on eye tracking measures of attention and executive function may reflect alterations in white matter tracts.

**P.036**
Clarithromycin induced sleep paralysis: a case report and review of literature

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**Background:** Clarithromycin is a macrolide antibiotic, which has been successfully used for treating narcolepsy without cataplexy and primary hypersomnia. Sleep-paralysis has not been reported as a side effect of this medication. **Methods:** We report a 44-year-old right-handed female, who presented with three episodes of paralysis over 2-day. Each spell occurred upon awakening or while falling asleep lasting less than 2-minute. Only one episode was accompanied by tingling and numbness. She denied cataplexy, sleep attacks, hypnopompic and hypnagogic hallucinations. She had no history of similar episodes. She had never experienced migraine with or without aura. She was obese and suffered nocturnal snoring. She had recently been started on Clarithromycin for pneumonia. Her neurological examination was normal. **Results:** Brain MRI was normal. Stroke work up including carotids CT angiogram, 24-hour Holter monitoring and echocardiogram were unremarkable. Polysomnogram when she was not on Clarithromycin indicated mild obstructive sleep apnea and no narcolepsy. She had no further episodes of sleep paralysis after Clarithromycin was discontinued. **Conclusions:** We believe that administration of Clarithromycin was the cause of sleep paralysis. We hope that this case report increases awareness about this particular side effect of Clarithromycin and leads to further investigation about the etiology of sleep paralysis.

**P.038**
A case report of an interesting paraneoplastic voltage-gated channelopathy

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**Background:** Morvan syndrome is an autoimmune paraneoplastic disorder affecting of voltage-gated potassium channels, most commonly the CASPR-2 subunit. The disorder is primarily characterized by hyperexcitability of both the central and peripheral nervous system accompanied by autonomic dysfunction. Clinically, the syndrome manifests as confusion, hallucinations, insomnia, hyperhidrosis, orthostatic hypotension, and muscle cramps with myoclonus. The disorder is primarily characterized by hyperexcitability of both the central and peripheral nervous system accompanied by autonomic dysfunction. Clinically, the syndrome manifests as confusion, hallucinations, insomnia, hyperhidrosis, orthostatic hypotension, and muscle cramps with myoclonus. **Methods:** We report a case of Morvan Syndrome in a 56 year old male with previous history of thymic squamous carcinoma accompanied by paraneoplastic myasthenia gravis and myositis. His clinical presentation was notable for subacute onset of muscle cramping, insomnia, which progressed to also include visual and auditory hallucinations. He also had notable dysautonomic symptoms including orthostatic blood pressure changes, sialorrhea, and hyperhidrosis. The diagnosis was confirmed with a positive serum assay for antibodies against the CASPR-2 subunit of voltage-gated potassium channels. **Conclusions:** This case is notable because to our knowledge it one of the first to document a voltage-gated channelopathy in association with previous thymic cancer (and not thymoma). Moreover, this is a patient presenting with two other other autoimmune syndromes, i.e. myasthenia gravis and myositis.

**P.039**
New association of anti-Hu positive limbic encephalitis and sensory ganglionopathy with small cell gastric tumour

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**Background:** While anti-Hu antibody associated encephalitis has been well-documented in association with a variety of neoplastic processes, paraneoplastic limbic encephalitis and sensory ganglionopathy with positive anti-Hu antibodies has not previously been linked in the literature with a neuroendocrine gastric tumour of advanced stage. **Methods:** Case report. **Results:** We present the case of an 86-year old woman who developed behavioural changes, paroxysmal anxiety attacks and poor balance several months after being diagnosed with poorly differentiated gastric small cell tumour in a clinical setting of weight loss and anemia. Anti-Hu antibodies were present. MRI showed signal abnormalities in the right mesial temporal lobe with contrast enhancement. Paroxysmal lateralized EEG changes were recorded and EMG/NCS showed absent sensory nerve responses. Behavioural symptoms stabilized under treatment with intravenous immunoglobulins, but sensory ataxia continued to worsen. The patient declined further therapy and deceased two months after transfer to palliative care. **Conclusions:** To our knowledge, this is the first report linking small cell gastric tumour with limbic encephalitis, sensory ganglionopathy-associated ataxia and anti-Hu antibodies. This description further broadens the clinical spectrum of anti-Hu syndrome.

**P.040**
Redefining true leukocytosis in the traumatic lumbar puncture

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**Background:** Clinicians rely on a correction formula (Predicted CSFWBC=CSFRBC×BloodWBC/ BloodRBC) to determine if a true CSF leukocytosis exists. This formula may overestimate true CSF leukocytosis leading to delayed diagnosis and treatment of meningiitis. **Methods:** A retrospective review of CSF data of 105 patients registered at 3 hospitals (Saskatoon, Canada) between 2011-2013 who met the following criteria: 1) CSF samples from lumbar puncture (LP) contained>1000 RBC/mm3; 2) a complete blood count (CBC) performed within 24 hours of LP; and 3) CSF not obtained due to high clinical suspicion of meningitis and was negative for microbial staining and culture. Regression analysis was performed to determine the relationship between actual and predicted CSF WBC values. **Results:** Mean adult age was 48.9 years; CSF profile (mean WBC

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