

P.004**Neurogenic orthostatic hypotension results in impaired information processing speed***L Robinson (London)* K Kimpinski (London)*

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Background: Neurogenic orthostatic hypotension (NOH) is characterized by a reduction in systolic blood pressure of ≥ 20 mmHg or diastolic blood pressure of ≥ 10 mmHg within three minutes of upright posture. NOH is prevalent in the elderly population who is at increased risk for cognitive decline, therefore it is imperative to investigate if there is a relationship between NOH and impaired cognition. **Methods:** Currently, 9 control subjects and 4 NOH patients have been recruited. Cognitive function is assessed using the symbol digit modalities test (SDMT) which assesses information processing speed and the Stroop test which measures response inhibition. SDMT and Stroop test are administered when the table is supine and during tilt. **Results:** NOH patients scored significantly worse on SDMT when lying ($p=0.018$) and standing ($p=0.004$) compared to the control group. Control subjects performed significantly better when standing for both SDMT ($p=0.008$) and Stroop ($p=0.026$), whereas NOH patients had similar scores when lying and standing for SDMT and Stroop. **Conclusions:** Preliminary results show that information processing speed is slower in NOH patients than controls in both the supine and standing positions. NOH patients have a more difficult time inhibiting unwanted responses compared to controls when standing, which is represented by a greater interference score in NOH patients.

P.005**Lisdexamfetamine precipitated pathological gambling***F Chaudhary (Christ Church) A Hirsch (Chicago) W MacPherson (Chicago)* J Nayati (Park Ridge)*

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Background: Lisdexamfetamine has not heretofore been reported to cause pathological gambling. Such a case is presented. **Methods:** A middle-aged woman, without past interest in gambling, gaming, or risk taking behavior, with childhood history of attention deficit hyperactivity disorder presented with difficulty focusing and concentrating. Lisdexamfetamine was started at 20 mg daily and gradually escalated due to lack of efficacy. At 70 mg daily, she began binging on sweets and gambling all day, every day at nearby riverboats, which she had never frequented previously. Upon reduction to 60 mg daily, the gambling resolved. Ritalin 20 mg every morning and 50 mg every afternoon was used without gambling reoccurrence. **Results:** Mental Status Examination: Alert, cooperative and oriented x 3 with good eye contact. Euthymic, without mania, thoughts logical and goal directed. **Conclusions:** Enhanced dopamine in the nucleus accumbens may induce hedonic activities including gambling, binging on sweets, or sexual activity (Moore *et al.* 2014). Lisdexamfetamine has been described to induce mania, and pathological gambling may have been an isolated manifestation of early mania. In those who have recently begun lisdexamfetamine, query should be made regarding change in gambling behavior and in those who are pathologically gambling, investigation should be entertained as to whether they are taking lisdexamfetamine.

P.006**Role of repeat antithyroid antibody testing in the diagnosis of Hashimoto's Encephalopathy***K Nedd (St. John's)* A Goodridge (St. John's)*

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Background: We present a case of a previously well 71-year-old woman who developed rapidly progressive dementia. She had several prolonged hospital admissions and extensive investigations were performed. Her illness was steroid-responsive with clinical features suggestive of Hashimoto's Encephalopathy (HE). However, multiple thyroid antibody panels acquired were initially normal, becoming elevated during subsequent relapses that occurred 3 years after initial presentation. The case signifies the importance of repeating antithyroid antibody levels in a patient with a clinical picture suggestive of HE. **Methods:** Case report. **Results:** The diagnosis of HE was established because of a compatible clinical picture including a relapsing encephalopathy with motor involvement and steroid responsiveness. In addition, although initial antithyroid antibody testing was negative, subsequent attacks were associated with significant elevations and reverted to normal with resolution of the attacks. **Conclusions:** In a patient with normal levels of antithyroid antibodies and a clinical presentation suggestive of HE, we recommend repeat antithyroid antibody testing to confirm diagnosis. Further studies are necessary to clarify the pathogenic role of elevated antithyroid antibodies in the mechanism of HE.

P.007**Familial idiopathic normal pressure hydrocephalus in a Canadian family***BC Shettar (London) S Mirsattari (London)**

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Background: Idiopathic Normal-pressure hydrocephalus (iNPH) is characterized by cognitive impairment, gait disturbance, enlarged ventricles with/without cerebral atrophy, with/without urinary incontinence, and normal cerebrospinal fluid pressure. Familial iNPH is very rarely described in the literature. A Canadian family with more than one generation of iNPH has never been described. **Methods:** Patient 1: 50-year-old female presented with wide-based and magnetic gait, multiple falls with subsequent freezing. LP with large volume tap was performed. Patient had ventriculo peritoneal (VP) shunt surgery. Patient 2: 52 year male (brother): Presented with long-standing cognitive impairment and fatigue. Montreal Cognitive Assessment (MOCA) was performed. Whole exome sequencing (WES) of both siblings as well as an unaffected first cousin was done. The father and grandmother of both patients was diagnosed with iNPH. **Results:** Patient 1: Opening pressure on LP was 22 cm-H₂O. She responded well to large volume tap. She had VP shunt resulting in improved gait. Patient 2: Opening pressure on LP was 16cm-H₂O. CSF flow study was slow for age indicative of NPH. MoCA score was 25/30. WES of patients and unaffected first cousin is underway. **Conclusions:** We present an undescribed Canadian family with iNPH in more than one generation. WES is underway for better understanding of genetic predisposition and inheritance of familial iNPH