

visual deficits as a result of his condition for which he would undergo rehabilitation.

**Setting:** Rehabilitation hospital, Outpatient clinic.

**Results:** The patient was sent to a rehabilitation hospital requiring moderate to maximum assistance with daily activities and mobility. He had extensive daily rehabilitation with physical, occupational, and speech therapy services. The patient's dramatic recovery and outpatient follow-up will be further discussed in this report.

**Discussion:** Bickerstaff's encephalitis is closely related to Miller Fisher Syndrome but generally distinguished by encephalopathy and hyperreflexia. The reason for these differentiating symptoms is the involvement of the brain stem in the disease process of Bickerstaff's encephalitis. Both of these conditions along with pharyngeal-cervical-brachial-weakness have anti-GQ1b antibodies. There is debate over whether these are separate conditions or different manifestations of the same anti-GQ1b syndrome. Plasmapheresis and IVIG therapy have been found to be effective treatments in these patients. It is worth analyzing more examples of these conditions, especially those with a favorable outcome, in order to gain a better understanding of them.

**Conclusions:** Bickerstaff's encephalitis is a rare condition related to others in the GBS spectrum. It is important to look at potential factors affecting recovery in less studied disorders such as these in order to have a greater understanding of their treatment and resulting prognosis.

**Level of Evidence:** Level V

#### Poster 226

##### Unusual Presentation of Eight and a Half Syndrome in a Patient with Acute Pontine Medullary Stroke: A Case Report

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**Disclosures:** Phillip Acevedo: I Have No Relevant Financial Relationships To Disclose

**Case/Program Description:** This 68-year-old woman with a past medical history of Type II diabetes mellitus, hypertension, and previous left cerebrovascular accident with residual right hemiparesis presented to the ER with nausea and vomiting, hyperglycemia, and visual disturbances. Examination revealed one and a half syndrome presenting with right intranuclear ophthalmoplegia (INO) and horizontal gaze palsy. MRI of brain revealed an acute punctate infarct at the right dorsal pontomedullary junction in addition to chronic lacunar infarcts. On day 5, she was transferred to an inpatient rehabilitation facility. However, on rehab day number five, the patient developed acute right facial droop, incapable of closing her right eye, slurred speech and otalgia.

**Setting:** Inpatient Rehabilitation Facility.

**Results:** Neurology and ENT consults were obtained, MRI was repeated and progression of the right pontomedullary infarct with new right facial nerve enhancement was noted. Eight-and-a-Half Syndrome was diagnosed. A 7-day course of prednisone and valacyclovir with gabapentin for pain control was begun. Occlusion therapy with OT was initiated for her diplopia. On day 19, patient was able to be discharged home with resolving slurred speech and facial palsy.

**Discussion:** Eight-and-a-half syndrome is caused by a lesion in the dorsal tegmentum of the caudal pons involving the parapontine reticular formation or abducens nucleus and MLF, and the nucleus/fasciculus of the facial nerve. It is a rare presentation of pontine stroke. Our patient's delayed presentation of facial palsy made the diagnosis very challenging.

**Conclusions:** A rare case of "eight-and-a-half" syndrome is a unique combination of "one-and-a-half" syndrome plus ipsilateral fascicular cranial nerve seventh palsy. Thus, the recognition of the syndrome is

important for appropriate management and to ensure good functional outcomes.

**Level of Evidence:** Level V

#### Poster 227

##### Cauda Equina Syndrome after Lumbar Disk Herniation: A Case Report

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**Disclosures:** Siena Ona: I Have No Relevant Financial Relationships To Disclose

**Case/Program Description:** HR is a 36-year-old man with chronic back pain who was admitted for sudden difficulty in ambulating associated with nonspecific lower extremity numbness, bowel dysfunction and urinary retention progressing to inability to void. He had a history of persistent chronic back pain which was diagnosed as severe sciatica on prior visits to the ED.

**Setting:** 75 bed Rehabilitation Hospital.

**Results:** On day of admission, MRI showed massive extrusion of L3-L4 disk material, segmental stenosis and severe narrowing of compression of thecal sac. Patient then underwent emergent lumbar decompression for cauda equina syndrome. Postop patient's weakness improved but he was still having weakness on bilateral dorsiflexion, and still had bowel dysfunction requiring suppositories and bladder dysfunction requiring in and out cauterization.

**Discussion:** The presentation of cauda equina syndrome varies from its classical presentation, especially in its early stages of compression. In this case for example, the patient's symptoms were attributed to a severe case of sciatica. Delayed diagnosis and treatment of this syndrome could lead to poor outcomes. National annual incidence of low back pain is 5%. One quarter of patients with back pain have sciatica. The most common cause of sciatica is herniation of the lower lumbar intervertebral disks. In contrast to sciatica, cases of CES after disk herniation are relatively rare. In one study, the incidence of CES due to lumbar disk herniation has been reported to range from 1% to 10% of operated disk cases.

**Conclusions:** Acute compression of the cauda equina is a neurologically compromising and debilitating condition. Physicians who evaluate low back pain must be able to recognize the signs and symptoms of this relatively rare but critical spinal syndrome and must expedite emergent evaluation and intervention.

**Level of Evidence:** Level V

#### Poster 228

##### Zolpidem for the Treatment of Neurologic Disorders: A Systematic Review

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**Disclosures:** Martin Bomalaski: I Have No Relevant Financial Relationships To Disclose

**Objective:** To perform a systematic review of all studies using zolpidem to treat neurologic disorders not including insomnia.

**Design:** Eligibility criteria included any article examining the use of zolpidem for non-insomnia neurologic disorders on humans, published and unpublished, written in English, and for all dates up to March 2015. Searched databases included PubMed, Scopus, Web of Science, Cochrane Library, and Embase. Positive publication bias was mitigated by searching [ClinicalTrials.gov](http://ClinicalTrials.gov) for unpublished studies. Two rounds of screening were performed based on title and then abstract, respectively.

**Setting:** N/A.

**Participants:** N/A.

**Interventions:** N/A.

**Main Outcome Measures:** N/A.

**Results:** Initial combined search results produced 2398 articles. After exclusion based on title 83 articles remained. An additional 19 articles were excluded upon review of abstracts, leaving 64 articles for full manuscript review. There were 29 studies treating movement disorders, subdivided into dystonia (n=10), Parkinson's disease (n=6), progressive supranuclear palsy (n=6), spinocerebellar ataxia (n=2), catatonia (n=3), and tardive dyskinesia (n=1); 24 studies treating disorders of consciousness, from causes such as traumatic brain injury (n=5), anoxic brain injury (n=6), and other encephalopathies (n=2); and 11 studies treating other neurologic conditions including stroke (n=4) and dementia (n=2). Study designs included case reports or series (n=35), single subject interventional (n=14), randomized controlled trials (n=6), nonrandomized controlled trials (n=4), and cross-over studies (n=4). Only 14 studies had greater than five subjects. Sedation was observed in eight studies.

**Conclusions:** Zolpidem has been observed to treat a large variety of neurologic disorders, most often related to movement disorders and disorders of consciousness. Much of what is known comes from case reports and small interventional trials. Additional research with larger randomized controlled trials is needed to better understand zolpidem's efficacy in treating these conditions.

**Level of Evidence:** Level III

**Poster 229**

**Athlete with Compression of Thenar Motor Branch and Thumb Digital Branch of Median Nerve: A Case Report**

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**Disclosures:** Naglaa Hussein: I Have No Relevant Financial Relationships To Disclose

**Case/Program Description:** A 36-year-old right handed analyst man without significant past medical history, complained of numbness of left thumb x 1year, intermittent without nocturnal exacerbation. This was associated with atrophy of his left thumb muscles. He is still able to hold objects without difficulty. He also has neck pain x 6 months without irradiation to the arms. No muscle weakness or change in bowel or bladder function. His MRI for left wrist and hand was normal. He uses kettlebell for muscle strengthening regularly for several years. He also practicing shooting gun and riding a mountain bike without gloves for 2 hours once a week for about 10 miles for 3-4 y.

**Setting:** Outpatient.

**Results:** Diminished sensation only at tip of left thumb. Atrophy and weakness of left thenar muscles. Spurling's, tincl and Phalen tests were negative bilaterally. Electrodiagnostic studies revealed unobtainable sensory nerve action potential I(SNAP) of left median to thumb only. Intact all SNAP of other upper limb and lower limb nerves including left median to digit II & III. Motor conduction study (MCS) of left median nerve to abductor pollicis brevis showed marked prolonged distal latency. MCS of left median nerve to second lumbrical was normal MCS of both ulnar, right median nerve and left peroneal nerve were normal. All F waves were within normal limits. Needle EMG for left flexor carpi radialis, first dorsal interosseous, and second lumbrical showed normal study. Left abductor pollicis brevis showed distant motor units only, without abnormal rest potential. Ultrasound of left wrist did not reveal any abnormality.

**Discussion:** Electrodiagnosis suggested a lesion of left thenar motor branch as well as a digital sensory branch to thumb. Patient's athletic performance could be the reason of compression. No evidence of carpal tunnel syndrome.

**Conclusions:** Compression of thenar motor branch and thumb digital branch of median nerve.

**Level of Evidence:** Level V

**Poster 230**

**Unconventional Dosing of Amantadine in a Patient with Traumatic Brain Injury: A Case Report**

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**Disclosures:** Vandana Sood: I Have No Relevant Financial Relationships To Disclose

**Case/Program Description:** A 23-year-old woman sustained severe traumatic brain injury (TBI) as an unrestrained driver in a high-speed motor vehicle collision (Glasgow Coma Scale = 3). The patient was admitted to the Disorders of Consciousness Program in an acute rehabilitation hospital in a suspected minimally conscious state (MCS), approximately 2 months post-injury. On physical exam, she was alert, awake, non-verbal, with spontaneous visual tracking in the left visual field. There was no evidence of spontaneous communication or reproducible command following. She was started on amantadine 100mg twice daily to promote functional neurobehavioral recovery. The patient emerged from a MCS and began to functionally communicate, however responsiveness was variable, with reduced verbal output at times. Her Orientation Log (O-Log) score was 8 out of 30. On two occasions, her amantadine was held due to loss of GI access. Both times, after stopping amantadine, she demonstrated paradoxical improvement, demonstrated by her mental status examination scores, verbalizations, and improved behavior. However, on both occasions, after a few days off amantadine, her mental status worsened again. Given this pattern, she was transitioned to an alternate-day dosing regimen, and ultimately was discharged on amantadine 100 mg every other day.

**Setting:** Tertiary care rehabilitation hospital.

**Results:** On this regimen she continued to improve rapidly as demonstrated by consistent functional object use, improved verbal communication and improved participation (O-Log = 21).

**Discussion:** This case illustrates a novel approach to amantadine dosing for functional recovery following severe TBI. We suspect that this alternative dosing regimen led to a more optimal pharmacokinetic and/or pharmacodynamics profile in this particular patient, possibly related to individual variability in metabolism, receptor occupancy, or other factors. The standard dosing regimen produced less cognitive improvement.

**Conclusions:** Conventional amantadine dosing twice daily may not be optimal for all patients with severe TBI. Alternative regimens should be studied for potentially better functional outcomes.

**Level of Evidence:** Level V

**Poster 231**

**Hemiballismus as the Presenting Symptom of Polycythemia Vera: A Case Report**

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**Disclosures:** Emma Nally: I Have No Relevant Financial Relationships To Disclose

**Case/Program Description:** A previously healthy 74-year-old man presented to the emergency department with acute onset of isolated right hemiballismus. Magnetic resonance imaging showed acute infarct in the left globus pallidus. Laboratory studies revealed hemoglobin of 16.8, hematocrit of 51, platelets of 708, and positive JAK2 V617F mutation; meeting diagnostic criteria for Polycythemia Vera (PV). Therapy was initiated with aspirin, statin, and hemodilution with resolution of slurred speech but persistence of hemiballismus. He was admitted to acute inpatient rehabilitation as he required an interdisciplinary approach to address his complex physical needs due to persistent hemiballismus in the absence of sensory or other motor deficits and medical management of PV.