

Review Article

Epilepsy and Sleep: Sleep Hygiene and Obstructive Sleep Apnea, Sleep Deprivation, Circadian Patterns and Epilepsy Surgery

Leslie E. Perry¹ and Jennifer L. DeWolfe^{2*}¹Department of Neurology, University of Alabama, USA²Department of Neurology and the UAB Epilepsy Center, University of Alabama, USA

Special Issue on

Epilepsy and Seizures

*Corresponding author

DeWolfe JL, Department of Neurology, University of Alabama at Birmingham and the UAB Epilepsy Center, 312 Civitan International Research Center, 1719 6th Avenue South, Birmingham, AL, 35216, USA, Tel: 12059343866; Email: jdewolfe@uab.edu

Submitted: 06 March 2014

Accepted: 19 October 2014

Published: 21 October 2014

Copyright

© 2014 DeWolfe et al.

OPEN ACCESS

Keywords

- Sleep hygiene
- Obstructive sleep apnea
- Epilepsy
- Sleep deprivation
- Circadian rhythm
- Epilepsy surgery

Abstract

The relationship between epilepsy and sleep is complex and dynamic. Sleep complaints and concomitant sleep disorders are common in people with epilepsy. Seizures and antiepileptic drugs can alter sleep architecture. There have been conflicting findings on the impact of sleep deprivation on seizures; however there is evidence to support the improved specificity of epilepsy diagnosis when a negative routine EEG is followed with a sleep-deprived study. The timing of seizure occurrence may be influenced by seizure onset localization; however much remains to be investigated regarding the impact of circadian rhythms and sleep patterns on seizure control. Lastly, epilepsy surgery has been shown to improve sleep quality in patients who remain seizure free. There have been advances in epilepsy and sleep research in light of newer investigational techniques, improved awareness of comorbid sleep disorders and the increasing prevalence of surgically-cured epilepsy patients. This article reviews the impact of sleep hygiene and obstructive sleep apnea on seizures, sleep deprivation on seizures, the circadian pattern on seizures, and finally the impact of epilepsy surgery on sleep.

ABBREVIATIONS

AED: Antiepileptic Drug; AHI: Apnea-Hypopnea Index; EDS: Excessive Daytime Sleepiness; EEG: Electroencephalogram; ESS: Epworth Sleepiness Scale; GABA: γ -Amino butyric Acid; IED: Interictal Epileptiform Discharge; IGE: Idiopathic Generalized Epilepsy; IIA: Interictal Epileptiform Abnormality; JME: Juvenile Myoclonic Epilepsy; NREM: Non Rapid Eye Movement; OSA: Obstructive Sleep Apnea; PSG: Polysomnogram; PWE: People With Epilepsy; REM: Rapid Eye Movement; TLE: Temporal Lobe Epilepsy; TMS: Transcortical Magnetic Stimulation; WASO: Wake After Sleep Onset

INTRODUCTION

Epilepsy and sleep share a bidirectional relationship with aspects of each affecting the other. In 2011, the NIH Sleep Disorders Research Plan suggested a 25-30% prevalence of sleep and circadian disorders amongst the general adult population [1-4]. Subjective sleep disturbances are twice as prevalent amongst people with epilepsy and include insufficient sleep, increased nocturnal and early morning awakenings, impaired sleep

initiation and most commonly, excessive daytime sleepiness [1,3,4]. Sleep disorders and epilepsy can have an additive negative impact on quality of life, work productivity and overall health [1,5].

Interrelation of epilepsy and sleep

Sleep can affect the expression of epilepsy by activating interictal epileptiform discharges and seizures [6]. IEDs occur most often during NREM sleep (particularly stage N2 and to a lesser degree N3 sleep) possibly due to thalamocortical hyper synchrony [2,3]. Their mediation occurs via the same projections responsible for generation of sleep spindles, K-complexes and slow waves during NREM sleep [6]. Conversely, during REM sleep seizures and IEDs are less frequent and IEDs manifest with a more constricted field [7]. The protective role of REM sleep is likely mediated by increased GABAergic activity inhibiting spread of epileptiform discharges, phasic cholinergic neuron activation of the ponto-mesencephalic tegmentum and inhibition of thalamocortical synchronization resulting in EEG desynchronization [6,8-10].

Epilepsy itself, individual seizures, IEDs and epilepsy treatments including AEDs can disrupt sleep architecture and increase sleep fragmentation [7,11-13]. Seizures during sleep are associated with increased N1 sleep and a reduction in daytime alertness [12]. Both diurnal and nocturnal seizures result in a reduction of REM sleep during the subsequent sleep period. Polysomnographic studies in patients with refractory epilepsy show abbreviated sleep time, increased sleep fragmentation, prolonged sleep onset and REM latency, increased stage shifts and more frequent awakenings [3]. Worse sleep efficiency, delay in sleep and REM latency, increased WASO and more frequent arousals were demonstrated on PSG in patients with medically refractory epilepsy compared to patients with controlled epilepsy [14]. These chronic effects may increase the risk of breakthrough seizures in some PWE [5].

Sleep hygiene, obstructive sleep apnea and epilepsy

Sleep hygiene encompasses the habits, behaviors and environment that impact the duration and quality of sleep. Inadequate sleep hygiene can result in poor quality sleep which may facilitate epileptic seizures [15]. There is a paucity of published studies evaluating sleep hygiene in PWE. Khatami, et al. evaluated mean total sleep time and mean time in bed during the week and weekends in PWE versus controls and found no statistical difference between the groups; however sleep-wake behaviors were not reported [16]. In another questionnaire-based study, Manni, et al. investigated the adherence to proper sleep hygiene in PWE, specifically evaluating consumption of caffeinated products and alcohol or tobacco smoking before bedtime, irregular sleep or frequent deprivation, evening napping, sleeping environment (temperature, brightness, noise level), and participation in stressful activities before bedtime. Although there was no statistical correlation between seizure frequency and sleep hygiene factors, PWE reported better sleep hygiene compared to normal controls, suggesting that PWE may avoid activities known to promote breakthrough seizures, many of which overlap with good sleep hygiene practices [17].

Sleep disorders commonly occur in PWE and can lead to EDS and contribute to intractable epilepsy [3,18]. Initially felt to be symptomatic of AEDs or seizures, EDS has been found to be associated with undiagnosed sleep disorders such as sleep-disordered breathing and restless legs symptoms independent of seizure frequency or AED side effects, with symptoms of the disorders more closely correlating with severity of EDS than seizure frequency or drug therapy [5,16]. There is increasing evidence that OSA is more prevalent in adults with epilepsy compared to the general population [19-21]. Malow, et al. observed that 33% of 39 patients with refractory partial epilepsy who underwent PSG unselected for sleep disturbances demonstrated at least mild OSA ($AHI \geq 10$) and ~13% with moderate or severe OSA [21]. A similar prevalence of OSA, 30% with $AHI \geq 10$ and 16% with moderate or severe OSA, was found in a retrospective study of PWE unselected for sleep complaints and epilepsy severity [20]. Chihorek, et al. demonstrated an increased prevalence of OSA amongst older adults with worsening seizure control or new onset seizures, proposing that chronic sleep deprivation unmasks seizures in susceptible persons [22]. The mechanisms underlying these findings are not known, however

impaired sleep quality (sleep fragmentation, frequent stage shifts and frequent arousals in addition to sleep deprivation) and the impact of acute and chronic effects of intermittent hypoxia and sympathetic activation on epileptogenic regions of the brain may be contributory [14,21]. AED therapy may also impact prevalence of OSA in PWE [23], however additional studies are needed to explore this relationship further. Both retrospective [24-27] and prospective [28,29] studies suggest that treating sleep apnea reduces seizure burden. One prospective pilot study demonstrated feasibility for a multisite randomized, double-blind design to screen for and treat OSA with CPAP in patients with refractory epilepsy and concomitant OSA to evaluate the impact on seizure control [29]. Larger controlled trials utilizing this design have not yet been published.

Sleep deprivation and epilepsy

For more than 50 years, sleep deprivation has been used as an activating technique for seizures, with studies published since the early 1960's suggesting its importance in improving the yield of EEG in diagnosing epilepsy [30,31]. Although this practice for EEG continues [32], a standardized sleep deprivation protocol does not exist. Day long sleep deprivation has increased IIA in patients with partial epilepsy [33]. Early afternoon EEGs following shortened overnight sleep (8 hours for < 4 y/o, 6 hours for 4-14 y/o, and 5 hours for > 14 y/o) resulted in 53% EEGs positive for IIA in people with suspected epilepsy [34]. Giorgi, et al. demonstrated IIAs on EEG following at least six hours of sleep deprivation in 41% of de novo patients with suspected seizures and previous normal routine EEG with 91% specificity for epilepsy diagnosis. Patients with focal epilepsy had increased positive yield on sleep deprived EEG following a normal initial routine EEG compared to a second routine EEG [35].

Controversy exists over whether EEG activation is related to effects of increased neuronal excitability or merely induction of sleep [3]. Badawy, et al suggests that the result is due to an imbalance between neuronal excitation and inhibition in the setting of sleep deprivation [36]. Studies utilizing TMS objectively determined levels of cortical excitability in PWE before and after sleep deprivation. Sleep deprivation increased cortical excitability in the bilateral cerebral hemispheres in idiopathic generalized epilepsies and unilateral hemispheres in focal epilepsy syndromes in people with newly diagnosed epilepsy naïve to AEDs. TMS findings are thought to represent an imbalance between inhibitory GABA_A mediated circuits and excitatory glutamate-mediated circuits, although the net effect has been disputed [36]. TMS studies in patients with JME demonstrated a more pronounced disruption in the balance between inhibitory and excitatory circuits of the primary motor cortex patients with JME when compared to normal controls [37, 38].

There is also evidence to suggest that selective loss of REM sleep has a proconvulsant role, although the exact mechanisms are not clearly understood [39]. Recent animal studies have shown that with REM sleep loss a complex cascade of intracellular and molecular events leads to increased noradrenalin levels and activation of Na-K ATPase activity which ultimately increases brain excitability [40].

Although several experimental studies in animal models, healthy controls, and PWE highlight the role of sleep deprivation and sleeping during an inappropriate circadian phase (i.e., in the morning) in enhancing sleep instability and possibly causing the occurrence of IIAs and epileptic seizures, there is disagreement over the precise benefit and application of this practice [35]. One study in patients with medically intractable focal epilepsy undergoing inpatient video-EEG monitoring randomized patients to every other night sleep deprivation (awake from 10 PM to 6 AM) or to normal sleep with both groups remaining awake from 6 AM to 10 PM. There was no difference in seizure frequency or secondarily generalized seizures in either group, thus it was suggested that acute sleep deprivation may not affect seizure frequency during inpatient monitoring in this population of PWE [41]. Scientific papers addressing sleep deprivation as an activating technique vary significantly by way of patient population and study design including length and type of EEG performed, timing of EEG after sleep deprivation and duration of sleep deprivation [6,35]. Other important variables such as timing of EEG since last seizure, concurrent AED therapy, patient age and use of activating techniques during EEG recording limit the comparison across distinct studies. Regardless, high specificity of sleep-deprived EEG has been demonstrated and the above questions do not negate its utility in the diagnosis and presurgical evaluation of patients [42]. These inconsistencies merely highlight need for future research and standardization of practice.

Circadian pattern and seizures

Studies have demonstrated a time-based distribution of peak incidence of seizure occurrence and interictal epileptiform abnormalities within a 24-hour day that correlates with certain epilepsy syndromes [43,44]. In patients with temporal lobe epilepsy, the peak incidence of seizures occurs in the late afternoon to early evening with a peak of 1500 hours in people with mesial temporal epilepsy [45,46]. Seizures, however, are more likely to generalize during sleep [12]. Frontal seizures occurred more often between 9PM and 5AM. Parietal seizures occurred most often from 4PM to 9PM. There were greater proportion of waking seizures between 5AM-11AM and 4PM-9PM with sleep-related seizures occurring from 11AM-4PM and 9PM-5AM [47]. The results of these studies, performed in an inpatient video EEG monitoring unit setting, correlated with the results of both an outpatient study utilizing ambulatory EEG [48] and an intracranial video EEG study [49].

Further evidence of circadian influences on epilepsy has been demonstrated, with one study showing an increased night time heart rate variation in people with localization-related epilepsy [50]. PWE have been shown to have a morningness chronotype in which individuals tend to go to bed and wake earlier and are more alert in the first part of the day [45]. Thus, chronotype has been demonstrated to influence timing of taking antiepileptic medications [51]. Despite differing time-based distributions of peak seizure incidence among epilepsy syndromes, chronotypes do not differ significantly between patients with specified epilepsy syndromes. It has been suggested that epilepsy itself rather than timing of seizures has a significant influence on chronotype and subjective sleep parameters [45].

An endogenously-mediated circadian pattern to limbic seizures has been demonstrated in rat models [52].

One study by Quigg, et al. suggests the circadian pattern to seizure occurrence is the result of passive entrainment [53], while a study by Stanley, et al. indicates that the 24-hr rhythm may be actively driven by aberrant circadian regulation promoting periods of excitatory and inhibitory balance throughout the day [54]. This latter concept is demonstrated by a phase shift of approximately 12 hours in the 24-hour rhythm of hippocampal spikes in an animal model of limbic epilepsy and elicits consideration of the impact of epilepsy itself on circadian patterns. It has been proposed that PWE have permanent structural changes that may impair normal circadian rhythms. This thought is supported by the finding of circadian abnormalities (impaired sleep-wake cycle, increased core temperature variability recordings and alterations in melatonin release) amongst animal models. Altered circadian input to the epileptic brain regions may have direct causality on excitatory-inhibitory imbalances associated with seizures [1,54,55].

Common methods to objectively evaluate individual human circadian rhythm phase include actigraphy (an indirect measure of sleep patterns) and biological markers such as variations in core body temperature, cortisol levels and the gold standard dim light melatonin onset (DLMO) [56,57]. In one study, correlating biological markers of circadian rhythm with time-based dispersion of seizure occurrence in PWE, temporal lobe seizures occurred more frequently 6 hours prior to DLMO and frontal lobe seizures mostly 6-12 hours after DLMO suggesting that seizure occurrence is synchronized to circadian rhythm phase in a non-random pattern [58]. The impact of individual circadian rhythms on chronotype in PWE is not well known [59]. It is also unknown whether subjective chronotypes align with objective sleep patterns and circadian rhythms and how each impacts seizure frequency in PWE.

Effects of epilepsy surgery on sleep

Vagus nerve stimulation has been associated with increased AHI and inducing OSA while improving daytime vigilance in PWE. Low VNS stimulus intensities and increasing off time can reduce AHI and improve quality of life in some PWE [60-63]. Less is known about the impact of epilepsy surgery on sleep. One study reported resolution of preoperative, PSG-diagnosed moderate OSA associated with an oxygen saturation as low as 62% in a patient with medically intractable epilepsy who underwent left frontal lobe resection. Postoperatively, the patient had marked reduction in IEDs and seizures with normalization of baseline oxygen saturation and resolution of OSA suggesting that IED and seizures may play a role in modulating upper airway control during sleep [64]. In 2010, Carrion, et al. assessed subjective sleep quality and EDS in a 48 patients with refractory TLE undergoing epilepsy surgery at 2 days and 3 months following surgical resection and found improved subjective sleep quality independent of gender, AED class, age or seizure frequency [65]. Zanzmera, et al. demonstrated improved objective sleep quality, sleep architecture and AHI on PSG three months following epilepsy surgery compared to preoperative baseline in patients with medically intractable refractory focal epilepsy who had Engel class I and II surgical outcomes [66]. A similar result was

demonstrated by Serafini, et al. who report a reduction in IIA in all study participants following temporal lobectomy for TLE. They verified an increase in total sleep time and REM sleep at 1 year follow-up with further increase in percentage of REM sleep at 2 years in patients who were seizure-free and controlled for significant AED changes during time observed. Their findings support improved sleep macrostructure (sleep onset latency, total sleep time, sleep efficiency index, number of awakenings duration of sleep stages and REM latency) following reduction of seizure and IIA burden after epilepsy surgery [67]. These studies indirectly confirm the role of epilepsy in disrupting sleep organization and may provide future insight into degree of reversibility of changes.

DISCUSSION AND CONCLUSION

While it is clear that an interaction between sleep and epilepsy exists—with sleep impacting the expression of IED and seizure activity and seizures, substrates underlying epilepsy and epilepsy treatments modulating sleep quality and sleep architecture—there are many unanswered questions regarding this dynamic reciprocal relationship. The current evidence demonstrates that PWE have better sleep hygiene than controls and suggests that worse sleep hygiene may be associated with worse seizure control. More comprehensive investigations into sleep hygiene measures in PWE are needed to relate underlying pathologies that disrupt sleep with factors such as sleep-wake behaviors, subjective and objective sleep measures, AED use, and seizure type and frequency. Obstructive sleep apnea and epilepsy are common disorders that are both associated with disrupted sleep and chronic sleep deprivation. Investigations have shown an increased prevalence of OSA in PWE with smaller studies suggesting improved seizure control after treating OSA. Larger prospective randomized, placebo-controlled studies are needed to definitively determine the impact of OSA treatment on seizure control in PWE.

Sleep deprivation can increase positive findings of IIA and seizures on EEG in some PWE, thus improving diagnoses of specific epilepsies. Although sleep deprivation increases cortical excitability as demonstrated in multiple TMS studies, further studies are needed to determine these underlying mechanisms. It may be the combination of increased excitability and capturing sleep that leads to increased yield of IIA and seizures on sleep deprived EEG. Additionally, it may be that chronic sleep deprivation has a larger impact on seizure frequency than acute sleep deprivation. Investigations controlling for age, amount of sleep deprivation, type and timing of EEG, activation techniques during EEG and AED use may help determine answers to these questions.

Investigations on the timing of seizures demonstrate that temporal lobe seizures occur most often during the day while frontal lobe seizures occur more commonly during sleep. Further investigation is needed to determine if individual circadian rhythms are aligned with sleep patterns in PWE and if adjusting these patterns affects seizure control.

While VNS has shown to improve daytime vigilance, high rates of stimulus intensities have been associated with increased incidence of OSA. In the few published studies in PWE following

epilepsy surgery, sleep quality was improved as documented by PSG in patients with a good surgical outcome. The cortical control of OSA mechanisms needs to be investigated further to understand the impact epilepsy surgery may have on sleep-disordered breathing. Overall, increased diagnosis and thus treatment of sleep disorders is likely to improve seizure control and may have additive improvements in quality of life in PWE.

CONFLICT OF INTEREST

LEP has no conflicts of interest. JLD performs or has performed research sponsored by NIH, Eisai, GlaxoSmithKline, Marinus and UCB Pharma.

REFERENCES

1. de Weerd A, de Haas S, Otte A, Trenité DK, van Erp G, Cohen A. Subjective sleep disturbance in patients with partial epilepsy: a questionnaire-based study on prevalence and impact on quality of life. *Epilepsia*. 2004; 45: 1397-1404.
2. Shelgikar AV, Chervin R. Approach to and evaluation of sleep disorders. *Continuum (Minneapolis)*. 2013; 19: 32-49.
3. Van Golde EG, Gutter T, de Weerd AW. Sleep disturbances in people with epilepsy; prevalence, impact and treatment. *Sleep Med Rev*. 2011; 15: 357-368.
4. Watson NF, Viola-Saltzman M. Sleep and comorbid neurologic disorders. *Continuum (Minneapolis)*. 2013; 19: 148-169.
5. Giorelli AS, Passos P, Carnaval T, Gomes Mda M. Excessive daytime sleepiness and epilepsy: a systematic review. *Epilepsy Res Treat*. 2013; 2013: 629469.
6. Díaz-Negrillo A. Influence of sleep and sleep deprivation on ictal and interictal epileptiform activity. *Epilepsy Res Treat*. 2013; 2013: 492524.
7. Foldvary-Schaefer N, Grigg-Damberger M. Sleep and epilepsy. *Semin Neurol*. 2009; 29: 419-428.
8. Busek P, Buskova J, Nevsimalova S. Interictal epileptiform discharges and phasic phenomena of REM sleep. *Epileptic Disord*. 2010; 12: 217-221.
9. Herman ST, Walczak TS, Bazil CW. Distribution of partial seizures during the sleep-wake cycle: differences by seizure onset site. *Neurology*. 2001; 56: 1453-1459.
10. Ng M, Pavlova M. Why are seizures rare in rapid eye movement sleep? Review of the frequency of seizures in different sleep stages. *Epilepsy Res Treat*. 2013; 2013: 932790.
11. Xu X, Brandenburg NA, McDermott AM, Bazil CW. Sleep disturbances reported by refractory partial-onset epilepsy patients receiving polytherapy. *Epilepsia*. 2006; 47: 1176-1183.
12. Bazil CW, Castro LH, Walczak TS. Reduction of rapid eye movement sleep by diurnal and nocturnal seizures in temporal lobe epilepsy. *Arch Neurol*. 2000; 57: 363-368.
13. Jain SV, Glauser TA. Effects of epilepsy treatments on sleep architecture and daytime sleepiness: an evidence-based review of objective sleep metrics. *Epilepsia*. 2014; 55: 26-37.
14. Zanznera P, Shukla G, Gupta A, Singh H, Goyal V, Srivastava A. Markedly disturbed sleep in medically refractory compared to controlled epilepsy - a clinical and polysomnography study. *Seizure*. 2012; 21: 487-490.
15. Shouse MN, da Silva AM, Sammaritano M. Circadian rhythm, sleep, and epilepsy. *J Clin Neurophysiol*. 1996; 13: 32-50.

16. Khatami R, Zutter D, Siegel A, Mathis J, Donati F, Bassetti CL. Sleep-wake habits and disorders in a series of 100 adult epilepsy patients--a prospective study. *Seizure*. 2006; 15: 299-306.
17. Manni R, Politini L, Ratti MT, Marchioni E, Sartori I, Galimberti CA. Sleep hygiene in adult epilepsy patients: a questionnaire-based survey. *Acta Neurol Scand*. 2000; 101: 301-304.
18. Bazil CW. Sleep, Sleep Apnea, and Epilepsy. *Curr Treat Options Neurol*. 2004; 6: 339-345.
19. Economou NT, Dikeos D, Andrews N, Foldvary-Schaefer N. Use of the Sleep Apnea Scale of the Sleep Disorders Questionnaire (SA-SDQ) in adults with epilepsy. *Epilepsy Behav*. 2014; 31: 123-126.
20. Foldvary-Schaefer N, Andrews ND, Pornsriniyom D, Moul DE, Sun Z, Bena J. Sleep apnea and epilepsy: who's at risk? *Epilepsy Behav*. 2012; 25: 363-367.
21. Malow BA, Levy K, Maturen K, Bowes R. Obstructive sleep apnea is common in medically refractory epilepsy patients. *Neurology*. 2000; 55: 1002-1007.
22. Chihorek AM, Abou-Khalil B, Malow BA. Obstructive sleep apnea is associated with seizure occurrence in older adults with epilepsy. *Neurology*. 2007; 69: 1823-1827.
23. Jain SV, Simakajornboon S, Shapiro SM, Morton LD, Leszczyszyn DJ, Simakajornboon N. Obstructive sleep apnea in children with epilepsy: prospective pilot trial. *Acta Neurol Scand*. 2012; 125: e3-6.
24. Devinsky O, Ehrenberg B, Barthlen GM, Abramson HS, Luciano D. Epilepsy and sleep apnea syndrome. *Neurology*. 1994; 44: 2060-2064.
25. Malow BA, Fromes GA, Aldrich MS. Usefulness of polysomnography in epilepsy patients. *Neurology*. 1997; 48: 1389-1394.
26. Vaughn BV, D'Cruz OF, Beach R, Messenheimer JA. Improvement of epileptic seizure control with treatment of obstructive sleep apnoea. *Seizure*. 1996; 5: 73-78.
27. Vendrame M, Auerbach S, Loddenkemper T, Kothare S, Montouris G. Effect of continuous positive airway pressure treatment on seizure control in patients with obstructive sleep apnea and epilepsy. *Epilepsia*. 2011; 52: e168-171.
28. Malow BA, Weatherwax KJ, Chervin RD, Hoban TF, Marzec ML, Martin C. Identification and treatment of obstructive sleep apnea in adults and children with epilepsy: a prospective pilot study. *Sleep Med*. 2003; 4: 509-515.
29. Malow BA, Foldvary-Schaefer N, Vaughn BV, Selwa LM, Chervin RD, Weatherwax KJ. Treating obstructive sleep apnea in adults with epilepsy: a randomized pilot trial. *Neurology*. 2008; 71: 572-577.
30. Mattson RH, Pratt KL, Calverley JR. Electroencephalograms of epileptics following sleep deprivation. *Arch Neurol*. 1965; 13: 310-315.
31. JANZ D. The grand mal epilepsies and the sleeping-waking cycle. *Epilepsia*. 1962; 3: 69-109.
32. Leach JP, Stephen LJ, Salveta C, Brodie MJ. Which electroencephalography (EEG) for epilepsy? The relative usefulness of different EEG protocols in patients with possible epilepsy. *J Neurol Neurosurg Psychiatry*. 2006; 77: 1040-1042.
33. Drake ME Jr, Pakalnis A, Phillips BB, Denio LS. Sleep and sleep deprived EEG in partial and generalized epilepsy. *Acta Neurol Belg*. 1990; 90: 11-19.
34. Kubicki S, Scheuler W, Wittenbecher H. Short-term sleep EEG recordings after partial sleep deprivation as a routine procedure in order to uncover epileptic phenomena: an evaluation of 719 EEG recordings. *Epilepsy Res Suppl*. 1991; 2: 217-230.
35. Giorgi FS, Maestri M, Guida M, Di Coscio E, Carnicelli L, Perini D. Controversial issues on EEG after sleep deprivation for the diagnosis of epilepsy. *Epilepsy Res Treat*. 2013; 2013: 614685.
36. Badawy RA, Curatolo JM, Newton M, Berkovic SF, Macdonell RA. Sleep deprivation increases cortical excitability in epilepsy: syndrome-specific effects. *Neurology*. 2006; 67: 1018-1022.
37. Manganotti P, Bongiovanni LG, Fuggetta G, Zanette G, Fiaschi A. Effects of sleep deprivation on cortical excitability in patients affected by juvenile myoclonic epilepsy: a combined transcranial magnetic stimulation and EEG study. *J Neurol Neurosurg Psychiatry*. 2006; 77: 56-60.
38. Civardi C, Boccagni C, Vicentini R, Bolamperti L, Tarletti R, Varrasi C. Cortical excitability and sleep deprivation: a transcranial magnetic stimulation study. *J Neurol Neurosurg Psychiatry*. 2001; 71: 809-812.
39. Placidi F, Zannino S, Albanese M, Romigi A, IZZI F, Marciani MG. Increased cortical excitability after selective REM sleep deprivation in healthy humans: a transcranial magnetic stimulation study. *Sleep Med*. 2013; 14: 288-292.
40. Mallick BN, Singh A. REM sleep loss increases brain excitability: role of noradrenaline and its mechanism of action. *Sleep Med Rev*. 2011; 15: 165-178.
41. Malow BA, Passaro E, Milling C, Minecan DN, Levy K. Sleep deprivation does not affect seizure frequency during inpatient video-EEG monitoring. *Neurology*. 2002; 59: 1371-1374.
42. Leach JP, Stephen LJ, Salveta C, Brodie MJ. Which electroencephalography (EEG) for epilepsy? The relative usefulness of different EEG protocols in patients with possible epilepsy. *J Neurol Neurosurg Psychiatry*. 2006; 77: 1040-1042.
43. Badawy R, Macdonell R, Jackson G, Berkovic S. The peri-ictal state: cortical excitability changes within 24 h of a seizure. *Brain*. 2009; 132: 1013-1021.
44. Hofstra WA, Grootemarsink BE, Dieker R, van der Palen J, de Weerd AW. Temporal distribution of clinical seizures over the 24-h day: a retrospective observational study in a tertiary epilepsy clinic. *Epilepsia*. 2009; 50: 2019-2026.
45. Hofstra WA, Gordijn MC, van Hemert-van der Poel JC, van der Palen J, De Weerd AW. Chronotypes and subjective sleep parameters in epilepsy patients: a large questionnaire study. *Chronobiol Int*. 2010; 27: 1271-1286.
46. Hofstra WA, Spetgens WP, Leijten FS, van Rijen PC, Gosselaar P, van der Palen J. Diurnal rhythms in seizures detected by intracranial electrocorticographic monitoring: an observational study. *Epilepsy Behav*. 2009; 14: 617-621.
47. Hofstra WA, Spetgens WP, Leijten FS, van Rijen PC, Gosselaar P, van der Palen J. Diurnal rhythms in seizures detected by intracranial electrocorticographic monitoring: an observational study. *Epilepsy Behav*. 2009; 14: 617-621.
48. Pavlova MK, Woo Lee J, Yilmaz F, Dworetzky BA. Diurnal pattern of seizures outside the hospital: is there a time of circadian vulnerability? *Neurology*. 2012; 78: 1488-1492.
49. Durazzo TS, Spencer SS, Duckrow RB, Novotny EJ, Spencer DD, Zaveri HP. Temporal distributions of seizure occurrence from various epileptogenic regions. *Neurology*. 2008; 70: 1265-1271.
50. Persson H, Kumlien E, Ericson M, Tomson T. Circadian variation in heart-rate variability in localization-related epilepsy. *Epilepsia*. 2007; 48: 917-922.
51. Hofstra WA, van der Palen J, de Weerd AW. Morningness and eveningness: when do patients take their antiepileptic drugs? *Epilepsy Behav*. 2012; 23: 320-323.

52. Quigg M, Clayburn H, Straume M, Menaker M, Bertram EH 3rd. Effects of circadian regulation and rest-activity state on spontaneous seizures in a rat model of limbic epilepsy. *Epilepsia*. 2000; 41: 502-509.
53. Quigg M. Circadian rhythms: interactions with seizures and epilepsy. *Epilepsy Res*. 2000; 42: 43-55.
54. Stanley DA, Talathi SS, Parekh MB, Cordiner DJ, Zhou J, Mareci TH. Phase shift in the 24-hour rhythm of hippocampal EEG spiking activity in a rat model of temporal lobe epilepsy. *J Neurophysiol*. 2013; 110: 1070-1086.
55. Hofstra WA, de Weerd AW. The circadian rhythm and its interaction with human epilepsy: a review of literature. *Sleep Med Rev*. 2009; 13: 413-420.
56. Hofstra WA, de Weerd AW. How to assess circadian rhythm in humans: a review of literature. *Epilepsy Behav*. 2008; 13: 438-444.
57. Pandi-Perumal SR, Smits M, Spence W, Srinivasan V, Cardinali DP, Lowe AD. Dim light melatonin onset (DLMO): a tool for the analysis of circadian phase in human sleep and chronobiological disorders. *Prog Neuropsychopharmacol Biol Psychiatry*. 2007; 31: 1-11.
58. Hofstra WA, Gordijn MC, van der Palen J, van Regteren R1, Grootemarsink BE1, de Weerd AW1. Timing of temporal and frontal seizures in relation to the circadian phase: a prospective pilot study. *Epilepsy Res*. 2011; 94: 158-162.
59. Hofstra WA, de Weerd AW. The circadian rhythm and its interaction with human epilepsy: a review of literature. *Sleep Med Rev*. 2009; 13: 413-420.
60. Hsieh T, Chen M, McAfee A, Kifle Y. Sleep-related breathing disorder in children with vagal nerve stimulators. *Pediatr Neurol*. 2008; 38: 99-103.
61. Malow BA, Edwards J, Marzec M, Sagher O, Fromes G. Effects of vagus nerve stimulation on respiration during sleep: a pilot study. *Neurology*. 2000; 55: 1450-1454.
62. Marzec M, Edwards J, Sagher O, Fromes G, Malow BA. Effects of vagus nerve stimulation on sleep-related breathing in epilepsy patients. *Epilepsia*. 2003; 44: 930-935.
63. Malow BA, Edwards J, Marzec M, Sagher O, Ross D, Fromes G. Vagus nerve stimulation reduces daytime sleepiness in epilepsy patients. *Neurology*. 2001; 57: 879-884.
64. Foldvary-Schaefer N, Stephenson L, Bingaman W. Resolution of obstructive sleep apnea with epilepsy surgery? Expanding the relationship between sleep and epilepsy. *Epilepsia*. 2008; 49: 1457-1459.
65. Carrion MJ, Nunes ML, Martinez JV, Portuguez MW, da Costa JC. Evaluation of sleep quality in patients with refractory seizures who undergo epilepsy surgery. *Epilepsy Behav*. 2010; 17: 120-123.
66. Zanzmera P, Shukla G, Gupta A, Goyal V, Srivastava A, Garg A. Effect of successful epilepsy surgery on subjective and objective sleep parameters--a prospective study. *Sleep Med*. 2013; 14: 333-338.
67. Serafini A, Kuate C, Gelisse P, Velizarova R, Gigli GL, Coubes P. Sleep before and after temporal lobe epilepsy surgery. *Seizure*. 2012; 21: 260-265.

Cite this article

Perry LE, DeWolfe JL (2014) *Epilepsy and Sleep: Sleep Hygiene and Obstructive Sleep Apnea, Sleep Deprivation, Circadian Patterns and Epilepsy Surgery*. *J Neurol Disord Stroke* 2(5): 1088.