



CPAP TREATMENT & EPILEPSY

by Regina Patrick RPSGT

A seizure is a sudden, temporary disturbance of neuronal signaling in the brain. The disturbance may result in a loss of consciousness or altered consciousness; abnormal motor phenomenon; autonomic nervous system impairment such as incontinence; and sensory disturbances such as auditory, olfactory, or visual hallucinations. If seizures are recurrent, a person may be diagnosed with epilepsy. A factor that can contribute to some types of seizures is insufficient sleep. Although obstructive sleep apnea (OSA) can be a cause of insufficient sleep, it is rarely suspected as playing a role in seizures. But recent research indicates that treating OSA may help some epileptic people with OSA gain better seizure control.

Seizures can be partial or generalized. A seizure is classified as partial if the origin of the dysfunctional signaling is limited to a discrete area (i.e., the focus) in the brain. A seizure is classified as generalized if the dysfunctional signaling involves a large area of the brain (e.g., one hemisphere) or encompasses the whole brain.

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A partial seizure may be either simple or complex. In simple partial seizure a person maintains consciousness throughout the episode but impaired neuronal firing may manifest in a variety of ways depending on the location of the focus and the pathway the signals travel. The person may have uncontrolled motor phenomenon (e.g., twitching of muscles); sensory dysfunction (e.g., olfactory hallucination such as smelling rotten meat); autonomic symptoms (e.g., urinary urgency); or psychic signs (e.g., sense of déjà vu [having experienced something before]). In a complex partial seizure, an episode may begin with an "aura" (which is in actuality a manifestation of a simple partial seizure) but as it continues, the person may have altered consciousness or may lose consciousness. For example, the person may have automatisms (i.e., involuntary aimless behavior) during a complex partial seizure but later have no memory of it.

Both simple and complex partial seizures can progress to a generalized seizure. A generalized seizure often involves a loss of consciousness. At its simplest, a generalized seizure may involve a one to ten second lapse of awareness after which the person may continue the pre-seizure activity unaware of the passage of time. However, during the seizure onlookers may note the person staring unresponsively or blinking or rolling his eyes. A tonic-clonic seizure is a generalized seizure at its worst. This type of generalized seizure begins with a sudden rigid extension of muscles which is followed by violent shaking, the result of rhythmic contractions. A tonic-clonic seizure may last from two to five minutes. Afterwards, a person may be confused, be extremely sleepy, or may complain of

headache or muscle aches or muscle weakness. Other types of generalized seizures are: tonic seizures which involve muscle rigidity but are not followed by a clonic phase; clonic seizures which involve violent rhythmic contractions but is not preceded by tonic phase; myoclonic seizures in which a person has brief involuntary jerking of the torso or extremities; and atonic seizures in which the skeletal muscles lose all tone causing the person to suddenly drop to the floor.

Complex partial seizures and generalized seizures are the most susceptible to being triggered by sleep deprivation. In some people with these types of seizures, good sleep hygiene can lower or eliminate the frequency of seizures. However even with good sleep hygiene, sleep disorders such as OSA can disrupt sleep thereby causing insufficient sleep, which then may contribute to seizures.

In obstructive sleep apnea, a person stops breathing (i.e., has apnea) for brief periods of time intermittently during sleep. This occurs because upper airway muscles relax too much during sleep allowing upper airway tissues such as the tonsils and adenoids to block the airway. With airflow blocked, the blood oxygen level falls. Once the blood oxygen falls to a certain level, the respiratory centers in the brain trigger an arousal. The arousal restores muscle tone of the upper airway which opens the airway allowing the person to take some deep breaths and restore the blood oxygen level to normal. Once restored, the person resumes sleep which may then allow apnea to recur.

The repeated arousals result in insufficient sleep (i.e., sleep deprivation). Various researchers have found increased activation of brainstem structures such as the thalamus during a sleep-deprived state. The thalamus plays a role in relaying signals involved in consciousness and sensory signals (except smell) from brainstem structures to the cerebral cortex and it relays motor signals from the cerebral cortex to the spinal cord. Hyperexcitability (i.e., excessive excitation) of the thalamus can set the stage for a seizure.

Several researchers have reported improved seizure control in OSA patients with epilepsy after their sleep apnea had been treated. Orrin Devinsky et al. reported the successful reduction of seizures in four patients treated with CPAP. Roy G. Beran et al. reported four patients in which CPAP treatment resolved or greatly reduced the frequency of seizures. Despite this, scientists remain unsure to what extent CPAP treatment may reduce seizures since the studies did not involve a control group. With this in mind, Dr. Beth Malow et al. of Vanderbilt University in Nashville, TN recently focused on designing a controlled, double-blind study to test whether a sham CPAP machine could feasibly be used as a control condition.

Their study involved 32 adult epileptic subjects who had been diagnosed with OSA. During an eight week baseline period, the subjects maintained good sleep hygiene (e.g., avoiding sleep deprivation) and kept a seizure diary in which they described the type and frequency of any seizures that occurred during this time. Fifty-nine percent of the subjects underwent continuous positive airway pressure (CPAP) titration to determine their therapeutic pressure while the remaining 41% (the control group) underwent a sham CPAP titration. Neither the subjects nor the research assistants could distinguish the sham CPAP machine from the therapeutic CPAP machine. However, the pressure used by the sham CPAP machine never reached a therapeutic level. Both groups continued to use the CPAP machine at its therapeutic or sham pressure for 10 weeks. The subjects maintained a seizure diary during the 10 week period and continued to keep their anti-seizure medication dosages at the prestudy level.

At the end of the 10 weeks, the researchers found that 28% of the CPAP-treated group had a 50% or greater reduction in seizures. Four people in the treated group became seizure free. By contrast, 15% of the sham CPAP-treated group had a reduction in seizures and only one person in the sham group became seizure free. Although the researchers' focus had been more on testing the feasibility of using a sham CPAP machine as a control condition, Malow et al. were excited to find that OSA treatment may have indeed lessened seizure activity in the treated group.

CPAP treatment in epileptics with OSA may not always improve seizure control. Recently, Italian researchers Silvia Miano et al., reported their experience with a five year old boy for whom CPAP treatment may have induced seizures. He had had symptoms of OSA since soon after birth. When he was two years old, his enlarged tonsils and adenoids were removed. Nevertheless, he continued to snore loudly and have OSA. At five years old, he underwent a polysomnographic (PSG) study and was prescribed CPAP therapy. Within a week of using the machine at its therapeutic pressure, his parents noted he had seizure-like activity (e.g., arm tremors, automatisms, disorientation) lasting about 30 seconds. Due to this, he underwent another PSG study while using the CPAP machine during which he had three seizures. The first two seizures occurred from stage 4 sleep and the last seizure from stage 2 sleep.

A comparison of the boy's pre- and post-CPAP treatment PSGs revealed that the percentage of non-REM sleep did not change. (It was 37% before CPAP treatment; 34% after CPAP treatment.) However, a sleep microstructure feature – cyclic alternating pattern – revealed some differences between the two studies. Cyclic alternating pattern is a phenomenon noted in some people during non-REM sleep in which transient arousals occur at regular intervals (typically about every 20 – 40 seconds) for a period of up to 2 minutes. On an electroencephalogram (EEG), the transient event (phase A) appears alternately with the tonic sleep EEG activity (phase B). Phase A has three subtypes: phase A1, phase A2, and phase A3 (listed by decreasing strength of arousal). Some scientists have noted that some epileptics dur-

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ing slow wave sleep have a high A1 index (i.e., the number of A1 arousals per hour of sleep).

Examination of the boy's pre-treatment PSG revealed that he did have a high A1 index of about 67 events/hour during slow wave sleep. After beginning CPAP therapy, the A1 index increased to 93 events/hour. Miano et al. suspect that the increased A1 activation during slow wave sleep may have been a factor in the boy's seizures.

It is estimated that OSA affects up to 33% of people with refractory partial seizures (i.e., seizures that do not respond well to standard treatment). Poorly controlled seizures can negatively impact quality of one's social life (e.g., the person may not be permitted to drive or the person may be afraid to go to public places for fear of having a seizure). Even when seizures are well controlled with anti-epileptic medications, their side effects (e.g., sleepiness, toxicity, nausea, and vomiting) may negatively impact one's life. Therefore, diagnosing and treating OSA in some epileptics may offer another avenue to reduce seizure frequency which may then allow the person to reduce the amount of anti-epileptic medication needed to control seizure frequency.

Regina Patrick is a Sleep Technologist at St. Vincent Mercy Sleep Center in Toledo, OH and appears regularly in Focus Journal. She can be reached at rpsgtwritr@aol.com.