and deformation of the medulla. Patient has been successfully treated with ASV.

1118 CENTRAL SLEEP APNEA IN A PATIENT WITH AN ACOUSTIC NEUROMA
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Introduction: Several reports have described central sleep apnea (CSA) or hypoventilation in patients with brainstem lesions. In one case, central hypoventilation occurred in a patient with acoustic neuroma and brainstem compression. We present a patient with CSA associated with acoustic neuroma without apparent brainstem compression.

Report of Case: A 50-year-old obese man with a history of acoustic neuroma was referred to sleep clinic with complaints of snoring, witnessed apneas, and daytime somnolence. Symptoms attributed to the tumor included reduced hearing and vertigo with left facial palsy and numbness. MRI reports described the lesion as a 1.3 x 0.7 x 0.7 cm mass located within the left internal auditory canal arising from the inferior vestibular nerve complex without evident brainstem compression. Split-night polysomnography demonstrated a baseline apnea hypopnea index (AHI) 76/hr, 48/hr central/mixed apneas and 16/hr hypopneas with an atactic pattern. During CPAP titration to 12 cm H2O, AHI decreased to 6.7/hr, but with continued reduced oxygen saturation (89%). Pulmonary function tests and an ABG determination showed a mild restrictive defect and PaCO2 34 mmHg. He was subsequently successfully titrated to ASV with 4 L O2, but was unable to tolerate it during a 12-month trial. He related that oxygen alone improved his symptoms. Ambulatory sleep testing on 4 L O2 demonstrated persistent respiratory events (REI 76.7/hr). He was then started on CPAP 12 cm with 4L O2 bleed-in. At the last clinic visit, the patient demonstrated improved PAP adherence and daytime symptoms with a residual AHI 7.9/hr.

Conclusion: In this case, an acoustic neuroma without apparent brain stem involvement was associated with CSA in an atactic pattern. We postulate that focal tumor effects on the pontine pneumotaxic and apneustic centers may have led to sleep disordered breathing.

1119 NOCTURNAL NON-INVASIVE VENTILATION (NNIV) FOR TREATING HYPERCAPNIC RESPIRATORY FAILURE
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Introduction: NNIV is a widely accepted treatment for sleep-related hyperventilation. We report two cases with disparate medical conditions in which NNIV treatment was effective in treating acute-on-chronic hypercapnic respiratory failure.

Report: A 73-year-old woman with severe kyphoscoliosis presented to the emergency department with worsening dyspnea. ABG on supplemental oxygen showed pH 7.37, pCO2 63 mmHg/L, pO2 89 mmHg/L, HCO3 36 mmol/L. Chest imaging was negative for pulmonary embolism and suggestive of pulmonary hypertension. Frequent nocturnal awakenings due to headaches prompted polysomnography, which revealed poor sleep efficiency, AHI 0/hour and oxygen nadir of 81%. Introduction of nocturnal BIPAP S/T titrated to 14/8 cm H2O, with a back-up rate of 10/hour and supplemental oxygen led to improvement in sleep efficiency and gas exchange. ABG on room air after 60 days of NNIV revealed pH 7.41, pCO2 50 mmol/L pO2 51 mmol/L, HCO3 31 mmol/L. The patient reported greatly improved sleep with fewer nocturnal awakenings, and increase in daytime energy and function.

A 48-year-old woman with lymphoma was hospitalized for somnolence and hypoxia with ambulatory O2 saturation 90%. Chest imaging excluded pulmonary embolism. ABG on supplemental oxygen showed pH 7.22, pCO2 97 mmol/L, pO2 129 mmol/L, HCO3 39 mmol/L. BIPAP S/T was started in the emergency department, and eventually titrated to 12/5 cm H2O with a back-up rate of 12/hour. Brain MRI showed diffuse leptomeningeal involvement. A portable sleep study revealed REI 0.65/hr, oxygen nadir of 62%, and Biot’s respiration suggestive of CNS etiology. She was continued on NNIV with supplemental oxygen. ABG, 20 days later, revealed pH 7.46, pCO2 51 mmol/L, pO2 62 mmol/L, HCO3 36 mmol/L. She reported improved daytime energy and function.

Conclusion: In these two patients with unrelated medical conditions, acute-on-chronic hypercapnic respiratory failure responded to NNIV with improvement of gas exchange, symptoms, and quality of life.

1120 PARASOMNIA TRIFECTA: RBD, NIGHTMARES AND SOMNAMBULISM
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Introduction: Case of mirtazapine treatment resulting in REM-behavior disorder, nightmares and somnambulism in a patient without Parkinsonism.

Report of Case: 67-year-old male Vietnam Veteran on treatment for major depressive disorder (MDD) and post-traumatic stress disorder (PTSD) was found to have mild OSA (AHI 13.9) with poor compliance on autoPAP therapy due to claustrophobia with the mask and frequent episodes of dream enactment. The patient reported a temporal relationship with his nocturnal disruptive behaviors and commencement of mirtazapine. The mirtazapine dose had recently been increased to 45mg which the patient associated with worsening somnambulism, RBD and nightmares. Additionally, worsening insomnia was associated with twice daily buproprion SR use. In consultation with the psychiatrist, we recommended buproprion XL once daily in the morning, which resulted in significant and swift improvement in insomnia, and initiated a mirtazapine taper, with goal to discontinue the medication. As the mirtazapine dose was reduced, the patient reported significant reduction in nocturnal disruptive behaviors, including dramatic improvement of RBD and significant reduction in nightmares and somnambulism. Additionally, low-dose clonazepam was initiated transiently to prevent withdrawal symptoms such as rebound anxiety and irritability from the alpha-2 antagonist taper, to decrease arousals and to improve CPAP compliance.

Conclusion: Medications used to treat mood disorders can result in significant sleep comorbidities. Identifying appropriate medications that can treat the mood and administering them at appropriate times can result in maximal benefit with minimal side effects to the patient.

1121 COBALT BLUES: A CASE REPORT OF COBALT INTOXICATION ASSOCIATED OBSTRUCTIVE SLEEP APNEA (OSA).
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**Introduction:** A recent study found serum levels of heavy metals were higher in patients with severe OSA.

**Report of Case:** 56-year-old man was referred for complaints of snoring, daytime sleepiness, headache and depression. In 2009, he had a left cobalt-chromium hip replacement for degenerative joint disease. In 2012, he developed insomnia, headache, crying spells, decreased concentration, and loss of appetite. Extensive diagnostic testing was unremarkable. He was prescribed fluoxetine for depression and propranolol for headaches without benefit. A Home Sleep Apnea Test was done 2/15/16 showed a respiratory event index (REI) of 30/hr. APAP was prescribed with good adherence and a residual REI of 1.8. However, all his symptoms persisted and a heavy metal screen was ordered. It showed a markedly elevated serum cobalt level 1.6 mcg/l (normal < 0.4 mcg/l) and coupled with his symptoms, cobalt intoxication. He underwent a left ceramic hip replacement on 5/19/16. Following surgery, all his symptoms resolved along with normalization of his cobalt levels. He requested a repeat HSAT which showed REI 12. We discontinued PAP therapy without recurrence of his sleep/wake complaints.

**Conclusion:** This patient presented with symptoms commonly associated with cobalt toxicity. Cobalt ions from metal-on-metal wear of cobalt prosthetic hips can cross into the systemic circulation and cause cobalt intoxication. The mechanism for OSA in cobalt toxicity could be explained by excessive trace metals released by the damaged tissue inducing oxidative stress and lead to a vicious cycle which reinforces chronic inflammation and OSA.

**1122**

**ACUTE ONSET STATUS CATAPLECTICUS IN AN ELDERLY MAN ORIGINALLY DIAGNOSED WITH SNCYPCE**

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**Introduction:** Narcolepsy with cataplexy can be the great masquerader of patient complaints with a diverse spectrum of presentations. We report acute onset status cataplecticus in a 64 year old gentleman with no prior history of a central hypersomnolence disorder. This case highlights the need for consideration of central hypersomnolence disorders secondary to a medical condition in the differential diagnosis of syncope.

**Report of Case:** 64 year-old patient, noted to have acute onset of paroxysmal episodes of loss of consciousness (LOC) presented to the emergency department. He denied prior history of syncope and noted a severe headache, dysarthria, and blurry vision preceding the onset of the episodes. The patient described predictable paroxysmal episodes of LOC in response to strong emotion. At an episode onset, he described relaxation of facial muscles that progressed into relaxation of lower extremity muscles, followed by a few seconds of LOC. The patient related that he could quickly return to consciousness without confusion if someone was present to rouse him. While hospitalized, he underwent a comprehensive work up for syncope that did not elucidate a discreet etiology for the episodes of LOC. Polysomnography and multiple sleep latency test (MSLT) were later performed and suggestive of narcolepsy with cataplexy with mean sleep onset latency of 2 minutes and 45 seconds and 4 sleep onset REM periods. The paroxysmal episodes the patients had described were captured on video-electroencephalography EEG monitoring in between the nap trials of his MSLT. MRI of the brain revealed white matter changes in the hypothalamus.

**Conclusion:** Narcolepsy with cataplexy due to a medical condition (NDMC) has been documented in patients with multiple sclerosis when plaques impair the hypothalamus, paraneoplastic syndrome, and Niemann-Pick type C disease. This case demonstrates a unique presentation of NDMC and highlights the need to consider central hypersomnolence disorders in the differential diagnosis even in advanced age populations.

**1123**

**A RARE CASE OF LATE PRESENTATION OF POST TRAUMATIC NARCOLEPSY**

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**Introduction:** Narcolepsy with cataplexy is a serious disabling sleep disorder characterized by excessive daytime sleepiness, abnormal rapid eye movement and sleep attacks. The development of human narcolepsy involves environmental factors acting on specific genetic background.

One of such environmental factor is traumatic brain injury. The patient in the case report had late presentation of narcolepsy several years after traumatic brain injury.

**Report of Case:** 39-year-old Male presented with 1 year history of progressively worsening excessive daytime sleepiness including while driving. Episodes were characterized by sleep attacks and sleep paralysis. No associated cataplexy and no hallucination. Patient also reported history of traumatic brain injury he sustained after falling off motorbike with extensive hospital stay but with full recovery 12 years prior to sleepiness symptoms. Physical Examination findings were normal with exception of postural head scar.

He subsequently had attended polysomnography which showed AHI 5.3 with 02 nadir 88% and no other abnormal findings on sleep study. Patient was subsequently started on AutoCPAP with no improvement in sleepiness symptoms reported with Epworth sleepiness scale 20. Overnight Polysomnogram with next day Multiple sleep latency test was then performed which showed 4 SOREMPS and mean sleep time latency of 4.5 minutes confirmatory of narcolepsy without cataplexy.

**Conclusion:** Hypersomnia can result when areas involving the maintenance of wakefulness are injured such as rostral pons, caudal midbrain and the thalamus. Irshad et al (2005) reviewed previous reported cases of posttraumatic narcolepsy with report indicating duration of narcolepsy symptom onset from the index event varied between few hours to 18 months.

The rarity of post traumatic narcolepsy in this case is due to the late presentation of Narcolepsy several years after traumatic brain injury. Our patient was started on modafinil.

**1124**

**NON-SURGICAL MANAGEMENT OF OBSTRICTIVE SLEEP APNEA IN A CHILD WITH DOWN SYNDROME**

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**Introduction:** Obstructive Sleep Apnea (OSA) occurs in at least 30 to 75 percent of children with Down Syndrome (DS). The mechanism includes soft tissue and skeletal alterations that lead to upper airway obstruction. Untreated childhood OSA not only affects cognitive development, behavior and quality of life but can also lead to cardiovascular and metabolic consequences. Routine OSA screening and evaluation should be performed and addressed in all DS children.

**Report of Case:** 14-year-old African American child with history of DS presented with the concern of loud snoring, apneic pauses, fragmented sleep and daytime somnolence. Physical exam revealed oropharyngeal crowding, tonsillar hypertrophy 3+. Polysomnogram (PSG) demonstrated severe OSA with AHI of 22.4, oxygen nadir of 60% and transtheanous CO2 elevation and was not able to tolerate positive airway pressure (PAP) trial. Tonsillectomy and adenoidectomy was