despite occurrence of above-mentioned episodes increasing the clinical suspicion of Narcolepsy. Polysomnography revealed sleep latency of 0 minutes, REM latency of 2.6 minutes, and mild OSA. Toxicology screen was negative. MSLT showed mean sleep latency of 1.2 minutes with 3 SOREMPs. CSF orexin could not be obtained due to technical constraints. He was diagnosed with Narcolepsy type-1 and started on Venlafaxine and Methylphenidate with resultant improvement in both daytime sleepiness, and the severity and frequency of cataplexy. However, his feeling of imbalance persisted, prompting repeat video EEG which demonstrated left temporal lobe seizure activity. Anti-epileptic regimen was optimized with improvement in sensation of imbalance.

Conclusion: Temporal lobe epilepsy and Narcolepsy Type-1 can have overlapping features that make it difficult to distinguish between the two. While exceedingly rare, the two entities may co-occur. Diagnostic and therapeutic management require coordination between Sleep Medicine and Neurology. While PSG, MSLT, and CSF Orexin can clarify the diagnosis of narcolepsy, neurologic work up should be pursued if symptoms are not completely attributable to Narcolepsy.

Support (If Any):

0853

THE ROLE OF SLEEP-DISORDERED BREATHING IN THE OPTIMIZATION OF PEDIATRIC EPILEPSY MANAGEMENT

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Introduction: There is a known bidirectional relationship between epilepsy severity and disordered sleep; however, both the potential scoring limitations of polysomnograms (PSGs) of children with epilepsy and the effect of treatment of sleep disordered breathing (SDB) on epilepsy severity in these children remain poorly understood.

Report of Cases: We describe a 7-year-old male with a history of prematurity (ex-34 weeks), bronchopulmonary dysplasia, recurrent aspiration pneumonia, and refractory nocturnal epilepsy who experienced acute hypoxemic respiratory failure in the setting of co-infection with rhinoenterovirus and parainfluenza 4 virus. The patient's epilepsy history is notable for multiple nocturnal seizures most days of the week for a period of several years. His mother endorsed that the nocturnal seizures began shortly after his SDB symptoms, and that the severity of his nocturnal seizures seemed to be associated with the quality of his sleep. Two years prior to admission, he underwent adenotonsillectomy for mild obstructive sleep apnea (AHI 2.5, REM AHI 8.9, minimum SpO2 86%). He continued to have persistent SDB symptoms (e.g. witnessed apneas, nocturnal arousals 5-10 times/night, daytime sleepiness) despite adenotonsillectomy. Notably, the background EEG (in the context of epilepsy) demonstrated obscured sleep architecture and led to unreliable staging of sleep states and cortical arousals. Given the disorganized background EEG, arousal-based hypopneas could have been underestimated. In the setting of acute respiratory failure, our patient was hospitalized and treated with noninvasive ventilation for 6 days. Collateral benefits included improvements in SDB (evidenced by decreased nocturnal awakenings, lack of obstructive features) and reduction in seizure frequency. His mother noted that this hospitalization was the first time in years that her son did not experience nocturnal seizures.

Conclusion: Sleep apnea can be an important modifiable factor in the treatment of pediatric epilepsy. Some PSGs of children with epilepsy may be falsely reassuring given the potential difficulty with scoring cortical arousal-based hypopneas. Therefore, careful clinical correlation should be made between nocturnal seizures and sleep symptoms, even in the context of reassuring PSG parameters.

Support (If Any): Cystic Fibrosis Foundation Training Grant, AIRE grant, NIH 2T32HL110952-06

0854

A CASE OF COMPLETE HEART BLOCK IN A PATIENT WITH SEVERE OBSTRUCTIVE SLEEP APNEA

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Introduction: Obstructive sleep apnea (OSA) has been associated with bradycardic arrythmias and cases of transient heart block. Heart block occurring during sleep has been described in up to 10% of patients with obstructive sleep apnea, most commonly during stage R sleep and through a mechanism known as vagal activation. Stage R sleep is associated with desynchronization of respiratory and cardiovascular functions, and the excessive autonomic response (vagal activation) occurs due to the stronger stimulation of chemoreceptors and baroreceptors induced by hypoxemia, intrathoracic pressure swings, and hemodynamic alterations. Treatment of the underlying sleep-disordered breathing will typically treat the bradycardic arrythmia as well.

Report of Cases: A 49-year-old male with a BMI of 30 kg/m , neck circumference of 16 inches, and obstructive sleep apnea presented to the sleep center to establish care for management of his preexisting OSA. He was previously diagnosed with moderate OSA in 2007 but had discontinued therapy shortly after due to Continuous Positive Airway Pressure (CPAP) intolerance. The patient complained of snoring, nocturia, and excessive daytime sleepiness (18/24 on Epworth Sleepiness Scale). During the split night study, the patient was noted to have 2:1 second degree heart block followed by a transient episode of complete heart block, manifested by 4 non-conducted p-waves. The patient remained asymptomatic throughout the night. Upon study completion, the patient denied any prior chest pain, presyncope, syncope, or medications that might cause heart block.

Conclusion: Bradyarrythmias can be seen in severe obstructive sleep apnea in the setting of excessive vagal stimulation through increased stimulation of chemoreceptors and baroreceptors. Treatment with PAP therapy can lead to prevention of heart block in 80-90% of these patients.

Support (If Any):

0855

A CHALLENGING CASE OF NOCTURNAL VISUAL HALLUCINATIONS IN AN ELDERLY WOMAN

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Introduction: Nocturnal hallucinations can be part of a wide array of different disorders, like sleep disorders (e.g. narcolepsy), visual impairment (e.g. Charles-Bonnet syndrome), neurodegenerative disorders (e.g. Lewy Body Dementia) and psychiatric disorders

(e.g. schizophrenia). We explore the differential diagnosis, the challenging diagnostic workup and the limited therapeutic options in this patient with complex visual nocturnal hallucinations.
Report of Cases: A 64 year old woman was referred with detailed and vivid visual nocturnal hallucinations (seeing her dog smoking a cigar, seeing someone in the room moving her bed, etc.). The hallucinations started 1-2 months ago, were mainly present at night (both when falling asleep and during the night) but occasionally also during the day. She had also been suffering from worsening daytime sleepiness for 3-4 months, and from memory problems for 1 year. Cataplexy and sleep paralysis were absent. Clinical neurological examination was normal. The patient had been diagnosed in the past with OSAS. She had been treated with CPAP for 3 years and recent polysomnography showed good control of her apnea. She had also been diagnosed with Crohn's disease, COPD and cardiomyopathy. Because of her age, the vivid hallucinations, and the memory problems, we first wanted to rule out Lewy Body Dementia. Neuropsychological testing ruled out dementia. MRI of the brain showed some white matter lesions, without substantial atrophy. At a subsequent multidisciplinary consultation, her cardiologist considered her cardiomyopathy as a contraindication for stimulants. Therefore, and because we believed the a priori probability for narcolepsy to be low, an additional MSLT was not considered useful. Based on further pulmonary tests, we hypothesized that hypoxia could play a role in the hallucinations and hypersomnolence. The hallucinations improved over time, the hypersomnolence remained, but we found there were no good treatment options available, considering the cardiac contraindications for stimulants.
Conclusion: There is a wide-ranging differential diagnosis for nocturnal visual hallucinations. The specific patient characteristics are important both for further diagnosis and treatment options.
Support (If Any): Supported by the Funds Malou Malou, Perano, Georgette Paulus, JMJS Breugelmans and Gabrielle, François and Christian De Mesmaeker, Managed by the King Baudouin Foundation of Belgium, No. 2021-J1990130-222081.

0856

CORRECTING SLEEP/WAKE CYCLE AND REDUCING COMBATIVENESS IN AN 84-YEAR-OLD PATIENT

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Introduction: Sleep disruption is a common occurrence in hospitalized geriatric patients. Such patients frequently arrive at the emergency department for behavioral changes due to delirium, which can often be mistaken for, or compounded by, underlying dementia. Behavioral symptoms may vary, including disinhibition, aggression, distress during care, mood disturbance, and paranoia, affecting approximately 90% of patients. Effectively treating these patients is challenging; antipsychotic use is a commonly implemented treatment in hospitalized settings, but is complicated by adverse side effects and an increased risk of CVA. In this case report, we discuss the use of an antipsychotic, olanzapine, to treat an 84 year old hospitalized patient presenting with sleep disruption and changes in mental status.
Report of Cases: An 84 year old gentleman with a history of Alzheimer's dementia with worsening behavioral changes was admitted for urinary tract infection, humeral fracture secondary to mechanical fall, and increasing aggression toward his caretaker. At home the patient was sleeping during the day and more was active and combative at night. While hospitalized, he had become aggressive with hospital staff due to his distress and was minimally oriented at baseline. We trialed the patient on olanzapine 5mg at night to facilitate sleep and reduce his aggression, in addition to environmental changes to reorient day and night. After a few days of this treatment, this patient was much more pleasant and oriented, able to hold conversation, and no longer aggressive. His initial disposition was to be transferred to a memory care facility, given increasing safety concerns expressed by the caretaker. However, following these changes, he was able to return home in the comfort of his family and caretaker.
Conclusion: Antipsychotic use in the elderly for sleep disruption and behavioral disturbances is heavily debated, though there may be some benefit to their use. The CATIE-AD trial and a Cochrane Review suggest Olanzapine may reduce aggression, particularly in Alzheimer's disease, and has the added benefit of sedation which aids in establishing sleep cycles. However, many patients discontinue antipsychotic treatment due to side effects and routine use is discouraged. When in use, regular monitoring should take place to evaluate for side effects associated with pharmacotherapy and continued therapeutic benefit should be reassessed.
Support (If Any):

0857

HOW TO FIX MASK LEAK FOR THOSE WITH NO TEETH

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Introduction: Mask fit is a crucial factor for patient adherence and effective use of PAP therapy. However, patients using dentures may face significant challenges to achieving a good mask seal, resulting in mask leak and inefficacy of therapy.
Report of Cases: A 67-year-old male with Obstructive Sleep Apnea (OSA) previously adherent to Bi-level Positive Airway Pressure (BPAP) therapy presented to clinic for complaints of mask leak. Due to a suspected dental infection, he had had all his teeth removed three years prior to this visit and had been instructed by his dentist to remove the dentures each night when he slept. However, removal of the dentures affected the fit of his full-face mask, and he experienced significant mask leak with return of pre-PAP symptoms. He was a habitual mouth-venter and preferred full-face masks. Despite trialing different masks, he was no longer able to tolerate his BPAP secondary to the leak and discontinued PAP therapy altogether. During his visit to the sleep center, a mask refit was performed with and without his dentures in place. While no masks sealed effectively with his dentures out, a satisfactory alternative was found if he wore his dentures. He was counseled to wear his dentures while using PAP, remove his dentures for the recommended minimum amount of time during the day instead of removing them for sleep, and to follow up with his denturist/prosthodontist for further instructions.
Conclusion: Edentulous patients may pose difficulties with mask fit, and a multi-modal approach may be necessary to effectively treat their sleep apnea. Luckily, our patient was able to find a mask that he found comfortable while wearing his dentures and had minimal leak. Alternatives may include consideration of positional therapy with lower pressures for a potential nasal mask, weight loss, hypoglossal nerve stimulator, or referral to an oral surgeon, denturist, or prosthodontist for guidelines on sleeping with dentures. Close monitoring of symptoms and download data is recommended following oral surgery and switch of mask interface.
Support (If Any):