideways relative to the rotation axis, up to 4.2cm to the right and left. The subject was held for 30s in the three test positions (Right, Centre and Left). Centripetal acceleration of the utricles modifies the perceived gravitational vertical, and so changes in the subject's position relative to the rotation axis will generally induce sensations of roll tilt. This roll tilt was measured using a chair-fixed subjective visual vertical (SVV). The subject used a rotating knob to orientate a randomly offset laser line to the perceived gravitational vertical; approximately 8 settings were made in each test position and the median value was taken to indicate the subjective roll tilt. Subjects also performed the SVV task when the chair was stationary (Static value). In each extreme chair position, the net acceleration on the head (gravitational and centripetal) was equivalent to a roll tilt of 11.7°, in opposite directions for the Right and Left chair positions. The 'SVV gain' was defined as the difference between the SVV tilts in the Right and Left positions, divided by 23.3°. Utricular 'asymmetry' was defined by comparing the mean SVV tilt in the three test positions (relative to the Static value) to the difference between the Right and Left SVV settings. A complete unilateral loss would be expected to produce zero SVV tilts when the remaining functioning utricle was positioned on the rotation axis. In this case, the mean SVV tilt would be equal to half the maximum range (100% asymmetry).

We tested 28 normal subjects (age 17 to 67 years) and 42 vestibular patients (29 Ménière's, 8 bilateral vestibular loss and 5 unilateral canal pareses of different aetiology). All patients had one or more additional vestibular function tests (calorics, VEMPs or conventional rotational testing). The total caloric response was defined by summing the peak slow phase eye velocities induced by 30°C and 44°C irrigations of each ear; unilateral caloric weakness was calculated using Jongkee's formula.
Results: SVV gain and asymmetry were normally distributed in the normal subject group (mean SVV gain = 0.55 ± SD 0.25; mean asymmetry = -6% ± SD 35%).
The patients with bilateral loss (e.g. total caloric response ≤ 20°/s) had significantly reduced SVV gains (mean = 0.23 ± SD 0.14). Combining all the patients with calorics (n=37) there was a significant positive correlation between SVV gain and total caloric response (R^2 = 0.14, p<0.03).

SVV gain for the Ménière's group (mean = 0.49 ± SD 0.24) was not significantly different to the normal controls. However, in the Ménière's patient group there was a significant correlation between utricular asymmetry and caloric weakness (R^2 = 0.26, p<0.01).

3/5 patients with unilateral loss (caloric weakness > 60%) had strong asymmetries (>60%) indicating ipsilaterally reduced utricular function. However, one other patient had a large SVV gain (1.3) and only 15% utricular asymmetry (VEMPs were also preserved in this patient); the final patient in the group had a very low SVV gain (0.02).
Conclusions: Unilateral centrifugation with SVV measurements gives additional, potentially useful information about utricular function. Bilateral vestibular loss produces low SVV gains and unilateral deficits produce asymmetric SVV responses.

Incidence of vestibular schwannoma and incidental findings on MRI and CT scans of patients from the direct referral audiology clinic

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Background: Direct referral audiology clinics (DRACs) by general practitioners and provision of hearing aids were introduced to the NHS to reduce the waiting time and pressure on ENT clinics. Patient with asymmetrical sensorineural hearing loss (ASHNL) usually get referred to ENT because of the worry of vestibular schwannoma (VS) although many of these of these patients did not have VS. To reduce cross referral rate between ENT and DRACs, a new protocol was designed locally to allow MRI scan to be requested directly from DRACs. However, there has not been any report of how many VS were diagnosed in such pathway and this could be useful for service planning for the health organisation.
Objective: To identify the number of cases of VS in patients referred to DRACs and incidental findings on MRI and CT scans in patients being investigated for VS.
Method: A prospective data collection of patients referred to DRACs and found to have ASNHL was undertaken. MRI and CT results and the audiograms of patients with VS were reviewed.
Results: 4100 patients were seen during 3.5 years periods. 398 scans were performed (298MRI and 100 CT). Six (1.5%) patients had VS. There were no differences in age (t-test p=0.266) and gender (Fisher's exact p>0.999) between the groups. Two hundred and seven (52%) scans were normal and twelve (3%) had significant incidental findings.
Conclusion: The incidence of VS from DRACs is low with 1.5% in comparison to 3-5% (literature looked at the whole ENT department). If no protocol was in place, over 300 patients would have added on to our ENT clinic. DRACs provide a cost effective way of managing certain group of patient with ASHNL in comparison to ENT clinic. Its service can be expanded to include these patients as an initial assessment point.

Visual dependency after Vestibular Neuritis

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Objectives
Vestibular patients often report 'visual vertigo', where dizziness and disorientation are brought on by complex or moving visual surroundings. These symptoms are believed to stem from increased visual dependency in response to vestibular injury. Such ineffective compensations strategies can hinder recovery and cause chronic symptoms. We examine the interplay between symptoms, visual dependency and psychological factors in the acute and recovery stages after vestibular neuritis (VN).
Methods
Twenty-five VN patients (mean age = 48.5, range = 20-75, 11 females) were tested acutely (mean=2 days, sd=1.4). Eighteen were followed up in the early recovery stage (mean=9.9 weeks, sd = 3.1). A control group of 20 VN patients (mean age 52.9, 7 females) were tested in the long term recovery stage (> 6 months from onset). Visual dependency was measured with the Rod and Disc test. Validated questionnaires were also completed - Dizziness Handicap Inventory (DHI); Hosptial Anxiety and Depression scale (HADS); Body Sensations Questionnaire (BSQ). Speilbergers Trait Anxiety Inventory and Situational Vertigo Questionnaire were given at the follow up only. All patients had bithermal caloric testing.
Results
Exploratory factor analysis of acute and short term follow up measures revealed 3 factors. The first factor accounting for 42% of the variance loaded recovery measure (DHI), visual dependency (acute and follow up), situational vertigo and follow up HADS and BSQ scores. Symptom score at follow up correlated significantly with acute and follow up visual dependency (R=0.66, p=0.001 and R = 0.54, p=0.01, respectively). The control group show a similar pattern, with those suffering from higher levels of handicap showing increased visual dependency. The second factor loaded acute HADS score and trait anxiety, whilst the third factor loaded acute and follow up caloric function only.

Conclusion
Patients suffering from higher levels of handicap show increased levels of visual dependency. Acute visual dependency is shown to be even more predictive of symptom recovery than follow up levels, highlighting the importance of acute compensatory strategies adopted after a vestibular insult. The so called chronic subjective dizziness that occurs in absence of peripheral weakness, which has been attributed largely to a psychogenic basis, may in fact be a product of inadequate central processes after vestibular injury, resulting in increased visual dependency and chronic symptoms.

Psychopathology of vestibular migraine: what is the mechanism?

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Background
There is a high prevalence of depressive and anxiety disorders in both individuals with migraine and also those with vestibular disease. Previous work has suggested that there is a specific association between migraine, vestibular disease, and anxiety disorders, but the precise mechanism is unknown. This study was designed to investigate the role vestibular symptoms play in the psychological symptoms associated with vestibular migraine.
Methods
Two groups of patients were studied. The first was a group of 39 patients with vestibular migraine according to Neuhauser's 2001 criteria. The second group comprised 44 patients with dizziness symptoms without migraine. Patients completed validated questionnaires (Beck Depression Inventory, Beck Anxiety Inventory, Vertigo Symptom Scale). Regression analysis was carried out to determine predictors of depressive and anxiety symptom scores.
Results
The migraine group had significantly higher scores on the anxiety (BAI median 19) ($p=0.03$) scales than the non-migraine group (BAI median 11). There was no difference in depression scores (BDI median 10 for migraine, 8 for non-migraine, $p=0.57$). The migraine group also scored more highly than the non-migraine group on the vertigo severity and frequency scales (VSS vertigo scale median 30 in the migraine group, 16 in the non-migraine group, $p=0.003$). Regression modelling showed that the high anxiety symptom scores were largely accounted for by the excess of vestibular symptoms.
Conclusion
This study has confirmed that patients with the vestibular subtype of migraine have high anxiety symptom levels when compared to non –migrainous dizzy controls, and suggests that this difference may be largely explained by high levels of vertigo symptoms.

Binocular Rivalry: merely a perceptual quirk?

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Our eyes work together in a synchronous and synergistic manner in order to obtain a single unified percept of the world. Simultaneous presentation of disparate images to each eye replaces the harmonious relationship with competition between the eyes giving rise to the beguiling phenomenon termed binocular rivalry. Exactly where in the visual pathway binocular rivalry is processed and whether rivalry can modulate an independent sensory system are questions that remain unanswered. In accordance with stimulus rivalry theory[2], visual cortical areas associated with binocular rivalry are analogous to the multi sensory vestibular cortical processing areas. We hypothesised that visuo- vestibular conflict induced by binocular rivalry could probe these multi sensory visual and vestibular cortical areas using a model of reciprocal inhibition. Here we present the first report that binocular motion rivalry can modulate the function of an independent sensory system. Furthermore our novel psychophysical data supports the notion that the vestibular cortex in man is lateralised to the non dominant hemisphere and that a central integrator co-processes visual motion and vestibular signals in order to modulate and influence the vestibular ocular reflex by top down cortical control mechanisms.

The human posterior parietal cortex acts as neural integrator during vestibular-guided path integration A bias in space and time but preserved velocity perception from a posterior parietal cortex lesion

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Summary
Humans can perceive changes in their angular orientation of their head or body in the dark using only vestibular cues of motion. Since the vestibular apparatus detects head motion and not position, the brain must convert signals of motion to that of position and mathematically, this could be achieved by a temporal integration of head velocity signals to obtain position. Although the *brainstem neural integrator* - a neural circuit that provides a position signal to the eye allowing accurate head-eccentric eye positions to be maintained - it is unknown if the brainstem neural integrator also provides the position signal to motion perception. An alternative mechanism is of a parallel perceptual neural integrator. One implication of a perceptual neural integrator would be the use of a perceptual time signal in deriving angular position. Here we show, in a patient with a right posterior parietal cortical stroke, lateralised deficits in vestibular-guided spatial orientation and impairment in motion duration judgements despite normal brainstem neural integrator functioning and preserved angular velocity perception. Spatial orientation and timing performance improved in parallel over time. We propose that in humans, a

perceptual temporal integration of vestibular signals, utilising interval timing mechanisms, is required for angular orientation and that this temporal integration is effected by the posterior parietal cortex.

The Contribution of Hearing to Normal Balance

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Introduction:
The classical description of normal human balance is said to rely on three sensory inputs: Vision, the peripheral vestibular system and proprioception. This information is relayed centrally, integrated and interpreted to provide gaze stabilization and postural control. Although anecdotal accounts suggest hearing loss to contribute to unsteadiness, no formal studies have demonstrated a clear relationship. We therefore undertook this study to assess hearing change and postural control in normal subject
Materials and Methods:
Twenty normal volunteers were recruited to this study. Postural control was assessed using Physio Fun™ software and a Nintendo Wii gaming console and balance board. Each subject was assessed in two environments: A normal clinic room and a standard soundproof audiological booth of similar dimensions. Each subject was tested standing upright for 30 seconds with their eyes open, eyes closed, with and without ear defenders and whilst standing on foam. The centre of gravity area of ellipse was recorded and data statistically compared using the Friedman 1-ANOVA test.
Results:
Statistically significant differences were observed in subjects with their eyes open and closed in both normal ($p= 0.0002$) and soundproof rooms ($p=0.0164$), and standing with eyes open on and off foam in both a normal and soundproofed environment ($p=0<05$). No significant difference was seen between groups when wearing ear defenders.
Conclusion:
Our results suggest this method provides a simple and inexpensive tool for assessing static postural control. In addition, ambient noise or hearing plays a significant role in postural control.

Guest Lecture:

Pharmacotherapy of vertigo, dizziness and nystagmus

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The prerequisite of the treatment of vertigo and dizziness is a specific diagnosis. The various forms of vertigo are treated with pharmacological therapy[1], physical therapy, psychotherapeutic measures and, rarely, surgery. Before treatment begins, the patient should be told that the prognosis is generally good for two reasons: (a) many forms of vertigo have a favorable natural course (e.g., the peripheral vestibular function improves or central vestibular compensation of the vestibular tone imbalance takes place) and (b) most forms can be successfully treated.[2] For pharmacotherapy there are basically seven groups of drugs that can be used (the "**7 'A's**"): antiemetics; anti-inflammatory, anti-menières, and anti-migraineous medications; anti-depressants, anti-convulsants, and aminopyridines. In the following the current pharmacotherapy of the most frequent forms of peripheral vertigo and dizziness as well as nystagmus will be summarized

Peripheral vestibular disorders
Acute vestibular neuritis. A prospective randomized placebo-controlled study (on 141 patients) showed that methylprednisolone alone significantly improves the recovery of peripheral vestibular function in patients with vestibular neuritis, whereas valacyclovir does not.[3] The combination of methylprednisolone and valacyclovir was not superior to steroid monotherapy.
Menière's disease (MD). In an open, non-masked trial, in which patients with MD received either a low dosage of betahistine-dihydrochloride (16 or 24 mg tid) or a higher dosage of 48 mg tid for at least 12 months it was demonstrated that the higher dosage is more efficient.
Vestibular paroxysmia. Vestibular paroxysmia is characterized by recurrent brief attacks of vertigo due to a neuro-vascular cross-compression of the vestibular nerve. In an open trial it was demonstrated that carbamazepine in a dosage of 400 to 600 mg per day significantly reduces the number of attacks of vestibular paroxysmia.[4]

Central vestibular, ocular and cerebellar disorders
Central vestibular forms of vertigo arise from lesions at the neuronal circuitry between the vestibular nuclei and the vestibulo-cerebellum as well as those between the vestibular nuclei, the vestibular and ocular motor structures of the brainstem cerebellum, thalamus, and vestibular cortex. On the one hand, these are clearly defined clinical syndromes of various etiologies, for example, upbeat or downbeat nystagmus (the quick phase of nystagmus beats upward or downward).
Downbeat and upbeat nystagmus and episodic ataxia type 2. Recent trials showed that the potassium channel blockers 3,4-diaminopyridine and 4-aminopyridine (in well tolerated dosages of 5 to 10 mg tid) are effective for the treatment of downbeat nystagmus[5-7] and upbeat nystagmus.[8] It was also demonstrated that the potassium channel blocker 4-aminopyridine (in well tolerated dosages of 5 to 10 mg tid) is effective for the treatment of episodic ataxia type 2 (EA 2)[9,10] (a hereditary disorder caused by mutations of the P/Q-calcium

channel gene CACNA1a; for ref. see [11]). Recent animal studies showed that 4-aminopyridine in therapeutic concentrations restores the diminished precision of pace-making in Purkinje cells.[12]

Vestibular migraine. Vestibular migraine is the most common cause of central recurrent attacks of vertigo. Characteristic features include recurrent attacks of various combinations of vertigo, ataxia of stance and gait, visual disorders, and other brainstem symptoms accompanied or followed by occipitally located head pressure, pain, nausea, or vomiting.[13-15] There is, however, an ongoing debate as to whether vestibular migraine is a clinical entity. Treatment is the same as for migraine with aura, i.e., for prophylactic therapy the use of beta blockers (metoprolol or propranol), topiramate or valproic acid for at least three to six months.[16]

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POSTERS:

Modulation of involuntary motor cortical mechanisms underlying the 'broken escalator' effect with tDCS

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Motor behaviour, including locomotion, is optimised to our usual surroundings thus uncommon environments unmask inappropriate motor behaviour; for example, the everyday experience of stepping onto a stationary escalator causes a 'stumble'. Critically, we stumble despite our full awareness that the escalator is broken. In the laboratory, this 'broken escalator' effect is reproduced when subjects step onto an obviously stationary platform (AFTER trials) having previously stepped onto a moving platform (MOVING trials). We hypothesised that the broken escalator aftereffect, despite being apparently impervious to volitional control, is mediated by motor cortical mechanisms. Thirty healthy volunteers received either sham or real transcranial direct current stimulation (tDCS; 2mA for 15 minutes over the primary motor cortex and supplementary motor area) whilst resting and prior to walking onto the moving platform. Subjects in the real tDCS group (compared to sham) displayed both a significantly larger trunk displacement in the 1st AFTER trial and a persistence of the locomotor after-effect into the 2nd AFTER trial. Our data show that increased cerebral motor cortex excitability augmented the involuntary 'broken-escalator' after-effect. Hence it appears that in man, the cerebral cortex dominates over subcortical regions in the control of involuntary locomotor action. These findings open the possibility of using direct current stimulation over cortical gait areas to improve locomotor learning in patients with neurological gait disorders.

All that glitters is not .... "All that is dizzy is not 'ear'!" Cautionary tales from the Balance Clinic

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The Department of Health Good practice Guide on "Provision of Adult Balance Services" draws on the information from both the documents Department of Health Action on ENT Balance Programme and The Royal College of Physicians Balance Disorders: Achieving excellence in diagnosis and management. The need for the balance of professional expertise as a network in the wider community is emphasised. The Guide recommends in some instances a rapid access of a selected,

prioritised patient cohort to Specialist or Supra-specialist centres. These Tertiary Centres require the full complement of several specialties ie:- of Otolaryngology, Audiovestibular Medicine, Neurology, Rehabilitation Medicine, Geriatrics ,Cardiology and also the expertise of multidisciplinary teams consisting of Audiologists, Audiological Scientists, Physiotherapists, Psychologists etc.

Patients can present to such centres with a confusing array of symptoms and it falls upon the clinicians from the various medical specialities to establish the diagnosis and the nuances of disability that results. The workup to determine the diagnosis underpins the application of the appropriate therapeutic process and modes of rehabilitation.

We present four cases where the diagnosis was difficult to establish, which were eventually proven to be 'not what they seemed to be'. What initially appears to be in the realm of otology can prove to be neurological. In some instances there can be dual or multiple pathology causing symptoms and influencing the clinical picture.

We emphasize caution in determining diagnosis of atypical clinical presentations and the need to follow patients through a period of time till the clinical state fully evolves and the diagnosis is clearly evident. We caution against early diagnostic labelling. We recommend the establishment of parallel, multispecialty clinics which can facilitate discussion of cases. Dizziness, imbalance, faints and falls fit within a spectrum of presentations which overlap each other. If there is poor response to therapeutic intervention there should be a rapid medical re-evaluation. We recommend early diagnosis, early treatment and rapid application of customised therapy to ensure better quality of health outcome, before disabilities become ingrained and permanent handicap results.

Instead of the term Neuro-otology, we would promote the use of a term "Neuro sensory disorders" to classify clinical presentations in the "balance" clinics.

The effects of visual stimulation on visual dependency measures in patients with visual vertigo

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Objective Patients with a vestibular disorder may develop an ov er-reliance on visual cues for balance, i.e. visually dependence, which leads to an exacerbation of symptoms when in busy, dynamic visual environments. Customised vestibular rehabilitation incorporating exposure to optokinetic stimulation (OKS) has been shown to improve subjective visually-induced dizziness. However, whether OKS exposure induces a reduction in visual dependency in this patient group is unknown. The purpose of this study is to examine the effects of customised vestibular rehabilitation incorporating OKS on visual dependency measures in patients with visually-induced dizziness. Moreover, an intensive treatment programme, which showed improvements in visual dependency in healthy adults was performed to investigate whether it could be tolerated by patients.
Methods Twelve patients with a peripheral vestibular disorder and visually-induced dizziness were randomly allocated into either Group C who received customised vestibular rehabilitation in isolation or Group OKS which combined customised vestibular exercises with exposure to optokinetic stimulation. Both groups attended treatment sessions for five consecutive days and were then provided with a progressive customised home rehabilitation program. Response to the treatment was assessed at baseline (Day 1), at the end of the five-day treatment, and at both four-week and eight-week follow up. Assessment included the 'Rod and Disc' test for visual dependency where subjects set the subjective visual vertical while facing a luminous rotating disc and the Functional Gait Assessment (FGA) to assess the patient's ability to perform complex gait tasks (i.e. walking with eyes closed). Subjective questionnaires concerning symptoms, symptom-triggers, perceived handicap from symptoms, and psychological state, were also used to evaluate treatment outcome.
Results Both groups showed significant improvements for depression, and perceived handicap scores post-treatment (p<0.05). FGA scores significantly improved for both groups (p<0.05). Results showed significant reductions (i.e. improvements) in subjective vertical tilt with the rotating disc for the OKS group only (p<0.05) indicating reduced visual dependency.
Conclusion Although customise exercises in isolation provide improvements for subjective symptoms and functional gait, only customised vestibular rehabilitation incorporating OKS exposure provides improvements in visual dependency. Furthermore, five-day intensive OKS exposure appears to be well tolerated by patients with visual vertigo and can be considered as an alternative form of treatment provision.

Vestibular perception after vestibular neuritis

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Introduction
Acute injury to the peripheral vestibular system can result in severe vertigo and nausea. Investigation into how the vestibular system compensates after such an injury has mainly focused on traditional tests of vestibular function (VOR, caloric). However recent imaging studies have shown wide spread cortical changes in acute vestibular neuritis (VN), of which conventional testing gives little insight. This study investigates the vestibular perceptual and VOR changes that occur after acute vestibular neuritis, in order to understand the cortical involvement in the compensation process.
Method
VN patients were studied in the acute (1-5 days after onset; N=31) and recovery (6-14 weeks; N=25) stages. Thirty one normal subjects served as controls. Subjects carried out two vestibular perceptual tests - 1. Vestibular perceptual threshold test – Patients were rotated from an initial acceleration of 0.5deg/s/s increasing by 0.5deg/s/s every 3 seconds and were instructed to press a direction specific button when they felt the rotation. 2. Angular velocity vestibular perceptual test -

Subjects indicated the perception of subjective rotational velocity by turning a tachometer wheel during 90deg/s angular (yaw) velocity steps in the dark. EOG recording measured oculomotor response to all rotations.
Results
Acutely, perceptual and oculomotor thresholds show similar patterns. Thresholds are raised above normal levels, with significant degree of asymmetry. At follow up there was an overall reduction in asymmetry, and a trend toward normal levels was observed. VOR responses to 90deg/s angular velocity steps showed similar results and are asymmetrically shortened below normal values. In contrast perceptual responses are remarkably symmetrical and are bilaterally reduced below that of the VOR.
Discussion
In acute VN both perceptual and oculomotor thresholds are asymmetrically elevated, and follow similar patterns of recovery during the compensation stages. In contrast perceptual responses to supra-threshold rotations differ significantly from that of the VOR and are bilaterally and symmetrically reduced. This perceptual 'shut-down', specifically in response to high velocity rotatory stimuli, may reflect a higher level cortical compensation mechanism responsible for suppressing the symptoms of intense vertigo felt in the acute stage of VN.

A trial of booklet-based self-management of dizziness with or without expert telephone support.

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Aims: This randomised controlled trial evaluated the cost effectiveness of booklet-based self-management of dizziness, with or without expert telephone support.
Method: In a single blind two-centre pragmatic controlled trial, 337 patients from 34 GP practices were randomised to a) self-help booklet with telephone support from a vestibular therapist, b) self-help booklet alone, or c) routine medical care. Symptoms (Vertigo Symptom Scale; VSS), disability (Hospital Anxiety and Depression Scale; HADS), handicap (Dizziness Handicap Inventory; DHI) and quality of life (EQ5D) were assessed by postal questionnaire at baseline, immediately post-treatment (3 months), and at one year follow-up.
Findings: All groups showed improvement in symptoms after 12 weeks, but by one year the routine care group had deteriorated while both treatment groups continued to improve and had significantly lower scores than the routine care group ($p = .01$). The two treatment groups did not differ significantly from each other at either time point. Changes in handicap scores were also significantly improved in both treatment groups relative to routine care at 12 months. Cost-effectiveness analysis concluded that both active treatments have increased benefit at increased cost that fall well within the NICE threshold for recommending treatment.
Discussion: Booklet-based self management of dizziness, both with and without additional telephone support from a vestibular therapist, appears to have long-term benefits for dizzy patients and may be a useful tool in a stepped care approach.

Independent processing of motion and colour binocular rivalry

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Binocular motion rivalry was found to asymmetrically modulate the vestibular ocular reflex (VOR). The aim of this experiment was to apply our novel finding in order to ascertain whether different types of binocular rivalry (motion vs. colour) are processed by a generic or specialist local visual cortical areas. In order to address this question we compared the effects of different forms of binocular rivalry upon the VOR, as colour and motion represent a dichotomy in visual cortical processing pathways. Currently it is unknown whether binocular rivalry processing involves low-level process (eye rivalry) or specialist cortical areas that are may be analogous to different forms of rivalry (stimulus rivalry). Here we demonstrate that motion rivalry asymmetrically modulates the VOR, whereas the colour rivalry has no effect. This finding suggests that motion and colour rivalry are independently rather than co-processed in the visual pathway.

Is the nausea which accompanies vestibular disorders motion sickness?

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BACKGROUND: The vestibular system is thought to be essential for the development of motion sickness (MS) since it has been shown for some stimuli that those with bilateral vestibular failure (BVF) cannot develop MS. Patients with unilateral vestibular lesions (UVL) experience symptoms similar to MS, such as nausea, vomiting and malaise, which can remain for many months after the initial lesion. These symptoms may be caused through similar mechanisms to MS, since both are provoked by unusual vestibular stimulation. Migraine sufferers are thought to be more susceptible to MS than controls; patients with vestibular migraine (VM), where there are symptoms of vertigo as well as nausea, may be even more susceptible.

OBJECTIVES: To investigate whether the symptoms experienced by chronic vestibular patients can be explained by an increased MS susceptibility, and to show whether those who have lost vestibular function are now immune to MS.

METHODS: 12 patients with UVL, 8 with VM, 8 with BVF, and 22 age matched controls reported their MS susceptibility before and after disorder using the Motion Sickness Susceptibility Questionnaire – Short form (MSSQ-Short); and underwent experimental testing of MS susceptibility by measuring their time to become moderately nauseous during off-vertical axis rotation (OVAR). In addition, both before and after motion, subjects' state of anxiety, as well as lying and standing blood pressure and heart rate were recorded.

RESULTS: MSSQ-Short results showed there was no general increase in MS susceptibility in patients after UVL. Equal numbers of UVL patients reported becoming both more and less motion sick since the disorder, changes that were not related to symptomatic recovery. In the VM patients, MS susceptibility was high from childhood and not significantly affected by the onset of vestibular symptoms. BVF patients reported reduced, but not absent, susceptibility since disease onset. Mean time to moderate nausea during OVAR in UVL patients of 7.7 minutes was similar to the control group mean of 8.1 minutes. VM patients' mean time was faster at 3.3 minutes, whereas all BVF patients tolerated 20 minutes of OVAR. State anxiety before motion did not correlate with susceptibility, however post-motion anxiety did. There was no relationship between orthostatic blood pressure or heart rate and MS.

CONCLUSIONS: The present study gives no evidence that symptoms of UVL are caused through the same mechanisms as MS, or that asymmetrical vestibular function increases MS susceptibility. It has been shown that whilst BVF confers immunity to MS, one vestibular system is enough to become motion sick. The increased MS susceptibility seen in individuals with VM is likely due to processes in the brainstem downstream of the peripheral vestibular system.

Alexander's law during high acceleration vertical head rotations

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Alexander's law states that the amplitude of the spontaneous nystagmus grows with increasing gaze in the direction of the fast phase. Using the search-coil method we employed head impulses in pitch at various eye-in-orbit elevation angles to test whether the normal VOR in the vertical plane has intrinsic properties that could account for Alexander's law in the behaviorally relevant high frequency range. We show that the gain of the VOR remained unaffected by eye-in-orbit position. These findings confirm previous results from a similar study with high acceleration horizontal head rotations and suggest that eye-in-orbit position does not directly modulate the activity in VOR pathways during unbalanced but reciprocal natural vestibular stimulation.

Gait and balance in patients with Parkinson's disease and small vessel disease using anodal transcranial direct current stimulation: a double blind, sham-controlled cross-over study.

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Falls are an increasing problem with age, constituting an important cause of morbidity and mortality in this population. Gait disorders are commonly seen in patients with ischaemic cerebral small vessel disease (SVD). Although the precise neural networks impaired in gait disorders due to SVD have not been fully elucidated, frontal basal ganglia-thalamocortical long-loop circuits are likely to be involved (Baezner J Neurol 2000). Parkinson's disease (PD) constitutes a further common cause of gait postural dysfunction – the management of which is therapeutically challenging. Recent research has shown that tDCS can depolarise cortical neurons and improve motor performance, in particular motor learning. We studied 17 patients with PD and 17 patients with SVD, and 20 age-matched healthy controls. All subjects performed baseline gait and balance tests, which were repeated after transcranial stimulation. Patients were randomly allocated into one of two groups (TRAINING or NO TRAINING). Subjects were also randomised to receive either real tDCS or sham stimulation. For the TRAINING group this occurred during the locomotor training, for the NO TRAINING group stimulation occurred during the repeat gait and balance tasks. We hypothesised that anodal tDCS applied over the supplementary motor area (SMA) - an area involved in movement initiation – and the primary motor cortex would improve gait and balance in PD and white matter disease and further augment improvements from physiotherapy. We found significant improvements in gait in the physiotherapy group, but no additional improvements with transcranial direct current stimulation. Our results suggest that anodal tDCS using our stimulation protocol does not improve locomotor learning in PD and SVD patients, but that clinician-delivered gait and balance exercises may improve locomotor function in these patients.

Hyperacusis: Evidence for cortical disinhibition

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Is hyperacusis due an impairment of cortical adaptation? One possible pathophysiological mechanism underlying hyperacusis is disinhibition at the cortical level that increases the gain of the afferent auditory signal. To investigate this hypothesis, a study was conducted, involving 12 patients and 13 controls, who underwent auditory testing, including recording of transient evoked otoacoustic emissions (TEOAEs) and cortical evoked auditory responses (CERA) potentials. The medial olivocochlear (MOC) suppression test and recording of the stapedial reflexes ascertained the absence of any distal efferent pathology.

CERA: characteristics of the N1-P2 component elicited by binaural presentation of repetitive 1000Hz tone bursts delivered in 3 successive blocks of 40 averaged responses at 40, 50, 60 and 70 dB SL were examined, including N1-P2 amplitudes, change in N1-P2 amplitude between blocks, N1 and P2 latencies, change in N1 and P2 latency between blocks and the N1 and P2 latency/stimulus intensity relationship.
The preliminary results of this study indicated a consistent trend for larger N1-P2 amplitudes in the patients' group at every intensity, with statistical significance for the three-block global average value at 60dB, with higher values in patients than controls (p=0.01). The intra-group analyses showed increased P2 latencies between blocks in patients at 40 dB SL, with marginal significance (p=0.05). The inter-group analyses indicated marginally longer P2 latencies in patients in block 1 at 60 dB SL (p=0.06), while significantly longer in patients than in controls for the global average at 60 dB SL (p=0.04).
TEOAE responses in patients with hyperacusis had a tendency of the larger amplitudes and wider range than in the control group.
These preliminary findings support the hypothesis that hyperacusis is a manifestation of an increased gain in the auditory system, as a result of cortical disinhibition.

Ocular Vestibular Evoked Myogenic Potentials (oVEMP) using air conducted sound. Test Parameters and Normative Data; Effect of body position on threshold.

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Aim. The aim of this study was: a) to accumulate normative data and to verify the viability of oVEMP testing in normal population and b) to determine the effect of body position on oVEMP threshold.
Material and Methods. 20 subjects enrolled in the study. 18 subjects completed oVEMP testing for data analysis in ages ranging from 12 to 72 - mean age 34,8 years, (10 male and 10 female). Each subject underwent a detailed neurotologic examination before testing. Comprehensive audiologic assessments were conducted for all subjects. Pure-tone thresholds were obtained from 0.250 to 8 kHz.
oVEMP and oVEMP threshold values were recording using an air-conducted stimulus of 500 Hz tone burst in two body positions; a) sitting upright position - with head level, b) lying supine position with chine 30^{o} toward the chest. The order of body positions and the order of the ears tested were randomized to control for fatigue.
Results. 18 subjects completed oVEMP testing. oVEMP were absent in 2 out of 20 subjects (10 %). The mean peak latencies (± standard deviation [SD]) of n1 and p1 for sitting upright position were: a) for right ears 10,46±0,62 msec and 15,51 ±1,03 ms and b) for left ears 10,68±0,75msec and 16,07±0,97 msec, respectively. The mean amplitude of Vp1-Vn1 ± SD, for sitting upright position were: a) for right ears 5,22 ± 5,44 μV and b) for left ears 6,74 ± 6,22 μV msec, respectively. The mean value ± SD for threshold responses were 84 ± 4,91 dB nHL (both sides and both body positions together).
Conclusion. We conclude that the normal oVEMP thresholds range from 75 to 90 nHL independently of body position. Moreover oVEMP are absent in a 10% of normal subjects without an obvious reason.
By studying specific population future research may be able to better understand the oVEMP neuronal pathway and its originating organ(s) when evoked by an air conducted stimulus.

Audio-Gyral & Somato-Gyral Illusions: visual reference & motion sickness

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The AudioGyral Illusion (AGI) is an error in sound localisation, which in the context of sudden whole-body rotational acceleration has some similarities to the much better known SomatoGyral Illusion (SGI), the false perception of self-rotation. The aim was to compare the AGI & SGI and to examine the effects of visual reference and possible relationships to motion sickness.
Subjects were seated in a completely enclosed cabin, rotated at 60 deg/s and stopped to stationary in 1.8s, according to a repeated measures design counterbalanced for order and rotation direction. A sound source (65dB, 2800Hz) was mounted in the enclosed rotating cabin. Subjects were asked to locate the sound, whether it seemed to move and if they perceived self-movement. Timed from rotation stop the duration of perceived sound movement and self-movement, were indicated via hand held push buttons. Participants were also asked to comment on the direction/quality of any perceived sound motion or self-movement. Expt 1 was a control condition in the dark (n=10 subjects) with the sound directly above the head; Expt 2 (n=16 subjects) the sound was directly in front, with conditions eyes open (EO) in the light and eyes closed (EC).
Expt 1: all subjects experienced SGI but not AGI. Expt 2: the SGI was significantly longer in duration than the AGI ($p<.001$) and both were shortened with eyes open ($p<.001$). The mean+/-SD seconds durations were: AGI: EC 17.41+/-8.09; EO 7.18+/-5.80; and SGI: EC 27.65 +/-9.86; EO 13.82+/-7.38. Higher motion sickness susceptibility correlated with longer AGI and SGI, both of which positively intercorrelated.
The SGI was longer and stronger than the AGI. Both illusions were shortened by having a visual reference (eyes open). The relationship of longer AGI and SGI with greater motion sickness susceptibility is possibly related to the central vestibular velocity storage time constant.

The Movement Frequency tuning of Motion Sickness is determined by biomechanical constraints on locomotion

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We assume that Motion Sickness (MS) is related to processing vestibular signals because the integrity of vestibular function is pre-requisite. MS susceptibility is greatest with vehicle motion circa 0.2Hz, progressively decreasing with higher and lower frequencies. The frequency, 0.2Hz, is also a 'watershed' in vestibular processing. Vestibular signals of head motion above 0.2Hz are interpreted as indicating 'translation'; lower frequencies are interpreted as 'tilt'. We hypothesise that these frequency characteristics originate in biomechanical limitations on body motion.
Subjects were 3 men; heights,1.73, 1.77, 1.8m; weights 60, 67, 75kg. Video analyses were made of the body tactics used running slaloms defined by sinusoidally patterned ground markers at speed. Each subject ran 16 slaloms which varied, harmonically, from 20m long x 4m wide to 4m long x 0.8m wide sinusoids.
Temporal frequencies ranged from 0.1Hz to run the longest slalom to 0.5 Hz for the shortest. Two stereotypical tactics emerged. Cornering using Whole body tilt aligning with the resultant Gravito Inertial Force (GIF) occurred in 100% of trials at 0.1Hz decreasing to 0% at 0.36 Hz. An alternative tactic of cornering by lateroplulsion of the legs, with trunk maintained earth upright and translating laterally occurred on 100% of trials at 0.5 Hz decreasing to 0% at 0.18Hz. Cross over between these tactics was at 0.22Hz.
The cross over frequency between preferential use of GIF alignment vs lateroplulsion at circa 0.22 Hz is determined by fundamental biomechanical constraints. Similarity in frequency with the tuning of vestibular processing and motion sickness susceptibility suggests that they originate in such biomechanical limitations. By extension, a cause of Motion sickness may be difficulty in selecting or effecting appropriate GIF alignment vs lateropulsive tactics to maintain stability under conditions of vehicle motion varying around 0.2Hz.