Abstract:
Epilepsy is the 4th most common neurological disease in the USA. The link between epilepsy and sleep has been known for a long time. An EEG after sleep deprivation is often used as a diagnostic tool in epilepsy. Sleep disorders have an increased incidence in epilepsy patients. It is known that sleep deprivation can precipitate an epileptic seizure. However, it has not yet been proven that CPAP, or any other modality of sleep disorder treatment, has led to better seizure control in epilepsy patients. In this report, we bring the case of a patient who has achieved excellent seizure control by using CPAP.

KEYWORDS: epilepsy, CPAP, obstructive sleep apnea, pharmacoresistant

Case Report

CPAP as a modality of epilepsy treatment in a patient suffering from obstructive sleep apnea

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Sažetak:

Ključne riječi: epilepsija, CPAP, obstruktivna apnea, farmakorezistentna
INTRODUCTION
The link between epilepsy and sleep has been thoroughly described. Its complexity is evident from the fact that while sleep deprivation can precipitate epileptic seizures, some epileptic seizures happen more often during sleep. The frequency of those seizures depends on the phases of sleep, the frequency of some is greater in phases of light sleep and others in deep sleep (1) sleep and sleep disorders. Recent studies have provided new insights into the links between the disorders that may facilitate differential diagnosis and treatment but may also improve our understanding of underlying pathophysiological mechanisms. Recent findings: Sleep and sleep deprivation have long been recognized to influence interictal epileptiform discharges and seizures. More recent studies have shown that primary sleep disorders such as obstructive sleep apnoea may worsen epilepsy and treatment of these sleep disorders can lead to improved seizure control. Seizures may interfere with night-time sleep structure and cause excessive day-time somnolence (EDS). Nocturnal seizures can lead to sleep fragmentation and thus cause EDS (eng. Excessive Daytime Sleepiness). Furthermore, it is proven that OSA (eng. “Obstructive Sleep Apnea”) could be twice as common in patients with epilepsy (2). A modality of OSA treatment is CPAP (eng. Continuous Positive Airway Pressure), a machine used to keep the air pressure in the airway positive, thus inhibiting airway obstruction in OSA.

CASE REPORT
In this report, we bring the case of a 62 y.o. male who presented to our emergency ward after a nocturnal seizure (December 13, 2018). His wife stated that he started “to snore”, and moments after that, he became rigid, did not respond to her calls, and had urine incontinence. When the seizure passed, he was confused and did not recognize his wife. Pertaining to his medical history, he has been treated for arterial hypertension, diabetes, atrial fibrillation and hyperlipoproteinemia, and the bloodwork at the date of the first seizure was as expected, considering his medical history. Brain CT (eng. Computerized Tomography) revealed chronic small vessel disease. Following this, an electroencephalography showed focal slowing localized fronto-centro-parietally. At this point, a Magnetic Resonance Imaging of the brain was done that corroborated the finding of small vessel disease, and the slices through the basal parts of the temporal lobes, as per the epilepsy protocol found no morphological abnormalities. Polysomnography was done due to the patient’s history of snoring. The AHI (eng. Apnea-Hypopnea Index) was 55.3, and during CPAP use, it fell to 4. At this point, the patient was prescribed a CPAP device, and during the delivery period, he had two tonic-clonic seizures of unknown onset (February 26, 2019). Levetiracetam was prescribed in the dose of 500mg BID with gradual ramp-up. Later, the CPAP was delivered, and he had a check-up at a sleep centre (June 6, 2019), at which point he had a satisfactory AHI, a somewhat raised central event index that was probably caused by his cardiac comorbidities and was advised to resume using the CPAP. Ever since he started using the CPAP, he had only one tonic-clonic seizure that happened when he was not using the CPAP device. Otherwise, no seizures were reported when using the CPAP.

DISCUSSION
In the case of our patient, CPAP was the therapeutic modality that allowed the best possible seizure control. As it can be seen, the same was not achieved solely through pharmacotherapy. Furthermore, the only reported seizure after the introduction of CPAP happened on the one night the patient was not using the device. Even though a cause-effect link could not be established after a single case, some of the variables in this interaction have been explored earlier. The interaction of CPAP therapy and seizure frequency hasn’t yet been made crystal clear. In their retrospective study, Li et al. have proven that patients with pharmacoresistant epilepsy have had a statistically significant improvement in seizure control while on CPAP (3) and treatment of OSA may decrease seizure frequency. However, it is unclear whether patients with medically refractory epilepsy have a higher incidence of OSA compared with well-controlled epilepsy patients and whether the two groups carry different risk factors. Purpose: This study aimed to investigate the presence of OSA in patients with refractory vs. well-controlled epilepsy and their associated risk factors. We also assessed the benefits of treatment of OSA with continuous positive airway pressure (CPAP). Malloy et al. have made a pilot study that had used a sham-CPAP device to conduct a randomized study. They have shown that 2 out of 5 patients with epilepsy with at least a mild form of OSA, and were using a CPAP device had an at least 50% reduction in seizure frequency. However, due to a small number of participants, no statistically significant result has been shown (4). Pornsriyom et al. have shown in their retrospective study that in patients who have epilepsy and OSA and were using a CPAP device, there was at least 50% reduction in seizure frequency in 74% of patients, compared to 14% of the same patients without a CPAP device (5). The meta-analysis done in 2016 by Lin et al. has shown that the number of positive outcomes in patients with epilepsy using a CPAP device was statistically significantly better than in epilepsy patients without a CPAP device (6). Hollinger et al. have done a retrospective study where they had taken data from the medical records of the sleep centre they worked at. By searching the records, they found 12 patients with an onset of OSA at the same time they had had an increase in seizure frequency; or had the first onset of an epileptic status. Out of those 21 patients, 12 had satisfactory compliance using the CPAP, and 4 of those patients had a decrease in seizure frequency (7). However, the only reported seizure after the introduction of CPAP was the therapeutic modality that allowed the best possible seizure control. As it can be seen, the same was not achieved solely through pharmacotherapy. Furthermore, the only reported seizure after the introduction of CPAP happened on the one night the patient was not using the device. Even though a cause-effect link could not be established after a single case, some of the variables in this interaction have been explored earlier. The interaction of CPAP therapy and seizure frequency hasn’t yet been made crystal clear. In their retrospective study, Li et al. have proven that patients with pharmacoresistant epilepsy have had a statistically significant improvement in seizure control while on CPAP (3) and treatment of OSA may decrease seizure frequency. 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center to identify patients with both sleep apnea and epilepsy. Characteristics of epilepsy, sleep history, presence of excessive daytime sleepiness [Epworth Sleepiness Scale (ESS). Another link between OSA and epilepsy can be seen from the epidemiology of those two diseases. The incidence of OSA is greater (10-15%) in patients with epilepsy than in the general population (4%). Mallow et al. have shown that a third of the patients who were candidates for neurosurgical treatment of epilepsy had OSA and have more frequent nighttime seizures than those without OSA (8) including validated measures of sleep-related breathing disorders (Sleep Apnea Scale of the Sleep Disorders Questionnaire [SA/SDQ]).

**Conclusion**
Sleep and epilepsy are tightly linked: a known method of seizure provocation is sleep deprivation; furthermore, the epidemiological data support the link. Even though CPAP treatment has not yet been proven to reduce seizure frequency in patients with epilepsy and OSA, there are multiple papers point in that direction. We had decided to go down that alley our patients’ treatment and have had excellent results. Hence, we conclude that polysonography should be considered in patients with epilepsy as a diagnostic method for OSA, and CPAP should be considered as a treatment method.

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**Conflicts of interest**
There were no conflicts of interest during the weighting of this paper.

**References**