021 PROFILES OF BENIGN POSITIONAL VERTIGO TESTED ON THE EPLEY OMNIAX CHAIR

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Introduction Benign positional vertigo (BPV) has a characteristic pattern of nystagmus specific to the affected semicircular canal and the underlying mechanism of canalithiasis (where otoconia float freely) or cupulolithiasis (where otoconia are adherent to the cupula).

Methods We analysed the nystagmus slow-phase velocity (SPV) profiles of 100 subjects with posterior-canalithiasis, 30 with lateral-canalithiasis, 10 with lateral-cupulolithiasis and 3 with anterior-canalithiasis. Subjects were examined on the Epley Omniax Rotator, a mechanical chair with real-time video-oculography. Video data was analysed using custom-made LabVIEW software. Nystagmus onset, duration, peak-velocity, peak-latency and time taken for the peak-velocity to halve (t50), were measured.

Results In posterior-canalithiasis, nystagmus occurred within 14.2 seconds of positioning and lasted 2.5–34.5 seconds. The median vertical peak-SPV was 37.3°/s. The median peak-latency was 2.9 seconds and the median t50 was 3.4 seconds. In lateral-canalithiasis, nystagmus onset was mostly immediate. With the affected ear down, the median peak-SPV was 52.2°/s and the median peak-latency was 3.6 seconds; the t50 was 7 seconds (median) and the paroxysms lasted 9.9–48.5 seconds. In lateral-cupulolithiasis, nystagmus onset was instantaneous. With the unaffected ear down, the median peak-SPV was 69.6°/s. The peak-latency (median 18.6 s) and t50 (median 34.5 s) were significantly prolonged compared to canalithiasis. For anterior-canalithiasis, the onset was 0–2.9 seconds, the peak-latency was 3–5.4 seconds, the t50 was 6.4–10.5 seconds and the duration was 13.4–23.1 seconds.

Conclusions Canalithiasis and cupulolithiasis produce distinct SPV profiles, which enable their identification and the separation of BPV from other causes of positional nystagmus.

022 PATIENT-INITIATED EVENT MONITORING FOR ACUTE VERTIGO

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Introduction The diagnosis of vestibular disorders may be facilitated by analysing patient-initiated capture of ictal nystagmus.

Methods Adults with a history of recurrent vertigo were taught to self-record spontaneous and positional-nystagmus at home while symptomatic, using video-goggles. Patients with final diagnoses of disorders presenting with recurrent vertigo were analysed: 121 patients with Ménière's Disease (MD), Vestibular Migraine (VM), Benign Positional Vertigo (BPV), Episodic Ataxia Type II (EAII), Vestibular Paroxysmia (VP) or Superior Semicircular Canal Dehiscence (SSCD) were included.

Results Of 43 MD patients, 40 showed high-velocity spontaneous horizontal-nystagmus (median slow-phase velocity (SPV) 39.7 degrees/second (°/s); Twenty-one showed horizontal-nystagmus reversing direction within 12-hours (24 on separate days). In 44 of 67 patients with VM, low velocity spontaneous horizontal (n=28, 4.9°/s), up-beating (n=6, 15.5°/s) or down-beating-nystagmus (n=10, 5.1°/s) was observed; Sixteen showed positional-nystagmus only, and seven had no nystagmus. Spontaneous horizontal-nystagmus with SPV >12.05°/s had a sensitivity and specificity of 95.3% and 82.1% for MD. Nystagmus direction-change within 12-hours was highly specific (95.7%) for MD. Spontaneous vertical-nystagmus was highly specific (93.0%) for VM. In the seven BPV patients, spontaneous-nystagmus was absent or <3°/s, and characteristic paroxysmal positional nystagmus was observed in all cases. Patients with central and MD-related positional vertigo demonstrated persistent nystagmus. Two patients with EAII showed spontaneous vertical nystagmus, one patient with VP showed short bursts of horizontal-torsional nystagmus lasting 5–10s, and one patient with SSCD demonstrated paroxysmal torsional down-beating nystagmus when supine.

Conclusions Patient-initiated vestibular event-monitoring is feasible and could facilitate rapid and accurate diagnosis of episodic vestibular disorders.

023 GENETIC CARRIER SCREENING FOR DUCHENNE MUSCULAR DYSTROPHY: THE OUTCOME OF OVER TWENTY YEARS OF GENETIC COUNSELLING ON DISEASE EPIDEMIOLOGY IN A SINGLE-CENTRE COHORT STUDY IN NEW SOUTH WALES (NSW), AUSTRALIA

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Introduction Duchenne Muscular Dystrophy (DMD), an X-linked recessive genetic disorder is maternally inherited in approximately two-thirds of affected boys. Female relatives have carrier risk. This study proposes that proactive genetic screening and counselling for patients' relatives, contributes to reductions in preventable cases and ultimately disease incidence.

Methods A retrospective study of cases born in NSW from 1991–2013 was completed, using an electronic database of live male and prenatally diagnosed patients with DMD, referred to our tertiary service. Proband genotype/phenotype, pedigree, carrier-risk and extent of cascade screening were reviewed. Variance analysis (two-way ANOVA) was used to analyse changing trends in preventable cases.

Results 77 cases were identified. Mean age at presentation fell by 14-months over time. Probands were defined as 'theoretically preventable' when disease was identified in previous generations, or in males aged over 6 years, within the same generation. Fifteen (19%) cases were preventable, with a statistically significant decline in such cases over time.

Cascade screening and prenatal testing of subsequent pregnancies was offered to all carrier mothers and female relatives in the mother's generation. Fifteen women underwent prenatal testing. Three affected male foetuses were identified, with one